



Effects of water, sanitation, and hygiene interventions on detection of enteropathogens and host-specific faecal markers in the environment: a systematic review and individual participant data meta-analysis

Andrew Mertens, Benjamin F Arnold, Jade Benjamin-Chung, Alexandria B Boehm, Joe Brown, Drew Capone, Prof Thomas Clasen, Erica Fuhrmeister, Jessica A Grembi, David Holcomb, Jackie Knee, Laura H Kwong, Audrie Lin, Stephen P Luby, Rassul Nala, Kara Nelson, Sammy M Njenga, Clair Null, Amy J Pickering, Mahbubur Rahman, Heather E Reese, Lauren Steinbaum, Jill Stewart, Ruwan Thilakarathne, Oliver Cumming, John M Colford Jr, Ayse Ercumen



Summary

Background Water, sanitation, and hygiene (WASH) improvements are promoted to reduce diarrhoea in low-income countries. However, trials from the past 5 years have found mixed effects of household-level and community-level WASH interventions on child health. Measuring pathogens and host-specific faecal markers in the environment can help investigate causal pathways between WASH and health by quantifying whether and by how much interventions reduce environmental exposure to enteric pathogens and faecal contamination from human and different animal sources. We aimed to assess the effects of WASH interventions on enteropathogens and microbial source tracking (MST) markers in environmental samples.

Methods We did a systematic review and individual participant data meta-analysis, which included searches from Jan 1, 2000, to Jan 5, 2023, from PubMed, Embase, CAB Direct Global Health, Agricultural and Environmental Science Database, Web of Science, and Scopus, of prospective studies with water, sanitation, or hygiene interventions and concurrent control group that measured pathogens or MST markers in environmental samples and measured child anthropometry, diarrhoea, or pathogen-specific infections. We used covariate-adjusted regression models with robust standard errors to estimate study-specific intervention effects and pooled effect estimates across studies using random-effects models.

Findings Few trials have measured the effect of sanitation interventions on pathogens and MST markers in the environment and they mostly focused on onsite sanitation. We extracted individual participant data on nine environmental assessments from five eligible trials. Environmental sampling included drinking water, hand rinses, soil, and flies. Interventions were consistently associated with reduced pathogen detection in the environment but effect estimates in most individual studies could not be distinguished from chance. Pooled across studies, we found a small reduction in the prevalence of any pathogen in any sample type (pooled prevalence ratio [PR] 0.94 [95% CI 0.90–0.99]). Interventions had no effect on the prevalence of MST markers from humans (pooled PR 1.00 [95% CI 0.88–1.13]) or animals (pooled PR 1.00 [95% CI 0.97–1.03]).

Interpretation The small effect of these sanitation interventions on pathogen detection and absence of effects on human or animal faecal markers are consistent with the small or null health effects previously reported in these trials. Our findings suggest that the basic sanitation interventions implemented in these studies did not contain human waste and did not adequately reduce exposure to enteropathogens in the environment.

Funding Bill and Melinda Gates Foundation and the UK Foreign and Commonwealth Development Office.

Copyright © 2023 The Author(s). Published by Elsevier Ltd. This is an Open Access article under the CC BY 4.0 license.

Introduction

Every year, diarrhoea kills an estimated 525 000 children younger than 5 years.¹ Enteropathogens (pathogens causing infections of the intestinal track) are transmitted from infected individuals' faeces to new hosts through a diverse set of interconnected environmental pathways, including contaminated water or food, hands, fomites, and vectors (eg, flies). Sources of faecal contamination

include open defecation, unsafe sanitation facilities that do not isolate waste from the environment, and domestic animals. Water, sanitation, and hygiene (WASH) improvements have been promoted to reduce childhood enteric infections by reducing environmental person-to-person exposure to pathogens transmitted via the faecal-oral route. Traditionally, trials of WASH interventions have primarily focused on documenting health outcomes,

Lancet Planet Health 2023;
7: e197–208

Division of Epidemiology and Biostatistics (A Mertens PhD, R Thilakarathne MPH, Prof J M Colford Jr), Division of Environmental Health Sciences (L H Kwong PhD), and Department of Civil and Environmental Engineering, College of Engineering (Prof K Nelson PhD, A J Pickering PhD), University of California, Berkeley, CA, USA; Francis I Proctor Foundation and Department of Ophthalmology, University of California, San Francisco, CA, USA (B F Arnold PhD); Department of Epidemiology and Population Health (J Benjamin-Chung PhD), Department of Civil and Environmental Engineering (Prof A B Boehm PhD), Department of Medicine (J A Grembi PhD), and Division of Infectious Diseases and Geographic Medicine (Prof S P Luby MD), Stanford University, Stanford, CA, USA; Department of Environmental Science and Engineering, Gillings School of Global Public Health, Michael Hooker Research Center, University of North Carolina, Chapel Hill, NC, USA (J Brown PhD, D Holcomb PhD, Prof J Stewart PhD); Department of Environmental and Occupational Health, Indiana University Bloomington, Bloomington, IN, USA (D Capone PhD); Department of Environmental Health, Rollins School of Public Health, Emory University, NE, Atlanta, GA, USA (Prof T Clasen PhD, H E Reese PhD); Department of Environmental and Occupational Health Sciences, University

of Washington, Seattle, WA, USA (E Fuhrmeister PhD); Department of Disease Control, London School of Tropical Medicine & Hygiene, London, UK (J Kneee PhD, O Cumming PhD); Department of Biobehavioral Health, Pennsylvania State University, PA, USA (A Lin PhD); Ministério da Saúde, Instituto Nacional de Saúde Maputo, Maputo, Mozambique (R Nala MPH); Kenya Medical Research Institute, Nairobi, Kenya (S M Njenga PhD); Mathematica, Princeton, NJ, USA (C Null PhD); Environmental Interventions Unit, Infectious Diseases Division, Dhaka, Bangladesh (M Rahman MBBS); Center for the Ecology of Infectious Diseases, University of Georgia, Athens, GA, USA (L Steinbaum PhD); Department of Forestry and Environmental Resources, North Carolina State University, Raleigh, NC, USA (A Arcumen PhD)

Correspondence to: Dr Andrew Mertens, Division of Epidemiology, School of Public Health, University of California, Berkeley, CA 94704, USA amertens@berkeley.edu

Research in context

Evidence before this study

Children in areas with poor drinking water, sanitation, and hygiene (WASH) conditions have increased diarrhoeal disease and reduced growth. Rigorous trials of WASH interventions from the past 5 years have shown mixed efficacy in reducing diarrhoeal disease in children and no improvements in child growth. Quantifying the effect of WASH improvements on enteric pathogens in environmental samples and on contamination originating from human versus animal sources might help elucidate if interventions successfully interrupt the causal pathway between poor WASH, environmental exposure to faecal pathogens, and child health. Most previous studies and meta-analyses on the effect of WASH interventions on faecal contamination in the environment have focused on faecal indicator bacteria (FIB). These studies have shown FIB reductions in water and on hands due to water treatment and handwashing, respectively, but no effects from sanitation. However, naturalised FIB can be present in the environment without faecal contamination, and poorly correlate with actual pathogen presence, affecting the interpretation of these findings. FIB also cannot distinguish between faecal contamination from humans and animals; this information can help identify whether the absence of health effects from sanitation interventions are due to zoonotic disease transmission from unsafely managed animal faeces.

Applications of advanced analytic techniques to environmental sampling in low-income countries allow detection and enumeration of a range of enteropathogens, and faecal markers associated with specific hosts for microbial source tracking (MST). The evidence on WASH effects on pathogens and MST markers has not been previously synthesised. Therefore, we did a systematic review and individual participant data (IPD) meta-analysis of WASH intervention studies to assess if interventions reduced the prevalence and abundance of enteropathogens or MST markers in the domestic environment.

Added value of this study

We identified six eligible intervention studies that measured enteropathogens and MST markers in environmental samples and obtained data from five studies. Studies consistently indicated that the interventions were associated with reduced detection of pathogens, and some pathogens (eg, adenovirus and Shigella) showed large reductions, but most effect

estimates in individual studies were null. The IPD meta-analysis approach allowed us to increase precision by pooling study-specific estimates to detect a small reduction in the prevalence of any type of pathogen in any type of sample. There was no overall intervention effect on the prevalence of human or animal MST markers. This study uses advanced methods to enumerate enteropathogens and host-specific faecal markers in a range of environmental samples, including understudied reservoirs such as soil. We provide the first synthesis of evidence of the effect of WASH interventions on these important targets to advance our understanding of the environmental mechanisms of interventions beyond the available evidence on effects on FIB.

Implications of all the available evidence

The environmental sampling in the studies in our review was mostly focused on onsite sanitation interventions that provided or promoted improved latrines with pits or septic tanks at the household or community level. The small reduction in pathogen prevalence in the environment, when pooled across all studies, might explain the small effect these interventions had on child health in the parent studies. Taken together, this evidence suggests that the sanitation interventions implemented in the studies in our review achieved a small reduction in faecal contamination in the environment. More comprehensive WASH interventions, such as safely managed water and sanitation, including safe disposal or treatment of excreta from both humans and animals, are potentially needed to reduce environmental contamination sufficiently to improve child health. We note that only a small number of intervention studies measured our targets of interest, and we identified no studies that assessed the effect of water treatment, hygiene, or safely managed or sewered sanitation interventions on pathogens and MST markers in the domestic environment, except for one study on a combined household water treatment and hygiene intervention in which data were not available for IPD analysis. Also, pathogen targets and analytic methods varied by study, reducing comparability. Future research would benefit from environmental sampling following implementation of a more diverse and comprehensive set of WASH interventions. Such studies should enumerate a common range of pathogen targets and use standardised laboratory methods for a given target and environmental matrix.

such as caregiver-reported diarrhoea, without measuring intermediate outcomes along the causal chain, such as pathogens in environmental samples to characterise exposure. Such measurements can illuminate underlying mechanisms of interventions and offer explanations for intervention success or failure. Inspecting the causal chain is especially important given the small or null effects on child diarrhoea and growth in WASH trials.²⁻⁶

To date, WASH intervention studies measuring environmental contamination have mostly relied on

faecal indicator bacteria (FIB), such as *Escherichia coli* as a proxy for a wide range of enteropathogens, including bacteria, viruses, protozoa, and helminths. Sampling has primarily focused on drinking water (and, to a smaller extent, hands and food) whereas other pathways, such as soil and surfaces, have received less attention.⁷ Household water treatment and handwashing have been associated with reduced FIB in drinking water⁸ and on hands,^{9,10} respectively, whereas sanitation interventions have had little effect on FIB in drinking water or on hands, objects,

surfaces, soil, and flies.⁷ However, FIB are imperfect predictors of faecal contamination, pathogen presence, and ultimate health risk. While *Escherichia coli* in drinking water is correlated with increased risk of diarrhoea,¹¹ FIB can also originate from non-faecal sources¹² and generally correlate poorly with pathogens in the environment.¹³ In addition, FIB are found in both human and animal faeces, and their detection in the environment cannot differentiate the source of contamination.^{11,12,14}

Applications of advanced molecular methods to environmental sampling in low-income settings can offer advantages over FIB measurements in characterising environmental contamination. These methods can directly detect a range of enteropathogens,^{15,16} and microbial source tracking (MST) methods aim to distinguish between human and animal faecal sources through detection of unique molecular characteristics of faecal microorganisms strongly associated with specific animal hosts.¹⁷ We assessed the effect of WASH interventions in low-income countries on enteropathogens and MST markers in the domestic environment with a systematic review and individual participant data (IPD) meta-analysis, which allows combining observation-level data from studies with standardised statistical methods.

Methods

Search strategy and selection criteria

We did a systematic review and individual participant data meta-analysis on the effects of WASH interventions on enteropathogens and MST markers in environmental samples. We searched PubMed, Embase, CAB Direct Global Health, Agricultural and Environmental Science Database, Web of Science, and Scopus (appendix pp 13–14). We included studies meeting the following inclusion criteria: (1) prospective studies with a water, sanitation, or hygiene intervention and concurrent control (ie, randomised controlled trial, matched cohort, controlled before-and-after study), consistent with previous WHO burden of disease reviews;^{18,19} (2) measured pathogens or MST markers in environmental samples; and (3) measured child anthropometry, diarrhoea, or pathogen-specific infections. We restricted the search to studies measuring child health outcomes to estimate associations between environmental contamination and child health in a separate analysis.²⁰ We included studies published from Jan 1, 2000, to Jan 5, 2023, to reflect recent advances in laboratory methods but we did not limit our search to any specific method (eg, molecular, culture-based, and microscopy). We excluded studies that only measured FIB. We limited our search to studies in English. Using Covidence systematic review software, one reviewer (AM) screened abstracts, and two independent reviewers (AM and RT) examined the full texts of short-listed articles with differences resolved with a third reviewer (AE). One reviewer (AM) forward and backward searched the citations of included articles (ie, checked the bibliographies of included studies and the studies

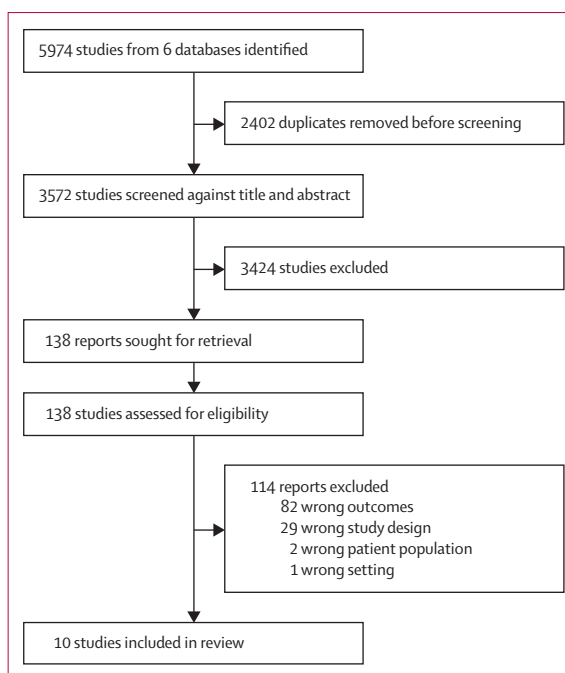


Figure 1: Study selection

that cited the included studies). We followed PRISMA reporting guidelines (figure 1, appendix pp 15–20). We used an adapted version of the Newcastle-Ottawa scale to evaluate bias (appendix pp 21–23).²¹

Data collection and analysis

For each eligible study, we requested individual sample data from authors, and excluded studies in which data were not shared. Before sharing data, all personal identifiers such as GPS locations were removed, and indirect identifiers such as sampling dates were coarsened to a monthly resolution.

We did not prespecify specific enteropathogens or markers as outcomes because each study measured a different set of targets. We used the pathogens and MST markers measured in the included studies to generate two composite measures as our primary outcomes: (1) detection of any pathogenic target or (2) any MST target, in any sample type collected during the same sampling round from the same compound, where a compound was defined by the original studies as a set of households with common courtyards, water sources, or latrines. Because many targets were infrequently detected in individual studies, composite outcomes allowed us to pool information from studies that focused on different targets and sample types, leveraging the IPD approach for increased statistical precision. We also analysed the prevalence of any pathogen and any MST marker separately for each sample type (eg, water, hand rinses, soil, and flies). Secondary outcomes included the prevalence of specific pathogen class (any viruses, any bacteria, any protozoa,

See Online for appendix

any helminths), the prevalence of MST markers from specific host types (human or animal), and the prevalence and abundance of individual enteropathogens and MST markers. We excluded general MST markers that are not host-specific from our analysis.

We compared outcomes between the intervention and control groups of each study. We estimated prevalence ratios (PRs) using modified Poisson regressions.²² For abundance outcomes, we used linear regressions to estimate differences in \log_{10} -transformed gene copies and negative binomial regressions to estimate ratios of soil-transmitted helminth (STH) egg counts. Because of repeated sampling or clustered designs in some studies, we used the Huber Sandwich Estimator to calculate robust standard errors using either the randomisation cluster or compound as the unit of independence.²³ For abundance measures, we imputed values below the limit of detection (LOD) with half the LOD, and values below the limit of quantification (LOQ) with the midpoint between the LOD and LOQ using LOD and LOQ values supplied by data contributors. We limited our analysis of abundance measures to targets in which more than 50% of samples were within the range of quantification (ROQ).

Although estimated intervention effects from randomised trials should be unconfounded, covariate adjustment might increase statistical efficiency and improve exchangeability with matched cohorts and non-randomised trials.²⁴ Therefore, we adjusted estimates for the treatment group and potential confounders chosen via a prescreening procedure. A prespecified set of potential confounding covariates (ie, potential predictors of each outcome) was prescreened using bivariate likelihood ratio tests, and those associated with the outcome with a p value less than 0.2 were included in the model for each outcome. As a sensitivity analysis, we also used LASSO penalised regressions to select adjustment covariates. We prescreened the following variables if they were measured within an included study: number of people in the household, age and education of primary caregiver, asset-based household wealth, number of rooms, construction materials (eg, walls, floor, and roof), access to electricity, land ownership, and if anyone in the household works in agriculture. These variables reflect sociodemographic conditions that are commonly considered potential confounders in WASH studies. When analysing binary outcomes, we only included one potential confounder per ten positive samples, or per ten negative samples if less than 50% of samples were negative, as per recommended best practices for the numbers of events per variable.²⁵ We did not estimate prevalence ratios for targets with less than five positive or negative values for a given sample type. We did a complete case analysis for missing outcomes. For continuous covariates, we excluded any with more than 50% missingness, and for categorical covariates, we used a missing category. Given the heterogeneity across studies (eg, local WASH conditions, climate, urbanisation, population density, regional infectious disease patterns,

and intervention type), we first individually estimated study-specific effects, and then pooled the effect estimates across studies using random-effects models, which allow for heterogeneity in intervention effects by assuming study-specific estimates come from a normal distribution of true effects.²⁵ Estimates were pooled when outcome data were available for four or more studies and were fit using restricted-maximum likelihood with the metafor package (version 3.0-2) in R.²⁶ We did not pool abundance estimates because of issues in standardising quantitative PCR (qPCR) methods across sites and the small number of available abundance estimates.^{27,28}

We did subgroup analyses by season (dry vs wet), animal ownership (at least one vs no animal owned) and pathogens (with vs without zoonotic transmission). The wet season for each study was defined as the 6 months of highest country-level average rainfall.²⁸ Reported animal ownership was intended as an indicator for the potential presence of animal faecal contamination; we note that this is an imperfect proxy and there is no standardised metric for capturing the likelihood of animal faecal contamination in the domestic setting.²⁹ The pathogens we considered as potentially zoonotic were *Campylobacter jejuni*, *Campylobacter coli*, *Salmonella*, *Yersinia enterocolitica*, *Clostridioides difficile*, *Cryptosporidium*, *Giardia*, and *Ascaris*.³⁰ We classified *Ascaris* as potentially zoonotic because *Ascaris lumbricoides* and *Ascaris suum* cross-infect humans and pigs, and the microscopy methods used in the studies in our review do not distinguish between them.^{30–32} When studies detected virulence genes associated with specific *E coli* pathotypes (ie, enteroaggregative *E coli*, enteropathogenic and enterohemorrhagic *E coli*, Shiga toxin-producing *E coli*, enteroinvasive *E coli*, and enterotoxigenic *E coli*), we classified Shiga toxin-producing *E coli* and enteropathogenic *E coli* (due to atypical enteropathogenic *E coli*) as zoonotic.³⁰ We used linear regression models estimating prevalence differences to assess additive interaction by examining the p values on the interaction terms between the treatment and the indicator variable for the subgroup; additive interaction has been argued to better capture public health importance than multiplicative interaction.³³

We also assessed heterogeneity by study-level characteristics, including setting, study design, intervention uptake and time between intervention onset, and environmental sampling. There was little heterogeneity in urbanicity within any individual study. Therefore, we pooled estimates separately for rural versus urban studies. We also separately pooled estimates from randomised versus quasi-experimental studies, studies with high versus low intervention uptake and studies with shorter (≤ 1 year) versus longer (> 1 year) follow-up between intervention onset and sampling. We compared pooled estimates between strata with Wald tests. Analyses were done in R version 4.0.4. Analysis scripts are publicly

	Parent study	Study design	Intervention	Time between intervention and environmental sampling	Location	Sample types	Targets	Analytic method	Number of samples
Capone et al (2022) ³⁷	About 2 years	..	Flies caught in latrine and kitchen	Panel of 16 enteric pathogens and MST markers	qPCR	86
Boehm et al (2016) ⁴⁰	WASH Benefits Bangladesh	Cluster-randomised trial	Latrine upgrades, child potties, and scoops for faeces disposal	4 months	Rural Bangladesh	Stored drinking water, child hands, and soil	Rotavirus, general, human, avian, and ruminant faecal markers	qPCR	1482
Holcomb et al (2021) ⁴¹	MapSan	Controlled before and after study	Latrine upgrades	About 1 year	Urban Mozambique	Source and stored water, household and latrine soil, and food	General, human, and avian faecal MST markers	qPCR	353
Odagiri et al (2016) ⁴²	Total Sanitation Campaign	Cluster-randomised trial	Latrine upgrades	About 1 year	Rural India	Source water	<i>V cholerae</i> , rotavirus, and adenovirus, and general, human, and animal faecal markers	qPCR, microscopy	60
Reese et al (2017) ⁴³	Gram Vikas	Matched cohort study	Latrine upgrades and piped water	6–10 years	Rural India	Source and stored water	<i>Vibrio cholerae</i> , Shigella	Slide agglutination serotyping	3452
Kwong et al (2021) ⁴⁴	About 2 years	..	Courtyard soil	Soil-transmitted helminths	Microscopy	1396
Steinbaum et al (2019) ⁴⁵	WASH Benefits Kenya	Cluster-randomised trial	Latrine upgrades, child's potties, and scoops for faeces disposal	About 2 years	Rural Kenya	Courtyard soil	Soil-transmitted helminths	Microscopy	2149
Fuhrmeister et al (2020) ⁴⁶	16–35 months	..	Stored drinking water, children's and mothers' hands, and soil	Pathogenic <i>Escherichia coli</i> , norovirus, Giardia	qPCR	2601
Capone et al (2021) ⁴⁷	About 1 year	..	Household and latrine soil	Panel of 18 enteric pathogens	qPCR	88

qPCR=quantitative PCR. MST=microbial source tracking.

Table 1: Characteristics of included publications

available (<https://github.com/amertens/wash-ipd>). Our systematic review search strategy and statistical analysis plan were preregistered and are available on Open Science Framework

Role of the funding source

The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

Results

The systematic review was done on Jan 19, 2021, and returned 3572 results after removing duplicates. Of these, 3424 were excluded by abstract screening, and of 138 short-listed studies, ten were eligible after full-text screening. The ten articles reported environmental assessments from six unique intervention studies: the WASH Benefits Bangladesh (WBB)³ and WASH Benefits Kenya (WBK) trials,⁴ the MapSan study in Mozambique,³⁴ the Gram Vikas study in India,³⁵ the Total Sanitation Campaign (TSC) trial in India,⁶ and the CHoBI7 trial in

Bangladesh³⁶ (table 1). Data were obtained from all studies except CHoBI7 in which individual participant data were not shared; this trial was excluded from our analysis. For MapSan, additional data were shared from an analysis unpublished at the time of the search.³⁷ For the TSC trial,⁶ only data on village-level source water quality were available. For the WBB and MapSan trials, multiple substudies within the trials collected samples from different subsets of participants at different times; therefore, we report the results of individual publications separately rather than combined by trial.

All studies assessed WASH intervention effects on diarrhoeal disease and growth in children younger than 5 years. Three studies were cluster-randomised controlled trials (WBB, WBK, and TSC). MapSan was a controlled before-and-after study with control and intervention sites matched on compound size and time of enrolment. Gram Vikas was a matched cohort study in which control and intervention villages were matched on 12 preintervention WASH and socioeconomic characteristics. With the Newcastle-Ottawa scale, studies

For more on the search strategy and statistical analysis plan see <https://osf.io/8sgzn/>.

had low risk of bias due to blinded outcome assessments, with the Gram Vikas and MapSan studies having a lower rating due to higher loss to follow-up and lack of randomisation (appendix pp 21–23). WBB, WBK, TSC, and Gram Vikas were done in rural settings, and MapSan in an urban setting. All included studies evaluated sanitation interventions (table 1). TSC and MapSan focused on sanitation alone. The WBB and WBK trials included individual and combined WASH and nutrition interventions but pathogens and MST markers in environmental samples were only measured in the sanitation and control groups. The Gram Vikas study evaluated a combined piped drinking water and sanitation intervention. The CHoBI7 trial (excluded because no data were shared) evaluated a combined water treatment and hygiene intervention. No included studies evaluated drinking water supply and treatment or hygiene interventions alone.

All sanitation interventions evaluated were onsite (ie, non-sewered) technologies delivered at the household or community level. None of the interventions met the Sustainable Development Goal standard of safely managed sanitation because no intervention included excreta treatment or offsite removal, and they would be classified as basic sanitation (or limited sanitation if participants shared latrines with neighbours, such as in the MapSan trial). The WASH Benefits studies provided new or upgraded improved latrines for each household in enrolled compounds, child potties and sani-scoops for faeces removal. In the WBB trial, latrines were dual-pit latrines with a water seal, and in WBK plastic latrine slabs were used to improve existing latrines. MapSan provided pour-flush latrines draining to septic tanks, shared by multiple households. TSC promoted construction of a pour-flush latrine with a single pit and Y-joint for a second pit, subsidised post hoc by government funding. In the Gram Vikas study, a non-governmental organisation provided materials for the construction of pour-flush latrines in each household in selected villages and built community water tanks and piped distribution systems providing household connections. When every household in the village completed latrine construction, the water system was turned on for the whole village.

Latrine access and use was higher in intervention households than control households in all studies. Definitions of latrine quality varied, including improved, clean, hygienic, or functional latrines, or latrines with a functional water seal, as observed by field staff. In four studies, 78–97% of intervention recipients had access to these types of facilities, compared with 18–45% of controls.^{3,4,35,38} The TSC trial had the lowest effect on latrine access, with 38% of intervention compounds having functional latrines compared with 10% of controls.⁶ Latrine use in intervention households was variable and especially low among children, and safe management of child and animal faeces was uncommon. In WBB, 94% of adults were observed to defecate in a hygienic

latrine in structured observations but only 54% of children were observed using the latrine or potty and 15% of animal faeces were observed to be removed with the sani-scoop.³⁹ In WBK, reported safe disposal of child faeces dropped from 77% 1 year after intervention to 37% after 2 years.⁴ In TSC, 50% of households reported children using a latrine,⁶ and in Gram Vikas, 35% of intervention villages reported disposing of child faeces in improved latrines.³⁵

Environmental samples were collected from 4 months⁴⁰ to 6–10 years³⁵ after intervention delivery, with most studies collecting samples 1–2 years after intervention (table 1). Sample types included source and stored drinking water, children's and mothers' hand rinses, soil from the courtyard, household and latrine areas, and flies caught in latrines and kitchens. Food samples were collected in one study⁴¹ but were not included in our analysis because only nine samples were positive for MST targets. The number of samples in individual studies varied from 60⁴² to 2107.³⁵ Our pooled dataset included 12 184 samples, with 40 156 observations for pathogen or MST marker prevalence.

The studies measured a range of bacterial, viral, protozoan, and helminthic pathogens, including pathogenic *E coli*, *Vibrio cholerae*, *Shigella*, *C jejuni*, *C coli*, *Salmonella*, *Yersinia*, *C difficile*, rotavirus, norovirus, sapovirus, adenovirus, astrovirus, enterovirus, *Cryptosporidium*, *Giardia*, *Entamoeba histolytica*, *A lumbricoides*, and *Trichuris trichiura* (appendix pp 25–38). The MST markers included human (HumM2, HF183, BacHum, *Methanobrevibacter smithii*), animal (BacCan, BacCow), ruminant (BacR), and avian (GFD) faecal markers (appendix pp 25–38). Most studies used qPCR or RT-qPCR (table 1). One study used slide agglutination serotyping to detect *V cholerae* and *Shigella*.⁴³ One study detected *Cryptosporidium* oocysts and *Giardia* cysts with direct fluorescent antibody microscopy.⁴² Two studies enumerated STH eggs by microscopy.^{44,45}

Many targets had low or no variation. Of 267 unique combinations of study, sample type, and target, 18 had no positive values, 41 had less than ten positive values, and two had less than ten negative values. Therefore, 206 of 267 sample-target combinations had sufficient variability to estimate a PR and be individually included in our IPD analysis. Among these, pathogen prevalence ranged from 7 (1.4%) of 496 for *Giardia* on mothers' hands⁴⁶ to 886 (62.1%) of 1426 for *Ascaris* in soil,⁴⁴ and the prevalence of MST markers ranged from 12 (2.4%) of 493 for HumM2 on children's hands⁴⁰ to 356 (97.5%) of 365 for BacCow on mothers' hands.⁴⁶

Interventions decreased the prevalence of any pathogen in any sample type in most individual studies but confidence intervals for PRs often crossed the null (figure 2). Among individual sample types, pathogen prevalence was most markedly reduced in flies in Capone and colleagues (adjusted PR 0.37 [95% CI 0.16–0.85]; figure 2).³⁷ Pooled across studies, there was a small reduction in the prevalence of any

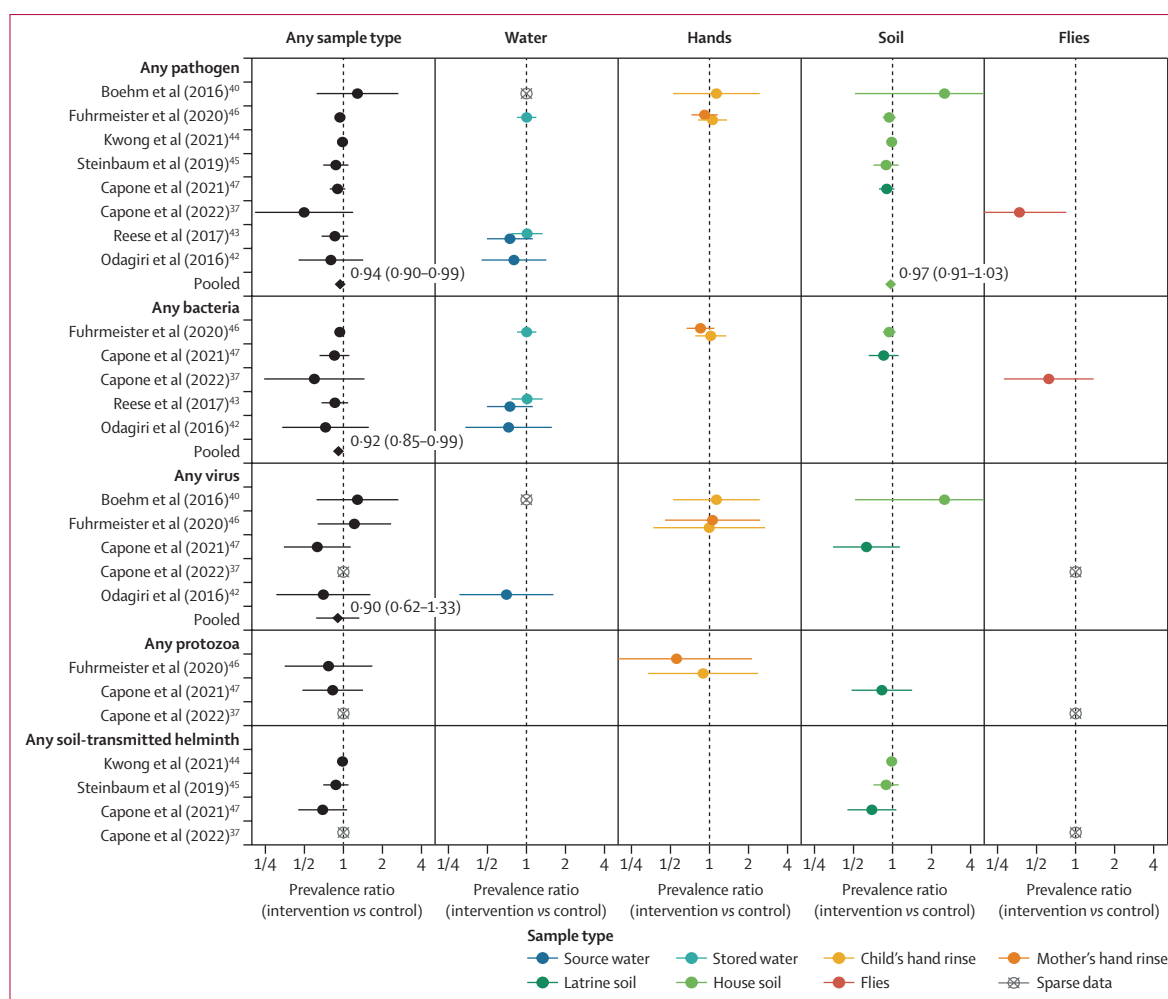


Figure 2: Forest plots of water, sanitation, and hygiene intervention effects on the prevalence of any enteropathogen or type of enteropathogen (any bacteria, virus, protozoa, or soil-transmitted helminth) in different types of environmental samples

Pooled estimates are presented when there are four or more study-specific estimates for a specific sample type and target combination and are denoted with diamond-shaped points. Grey crossed points denote data that were too sparse to estimate a prevalence ratio (ie, less than ten positive observations). Samples of the same type from different locations (source vs stored water, flies in kitchen vs latrine, and soil from courtyard vs latrine) or different individuals (children’s vs mothers’ hands) are plotted separately. Point estimates and confidence intervals are given next to pooled estimates. Study-specific effects are independently estimated for each individual study, adjusting for potential confounders, and then pooled across studies with random effects models.

pathogen detected in any sample type (pooled adjusted PR 0.94 [95% CI 0.90–0.99]; figure 2). Intervention effects on MST markers were inconsistent and mostly small or null across all sample types, with a null pooled effect (1.01 [0.98–1.04]; figure 3).

Interventions reduced the prevalence of any bacterial pathogens in any sample type (pooled adjusted PR 0.92 [95% CI 0.85–0.99]) and the intervention effects were protective in all individual studies, although with varying precision (figure 2). Interventions did not reduce virus prevalence in any sample type, with a pooled adjusted PR of 0.90 (0.62–1.33) and inconsistent directions of effects across individual studies, or reduce virus prevalence within specific sample types (figure 2). Intervention effects in individual studies were generally in the protective direction for protozoa and helminths but we did not have sufficient

studies to pool estimates. Among specific pathogens, interventions had the strongest effects on the prevalence of adenovirus (0.21 [0.06–0.68]) and *Shigella* (0.28 [0.10–0.78]) in any sample type in Capone and colleagues,⁴⁷ driven by reductions in soil around latrines (appendix p 2).

Overall, intervention effects on MST markers were negligible, with no detected effects on both human markers (pooled adjusted PR 1.00 [95% CI 0.88–1.13]) and animal markers (1.00 [0.97–1.03]; figure 3, appendix p 3). Interventions had the largest effects on any animal marker in stored water in Boehm and colleagues⁴⁰ (adjusted PR 0.69 [95% CI 0.50–0.95]; figure 3), driven by a reduction in the ruminant BacR marker (0.62 [0.43–0.90]; appendix p 3), and on the human marker HF183 in any sample type (0.67 [0.48–0.95]; appendix p 3) in Holcomb and colleagues.⁴¹

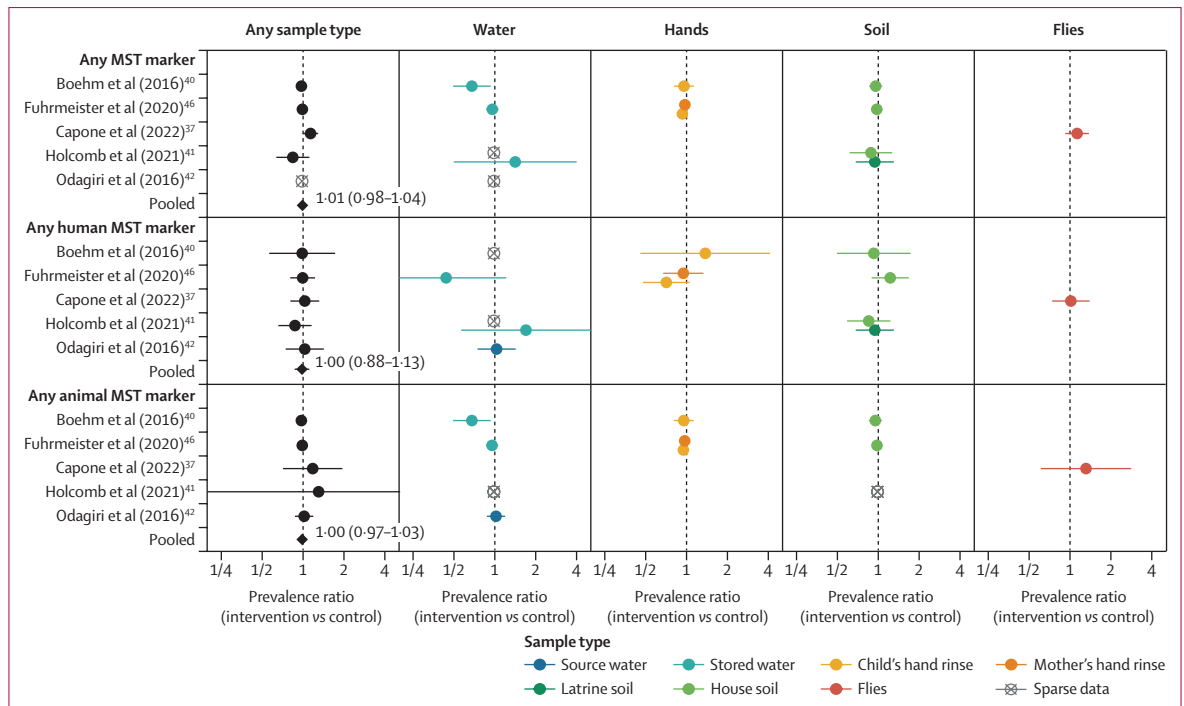


Figure 3: Forest plots of water, sanitation, and hygiene intervention effects on the prevalence of any MST marker or type of MST marker (human or animal MST markers) in different types of environmental samples

Pooled estimates are presented when there are four or more study-specific estimates for a specific sample type and target combination and are denoted with diamond-shaped points. Grey crossed points denote data that were too sparse to estimate a prevalence ratio (ie, less than ten positive observations). Samples of the same type from different locations (source vs stored water, flies in kitchen vs latrine, and soil from courtyard vs latrine) or different individuals (children’s vs mothers’ hands) are plotted separately. Point estimates and confidence intervals are given next to pooled estimates. Study-specific effects are independently estimated for each individual study, adjusting for potential confounders, and then pooled across studies with random effects models. MST=microbial source tracking.

Of all observations, 20125 (50%) of 40224 had abundances quantified, including STH egg counts^{44,45} and gene copies of enteropathogens and MST targets^{37,40,46} (appendix pp 4–5). Of these, 5377 (27%) of 20187 were below the specific study-reported LOD, 5875 (29%) of 20187 below the study-reported LOQ, and 8884 (44%) of 20187 within the study-reported ROQ. Of targets enumerated within specific sample types, only 8 (18%) of 45 had more than 50% of samples within the ROQ and were included in our analysis. The abundance of the BacCow animal marker was lower in mothers’ hand rinses in the sanitation intervention group in Fuhrmeister and colleagues,⁴⁶ with an adjusted log₁₀-transformed difference of -0.28 (95% CI -0.49 to -0.07) per pair of hands (table 2). The interventions had inconsistent effects on the abundance of other MST targets and STH egg counts, with confidence intervals crossing the null (table 2).

Intervention effects differed by season, but the direction of effects was inconsistent (appendix pp 5–6). Animal ownership was high (>80%) in all studies except Gram Vikas (appendix pp 39–42), but there were no consistent differences in intervention effects when households were stratified by animal presence (appendix p 7), and no differences in intervention effects on pathogens with possible zoonotic transmission versus only human hosts (appendix p 8). In Wald tests, there

were no significant differences in pooled estimates between the one urban study (MapSan) and the four rural studies (p=0.25), between randomised and quasi-experimental studies (p=0.43), between studies with 1 year or less and more than 1 year of follow-up (p=0.51) or between the four studies with high latrine access among intervention recipients compared with the TSC trial with lower access (p=0.57). Adjustment covariates were measured differently across studies, but most had low missingness when measured (appendix pp 39–42). Unadjusted and adjusted estimates were similar (appendix pp 10–11), and results were also similar with bootstrapped LASSO penalised regression models to select adjustment covariates (appendix p 12).

Discussion

Our IPD analysis of five intervention studies, mostly focused on household-level and community-level onsite sanitation improvements, indicates a small overall reduction in pathogen prevalence in the environment associated with the interventions. Although the effects of interventions on pathogen prevalence within individual studies had variable precision, point estimates of intervention effects were consistently in the protective direction across studies when aggregated across pathogen and sample types, despite differences in

	Sample	Target	Sample	Range of quantification	Control	Intervention	Intervention effect (95% CI)	p value	Wilcoxon p value*
Capone et al (2022) ³⁷	Latrine soil	Human (BacHum)	173	77.5%	3.8 (1.3), 3.8 (2.4-4.5)	4.0 (0.9), 4.2 (3.4-4.6)	0.14 (-0.19 to 0.47)	0.41	0.07
Holcomb et al (2021) ⁴¹	Latrine soil	Human (<i>Methanobrevibacter smithii</i>)	113	51.3%	6.7 (0.6), 6.5 (6.2-7.0)	6.5 (0.5), 6.3 (6.3-6.6)	-0.14 (-0.38 to 0.11)	0.27	0.58
Kwong et al (2021) ⁴⁴	House soil	<i>Ascaris</i>	1426	100.0%	2.3 (6.7), 0.7 (0.0-2.5)	2.2 (6.9), 0.6 (0.0-2.3)	0.97 (0.68 to 1.38)†	0.85	0.54
Kwong et al (2021) ⁴⁴	House soil	<i>Trichuris</i>	1426	100.0%	1.6 (5.0), 0.4 (0.0-1.8)	2.0 (5.0), 0.4 (0.0-2.2)	1.22 (0.87 to 1.71)†	0.26	0.17
Steinbaum et al (2019) ⁴⁵	House soil	<i>Ascaris</i>	2101	100.0%	2.2 (18.8), 0	1.4 (9.3), 0	0.65 (0.33 to 1.28)†	0.21	0.33
Steinbaum et al (2019) ⁴⁵	House soil	<i>Trichuris</i>	2102	100.0%	0.2 (1.8), 0	0.2 (1.0), 0	0.73 (0.36 to 1.48)†	0.38	0.39
Fuhrmeister et al (2020) ⁴⁶	Child's hand rinse	Animal (BacCow)	365	75.9%	3.6 (1.4), 3.9 (3.5-4.4)	3.4 (1.4), 3.8 (1.2-4.2)	-0.17 (-0.47 to 0.12)	0.25	0.17
Fuhrmeister et al (2020) ⁴⁶	Mother's hand rinse	Animal (BacCow)	725	66.5%	3.3 (1.4), 3.8 (1.2-4.3)	3.0 (1.5), 3.7 (1.2-4.1)	-0.28 (-0.49 to -0.07)	0.01	0.01

Data are n, %, mean (SD), and median (IQR). Means are log₁₀-transformed gene copies for MST markers and mean egg counts for soil transmitted helminths (*Ascaris* and *Trichuris*). Intervention effects are shown as adjusted differences in log₁₀-transformed gene copies and ratios of helminth egg counts between the intervention and control arms. MST=microbial source tracking. *Non-parametric Wilcoxon rank sum test p value. †Marks ratio estimates from negative binomial model.

Table 2: Mean abundances of enteropathogen and MST targets by study group

setting, intervention design, and length of follow-up. In general, intervention effects were less consistent when disaggregated by pathogen and sample type, with confidence intervals overlapping with the null (figure 2), but interventions were consistently protective against bacterial pathogens and for source water, and the MapSan study consistently showed protective effects. Interventions generally did not reduce the prevalence of human or animal faecal markers in included studies (figure 3).

These findings add to a body of literature on the effectiveness of sanitation improvements in low-income countries in interrupting faecal-oral transmission. A previous systematic review found no effect of sanitation interventions on FIB in the environment.⁷ The small, pooled effect on pathogens in the environment in our analysis indicates that any reductions in pathogen transmission through environmental pathways was most likely small. This can help explain the null findings of the parent trials on child diarrhoea.^{3,4,6,34,35} Among the five included studies, only WASH Benefits Bangladesh found a significant reduction in diarrhoea³ and a reduction in parasite infections^{48,49} in the sanitation group compared with the controls. Diarrhoea was reduced by 2.2 percentage points on the absolute scale, compatible with a small reduction in pathogen transmission. Taken together, these findings indicate that the sanitation interventions in the studies in our review did not sufficiently isolate faecal waste from the environment, despite most of them achieving high levels of latrine access and use by adults. Young children's faeces are a dominant source of faecal contamination in the household environment,⁵⁰ whereas animal faeces make up the majority of global faecal waste⁵¹ and are associated

with increased domestic contamination.⁵² Therefore, containment of adult human waste might be insufficient to reduce environmental contamination in settings with continued child open defecation and high exposure to animal waste.⁵³ Only the WASH Benefits trials included tools for child and animal faeces management (potties and scoops).^{39,54} Notably, in two studies nested within WASH Benefits Bangladesh, we found reduced prevalence of ruminant (BacR) markers in stored water and reduced abundance of animal markers (BacCow) on mothers' hands (appendix p 3). Also, the sanitation intervention in this trial only reduced pathogen prevalence in households with animals (appendix p 7). The reduction in animal faecal contamination can help explain the unique health effects in this trial. In our analysis, only the MapSan study had a reduction in a human (HF183) marker. More comprehensive sanitation programmes, such as safely managed sanitation services that include safe removal in addition to containment of faecal waste, and interventions targeting child and animal faeces, can potentially more effectively interrupt environmental pathogen transmission.⁵⁵

It is possible that current approaches to environmental sampling have affected the ability to detect intervention effects on pathogen presence in the environment. Faecal contamination in the domestic environment varies spatially and temporally,^{56,57} and pathogen presence in the environment is intermittent, depending on the presence of infected individuals, shedding rates and pathogen fate and survival in environmental reservoirs.⁵⁸ Different pathogens have different predominant transmission pathways, and specific pathogens might cause illness through a particular pathway too infrequently to capture

with cross-sectional grab samples. Additionally, pathogen prevalence and abundance in the environment is typically low,⁵⁸ and in some cases too rare to estimate the effects of interventions. Any reductions in pathogen presence might be more apparent with larger sample sizes or repeated sampling with high temporal and spatial resolution, which is costly for available pathogen detection methods, or by analysing larger quantities of composite samples. In addition, human MST markers have low specificity and sensitivity in settings with widespread faecal contamination in the environment.^{28,59} Also, molecular methods for pathogen detection do not provide information on viability, and the clinical implications of small amounts of pathogen DNA or RNA detected in a sample are unclear. Although FIB have limitations in terms of low specificity to faecal sources and poor correlation with pathogens, culture-based FIB enumeration captures viable organisms, and large numbers of temporal or spatial samples can be analysed at low cost. Therefore, studies evaluating the environmental effect of WASH interventions can benefit from combining molecular pathogen measurements with culture-based FIB measurements to leverage the different strengths of these approaches. Pathogen-specific testing can supplement FIB data to identify the specific causes through which WASH interventions improve health or the effects of targeted interventions on specific pathogens. Advances in technology that reduce the costs of molecular diagnostics or increased funding for environmental testing within WASH trials might allow broader use of pathogen detection methods to estimate intervention effects on environmental contamination more precisely.

Our analysis had some strengths and limitations. The IPD meta-analysis allowed us to estimate intervention effects across studies with consistent statistical methodology, variable definitions, and covariate selection.⁶⁰ The individual studies in our review were designed and powered to detect effects on child health, and samples were collected and analysed to detect pathogens and MST markers among smaller subsets of study households. Pooling effect estimates across studies increased our statistical power to detect a small overall effect on pathogens that individual studies were underpowered to detect. However, pooling assumes that individual studies are sufficiently homogeneous despite implementing different interventions in different settings. Therefore, pooled estimates should be interpreted in conjunction with estimates from individual studies. In our analysis, we detected no statistical heterogeneity between studies, and low-precision estimates from individual studies were qualitatively aligned with high-precision pooled estimates, suggesting that pooling data did not obscure any study-specific trends. Similarly, because studies measured different targets in different environmental matrices and many targets were detected infrequently,

we relied on composite measures, such as detection of any pathogen in any sample type, to allow pooling effect estimates across studies. These measures combine data on different pathogens in different sample types and obscure nuances on which specific pathogens along which pathways are influenced by interventions. Therefore, effects on these composite outcomes should be interpreted in tandem with pathogen-specific and pathway-specific estimates. Studies assessing the effects of WASH interventions on environmental contamination should strive to have sample sizes that allow estimating intervention effects on pathogen prevalence and abundance in environmental matrices with precision, informed by a priori power calculations. Standardised measurement and reporting of a harmonised panel of enteropathogens in a consistent set of environmental matrices can also allow better comparability of pathogen-specific data for future IPD meta-analyses.¹⁵

Only a small number of studies met our inclusion criteria, limiting the generalisability of our findings. Three of the studies were from rural southeast Asia, with a defined monsoon season coinciding with peak diarrhoeal disease incidence, one from rural Kenya with two distinct rainy seasons, and one from drier and urban Maputo, Mozambique. This is consistent with previous WASH reviews indicating that Bangladesh, India, and Kenya are well represented in the published literature, and evidence from other settings is scarce.⁶¹ Four of the included studies focused on onsite sanitation and one evaluated a combined piped water and sanitation intervention. Therefore, we were unable to explore the effects of individual water supply and treatment and hygiene interventions, and more comprehensive sanitation modalities that safely managed sanitation services and sewer connections. The one study identified by the systematic review, but in which data were not shared, was a combined handwashing and water treatment intervention that reduced *V cholerae* in stored water but not source water.³⁷ This is in contrast to the null effects of the Gram Vikas and TSC interventions on *V cholerae* in drinking water, and including this study in our IPD analysis would have contributed an additional effect estimate in which the intervention reduced pathogen prevalence. However, the study was done in high-risk populations (household contacts of cholera patients) and is not directly comparable to the studies included in our analysis that focused on general paediatric populations. Additionally, although the studies tested a diverse set of sample types, including understudied pathogen reservoirs, such as soil, not all pathways were captured. For example, contaminated food has been identified as a dominant pathogen transmission pathway in previous research,⁶² but only one study in our review sampled food and none of the three that tested MST markers were detected above our data sparsity cutoff to estimate treatment effects.

The basic sanitation interventions in our review resulted in a small reduction in the environmental presence of enteropathogens, consistent with the previously reported scarce health effects. Our results suggest that these sanitation interventions failed to contain human waste and thus prevent exposure to enteropathogens in these populations. More comprehensive approaches are needed to catalyse major health gains. Countries that have universal access to effective sanitation have seen remarkable improvements in health.⁶³ Public health programmes in low-income countries should pursue transformative WASH approaches that encompass the full chain of excreta management, including safe removal rather than mere containment and address child and animal faeces to interrupt environmental pathogen transmission more effectively. Also, our review indicates a scarcity of water supply, water quality, and hygiene trials that measured pathogens in the environment. Future studies should assess the effect of such interventions on environmental contamination by measuring a harmonised set of pathogens with adequately powered sample sizes and by measuring FIB more frequently in time and space to capture variability, and include understudied pathways, such as soil, food, and flies.

Contributors

AM, BFA, OC, JMC, and AE contributed to the conceptualisation of the study. OC and JMC acquired the funding. AM, DC, EF, JK, LK, AJP, HR, and AE contributed to the data curation. AM and RT did the formal analyses. AM, BFA, JBC, RT, and AE contributed to the methodology. AM contributed to the visualisation of the study. AM and AE contributed to the writing of the original draft. AM, BFA, JBC, ABB, JB, DC, TC, EF, JAG, DH, JK, LK, AL, SPL, RN, KN, SMN, CN, AJP, MH, HER, LS, JS, RT, OC, JMC, and AE contributed to the review and editing of the manuscript. AM and AE verified the data. All authors had access to the data and AM, OC, JMC, and AE were responsible for the decision to submit the manuscript.

Declaration of interests

JER reports that a portion of her salary is supported by an unrestricted donation to The London School of Hygiene & Tropical Medicine from Reckitt; the salary support is wholly unrelated to her role in the preparation of this manuscript. TC has interest in publicly traded companies. All other authors declare no competing interests.

Data sharing

The de-identified, aggregated individual participant data and data dictionary can be shared by request of investigators and after approval by the original trial data contributors. Data inquiries can be directed to Andrew Mertens at amertens@berkeley.edu. The statistical analysis code is available at <https://github.com/amertens/wash-ipd>.

Acknowledgments

This work was supported in part by grants from the Bill & Melinda Gates Foundation (grant number OPP1137224) and the UK Foreign and Commonwealth Development Office (grant number GB-1-114396). We thank all the participants and their families who took part in the included studies.

References

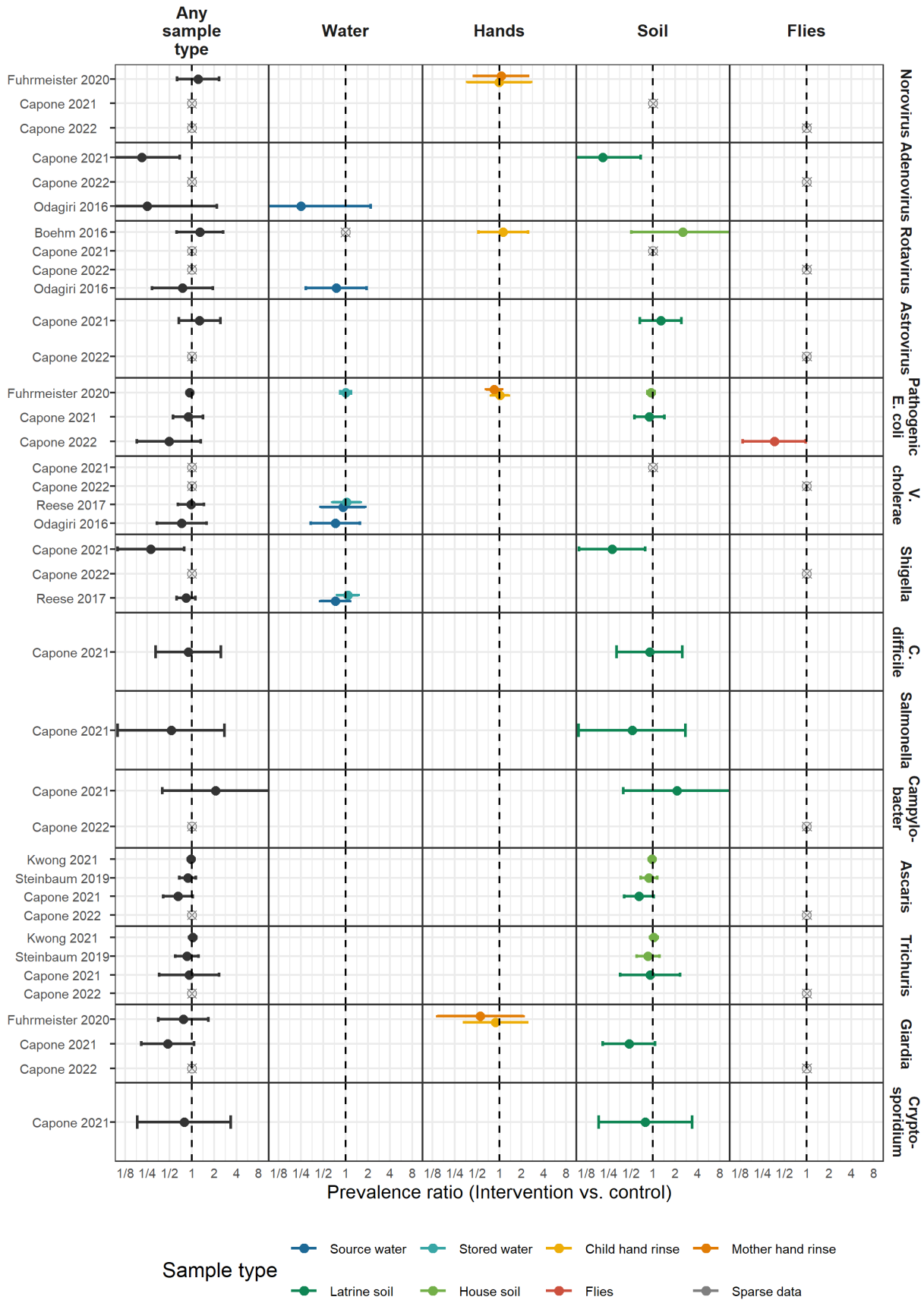
- GBD Diarrhoeal Diseases Collaborators. Estimates of global, regional, and national morbidity, mortality, and aetiologies of diarrhoeal diseases: a systematic analysis for the Global Burden of Disease Study 2015. *Lancet Infect Dis* 2017; **17**: 909–48.
- Humphrey JH, Mbuya MNN, Ntozini R, et al. Independent and combined effects of improved water, sanitation, and hygiene, and improved complementary feeding, on child stunting and anaemia in rural Zimbabwe: a cluster-randomised trial. *Lancet Glob Health* 2019; **7**: e132–47.
- Luby SP, Rahman M, Arnold BF, et al. Effects of water quality, sanitation, handwashing, and nutritional interventions on diarrhoea and child growth in rural Bangladesh: a cluster randomised controlled trial. *Lancet Glob Health* 2018; **6**: e302–15.
- Null C, Stewart CP, Pickering AJ, et al. Effects of water quality, sanitation, handwashing, and nutritional interventions on diarrhoea and child growth in rural Kenya: a cluster-randomised controlled trial. *Lancet Glob Health* 2018; **6**: e316–29.
- Knee J, Sumner T, Adriano Z, et al. Effects of an urban sanitation intervention on childhood enteric infection and diarrhoea in Maputo, Mozambique: a controlled before-and-after trial. *eLife* 2021; **10**: e62278.
- Clasen T, Boisson S, Routray P, et al. Effectiveness of a rural sanitation programme on diarrhoea, soil-transmitted helminth infection, and child malnutrition in Odisha, India: a cluster-randomised trial. *Lancet Glob Health* 2014; **2**: e645–53.
- Sclar GD, Penakalapati G, Amato HK, et al. Assessing the impact of sanitation on indicators of fecal exposure along principal transmission pathways: a systematic review. *Int J Hyg Environ Health* 2016; **219**: 709–23.
- Arnold BF, Colford JM Jr. Treating water with chlorine at point-of-use to improve water quality and reduce child diarrhoea in developing countries: a systematic review and meta-analysis. *Am J Trop Med Hyg* 2007; **76**: 354–64.
- Luby SP, Agboatwalla M, Raza A, et al. Microbiologic effectiveness of hand washing with soap in an urban squatter settlement, Karachi, Pakistan. *Epidemiol Infect* 2001; **127**: 237–44.
- Pickering AJ, Boehm AB, Mwanjali M, Davis J. Efficacy of waterless hand hygiene compared with handwashing with soap: a field study in Dar es Salaam, Tanzania. *Am J Trop Med Hyg* 2010; **82**: 270–78.
- Gruber JS, Ercumen A, Colford JM Jr. Coliform bacteria as indicators of diarrhoeal risk in household drinking water: systematic review and meta-analysis. *PLoS One* 2014; **9**: e107429.
- Devane ML, Moriarty E, Weaver L, Cookson A, Gilpin B. Fecal indicator bacteria from environmental sources; strategies for identification to improve water quality monitoring. *Water Res* 2020; **185**: 116204.
- Wu J, Long SC, Das D, Dorner SM. Are microbial indicators and pathogens correlated? A statistical analysis of 40 years of research. *J Water Health* 2011; **9**: 265–78.
- Hardina CM, Fujioka RS. Soil: the environmental source of *Escherichia coli* and enterococci in Hawaii's streams. *Environ Toxicol Water Qual* 1991; **6**: 185–95.
- Lappan R, Henry R, Chown SL, et al. Monitoring of diverse enteric pathogens across environmental and host reservoirs with TaqMan array cards and standard qPCR: a methodological comparison study. *Lancet Planet Health* 2021; **5**: e297–308.
- Liu J, Gratz J, Amour C, et al. A laboratory-developed TaqMan Array Card for simultaneous detection of 19 enteropathogens. *J Clin Microbiol* 2013; **51**: 472–80.
- Stoeckel DM, Harwood VJ. Performance, design, and analysis in microbial source tracking studies. *Appl Environ Microbiol* 2007; **73**: 2405–15.
- Wolf J, Hunter PR, Freeman MC, et al. Impact of drinking water, sanitation and handwashing with soap on childhood diarrhoeal disease: updated meta-analysis and meta-regression. *Trop Med Int Health* 2018; **23**: 508–25.
- Wolf J, Prüss-Ustün A, Cumming O, et al. Assessing the impact of drinking water and sanitation on diarrhoeal disease in low- and middle-income settings: systematic review and meta-regression. *Trop Med Int Health* 2014; **19**: 928–42.
- Mertens AN, Arnold BF, Benjamin-Chung J, et al. Associations between detection of enteropathogens and microbial source tracking markers in the environment and child enteric infections and growth: a systematic review and individual participant data meta-analysis. *SSRN* 2023; published online Jan 4. https://papers.ssrn.com/sol3/papers.cfm?abstract_id=4313717 (preprint).
- Wells GA, Shea B, O'Connell D, Peterson J, Welch V, Losos M, Tugwell P. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses. http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp (accessed May 8, 2022).
- Zou G. A modified poisson regression approach to prospective studies with binary data. *Am J Epidemiol* 2004; **159**: 702–06.
- Freedman DA. On The so-called "Huber Sandwich Estimator" and "Robust Standard Errors". *Am Stat* 2006; **60**: 299–302.

- 24 Cochran WG. The combination of estimates from different experiments. *Biometrics* 1954; **10**: 101–29.
- 25 Peduzzi P, Concato J, Kemper E, Holford TR, Feinstein AR. A simulation study of the number of events per variable in logistic regression analysis. *J Clin Epidemiol* 1996; **49**: 1373–79.
- 26 Viechtbauer W. Conducting meta-analyses in R with the metafor package. *J Stat Softw* 2010; **36**: 1–48.
- 27 Borchardt MA, Boehm AB, Salit M, Spencer SK, Wigginton KR, Noble RT. The environmental microbiology minimum information (EMMI) guidelines: qPCR and dPCR quality and reporting for environmental microbiology. *Environ Sci Technol* 2021; **55**: 10210–23.
- 28 Boehm AB, Van De Werfhorst LC, Griffith JF, et al. Performance of forty-one microbial source tracking methods: a twenty-seven lab evaluation study. *Water Res* 2013; **47**: 6812–28.
- 29 Lowe J, Ercumen A, Prottas C, Harris AR. Exploring the determinants and indicators of poultry feces management behaviors in rural Western Uganda. *Sci Total Environ* 2022; **834**: 155202.
- 30 Delahoy MJ, Wodnik B, McAliley L, et al. Pathogens transmitted in animal feces in low- and middle-income countries. *Int J Hyg Environ Health* 2018; **221**: 661–76.
- 31 Betson M, Nejsun P, Bendall RP, Deb RM, Stothard JR. Molecular epidemiology of ascariasis: a global perspective on the transmission dynamics of *Ascaris* in people and pigs. *J Infect Dis* 2014; **210**: 932–41.
- 32 Steinbaum L, Kwong LH, Ercumen A, et al. Detecting and enumerating soil-transmitted helminth eggs in soil: new method development and results from field testing in Kenya and Bangladesh. *PLoS Negl Trop Dis* 2017; **11**: e0005522.
- 33 VanderWeele TJ, Knol MJ. A tutorial on interaction. *Epidemiol Methods* 2014; **3**: 33–72.
- 34 Knee JS. Quantifying the impact of an urban onsite shared sanitation intervention on child health in Maputo, Mozambique: The Mapsan trial. July 25, 2019. <https://smartech.gatech.edu/handle/1853/63563> (accessed March 15, 2022).
- 35 Reese H, Routray P, Torondel B, et al. Assessing longer-term effectiveness of a combined household-level piped water and sanitation intervention on child diarrhoea, acute respiratory infection, soil-transmitted helminth infection and nutritional status: a matched cohort study in rural Odisha, India. *Int J Epidemiol* 2019; **48**: 1757–67.
- 36 Burrowes V, Perin J, Monira S, et al. Risk factors for household transmission of *Vibrio cholerae* in Dhaka, Bangladesh (CHOB17 Trial). *Am J Trop Med Hyg* 2017; **96**: 1382–87.
- 37 Capone D, Adriano Z, Cumming O, et al. Urban onsite sanitation upgrades and synanthropic flies in Maputo, Mozambique: effects on enteric pathogen infection risks. *Environ Sci Technol* 2022; **57**: 549–60.
- 38 Bick S, Buxton H, Chase RP, et al. Using path analysis to test theory of change: a quantitative process evaluation of the MapSan trial. *BMC Public Health* 2021; **21**: 1411.
- 39 Parvez SM, Azad R, Rahman M, et al. Achieving optimal technology and behavioral uptake of single and combined interventions of water, sanitation hygiene and nutrition, in an efficacy trial (WASH benefits) in rural Bangladesh. *Trials* 2018; **19**: 358.
- 40 Boehm AB, Wang D, Ercumen A, et al. Occurrence of host-associated fecal markers on child hands, household soil, and drinking water in rural Bangladeshi households. *Environ Sci Technol Lett* 2016; **3**: 393–98.
- 41 Holcomb DA, Knee J, Capone D, et al. Impacts of an urban sanitation intervention on fecal indicators and the prevalence of human fecal contamination in Mozambique. *Environ Sci Technol* 2021; **55**: 11667–79.
- 42 Odagiri M, Schriever A, Daniels ME, et al. Human fecal and pathogen exposure pathways in rural Indian villages and the effect of increased latrine coverage. *Water Res* 2016; **100**: 232–44.
- 43 Reese H. Effectiveness of a combined sanitation and household-level piped water intervention on infrastructure coverage, availability and use, environmental fecal contamination, and child health in rural Odisha, India: a matched cohort study. Nov 3, 2017 <https://etd.library.emory.edu/concern/etds/3t945q772?locale=en> (accessed June 21, 2021).
- 44 Kwong LH, Sen D, Islam S, et al. Effect of sanitation improvements on soil-transmitted helminth eggs in courtyard soil from rural Bangladesh: evidence from a cluster-randomized controlled trial. *PLoS Negl Trop Dis* 2021; **15**: e0008815.
- 45 Steinbaum L, Mboya J, Mahoney R, Njenga SM, Null C, Pickering AJ. Effect of a sanitation intervention on soil-transmitted helminth prevalence and concentration in household soil: a cluster-randomized controlled trial and risk factor analysis. *PLoS Negl Trop Dis* 2019; **13**: e0007180.
- 46 Fuhmeister ER, Ercumen A, Pickering AJ, et al. Effect of sanitation improvements on pathogens and microbial source tracking markers in the rural Bangladeshi household environment. *Environ Sci Technol* 2020; **54**: 4316–26.
- 47 Capone D, Berendes D, Cumming O, et al. Impact of an urban sanitation intervention on enteric pathogen detection in soils. *Environ Sci Technol* 2021; **55**: 9989–10000.
- 48 Lin A, Ali S, Arnold BF, et al. Effects of water, sanitation, handwashing, and nutritional interventions on environmental enteric dysfunction in young children: a cluster-randomized, controlled trial in rural Bangladesh. *Clin Infect Dis* 2020; **70**: 738–47.
- 49 Ercumen A, Benjamin-Chung J, Arnold BF, et al. Effects of water, sanitation, handwashing and nutritional interventions on soil-transmitted helminth infections in young children: a cluster-randomized controlled trial in rural Bangladesh. *PLoS Negl Trop Dis* 2019; **13**: e0007323.
- 50 Bauza V, Madadi V, Ocharo RM, Nguyen TH, Guest JS. Microbial source tracking using 16S rRNA amplicon sequencing identifies evidence of widespread contamination from young children's feces in an urban slum of Nairobi, Kenya. *Environ Sci Technol* 2019; **53**: 8271–81.
- 51 Berendes DM, Yang PJ, Lai A, Hu D, Brown J. Estimation of global recoverable human and animal faecal biomass. *Nat Sustain* 2018; **1**: 679–85.
- 52 Ercumen A, Pickering AJ, Kwong LH, et al. Animal feces contribute to domestic fecal contamination: evidence from *E. coli* measured in water, hands, food, flies, and soil in Bangladesh. *Environ Sci Technol* 2017; **51**: 8725–34.
- 53 Pickering AJ, Null C, Winch PJ, et al. The WASH Benefits and SHINE trials: interpretation of WASH intervention effects on linear growth and diarrhoea. *Lancet Glob Health* 2019; **7**: e1139–46.
- 54 Islam M, Rahman M, Unicomb L, et al. Assessing sustained uptake of latrine and child feces management interventions in rural Bangladesh. *PLoS One* 2022; published online July 20. <https://doi.org/10.1371/journal.pone.0236163>.
- 55 Amebelu A, Ban R, Bhagwan J, et al. The Lancet Commission on water, sanitation and hygiene, and health. *Lancet* 2021; **398**: 1469–70.
- 56 Levy K, Hubbard AE, Nelson KL, Eisenberg JNS. Drivers of water quality variability in northern coastal Ecuador. *Environ Sci Technol* 2009; **43**: 1788–97.
- 57 Ram PK, Jahid I, Halder AK, et al. Variability in hand contamination based on serial measurements: implications for assessment of hand-cleansing behavior and disease risk. *Am J Trop Med Hyg* 2011; **84**: 510–16.
- 58 Goddard FGB, Pickering AJ, Ercumen A, Brown J, Chang HH, Clasen T. Faecal contamination of the environment and child health: a systematic review and individual participant data meta-analysis. *Lancet Planet Health* 2020; **4**: e405–15.
- 59 Payment P, Locas A. Pathogens in water: value and limits of correlation with microbial indicators. *Ground Water* 2011; **49**: 4–11.
- 60 Holcomb DA, Stewart JR. Microbial indicators of fecal pollution: recent progress and challenges in assessing water quality. *Curr Environ Health Rep* 2020; **7**: 311–24.
- 61 Venkataramanan V, Collins SM, Clark KA, Yeam J, Nowakowski VG, Young SL. Coping strategies for individual and household-level water insecurity: a systematic review. *WIREs Water* 2020; **7**: e1477.
- 62 Amin N, Rahman M, Raj S, et al. Quantitative assessment of fecal contamination in multiple environmental sample types in urban communities in Dhaka, Bangladesh using SaniPath microbial approach. *PLoS One* 2019; **14**: e0221193.
- 63 Mackenbach JP. Sanitation: pragmatism works. *BMJ* 2007; **334** (suppl 1): s17–17.

1 **Effect of water, sanitation, and hygiene interventions**
2 **on detection of enteropathogens and host-specific fecal**
3 **markers in the environment: an individual-participant**
4 **data meta-analysis**

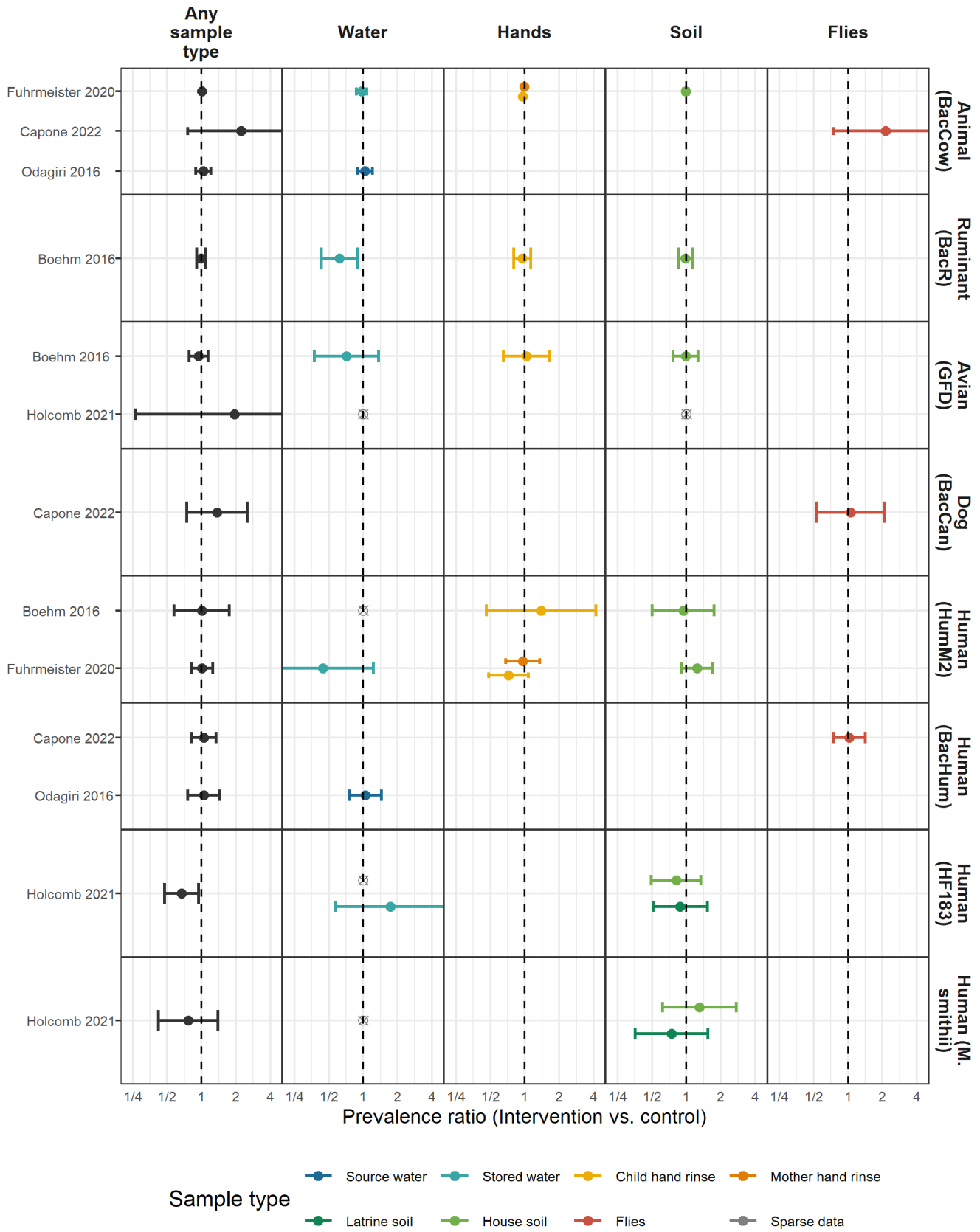
5 Andrew Mertens PhD, Benjamin F. Arnold PhD, Jade Benjamin-Chung PhD, Prof Alexandria B. Boehm PhD, Joe Brown
6 PhD, Drew Capone PhD, Prof Thomas Clasen PhD, Erica Fuhrmeister PhD, Jessica A. Grembi PhD, David Holcomb
7 PhD, Jackie Knee PhD, Laura H Kwong PhD, Audrie Lin PhD, Prof Stephen P. Luby MD, Rassul Nala MPH, Prof Kara
8 Nelson PhD, Sammy M. Njenga PhD, Clair Null PhD, Amy J. Pickering PhD, Mahbubur Rahman MBBS, Heather E. Reese
9 PhD, Lauren Steinbaum PhD, Prof Jill Stewart PhD, Ruwan Thilakaratne MPH, Oliver Cumming PhD, Prof John M.
10 Colford Jr., Ayse Ercumen PhD

11 **SUPPLEMENTARY FIGURES AND TABLES**
12



11

Figure S1. Forest plots of intervention effects on the prevalence of specific pathogens.

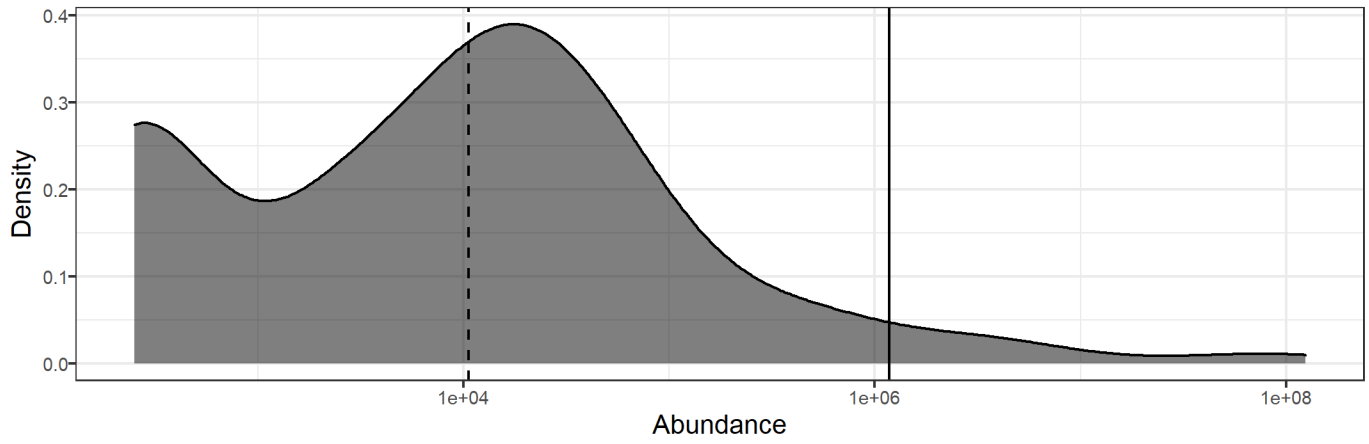


14

15 **Figure S2.** Forest plots of intervention effects on the prevalence of specific MST markers.

Fly

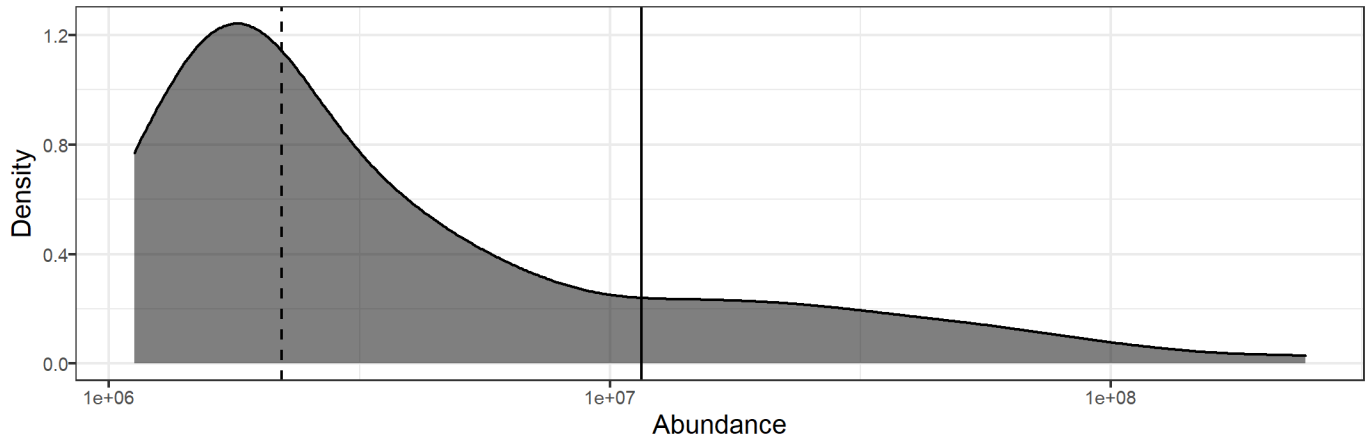
Human (BacHum)



16

LS

Human (M. smithii)



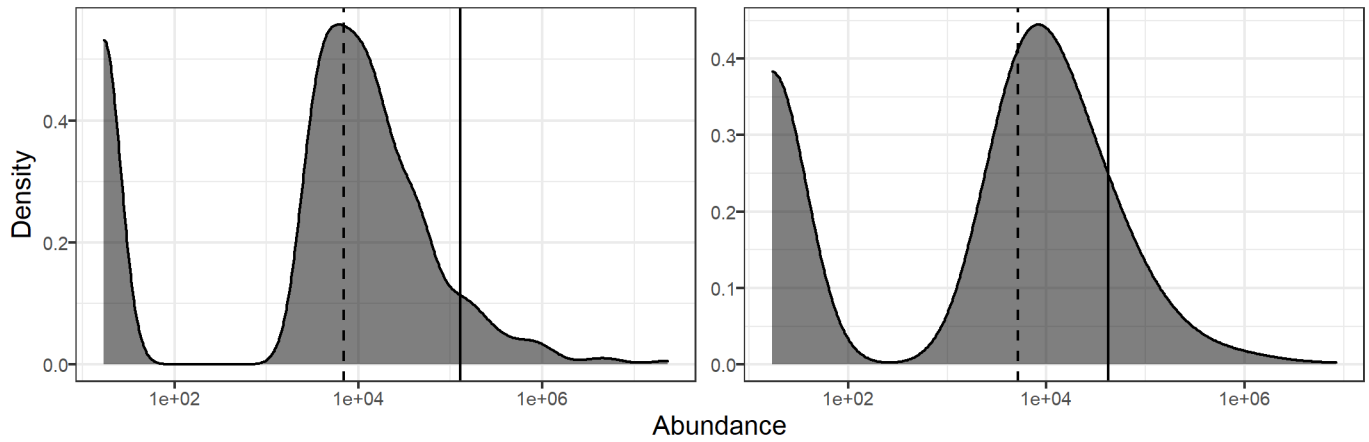
17

CH

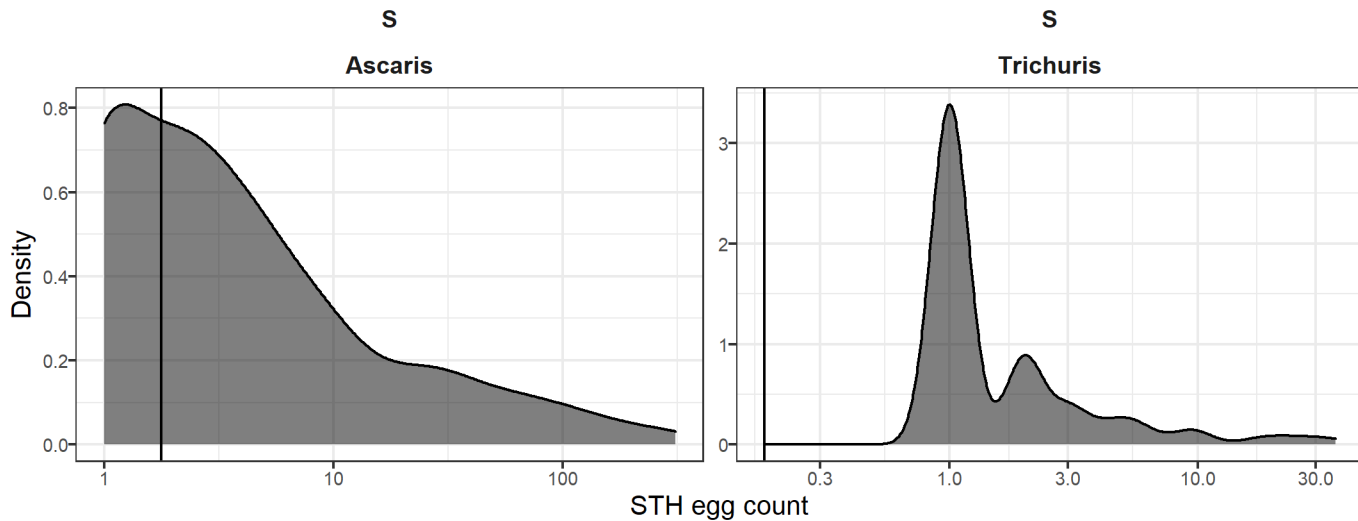
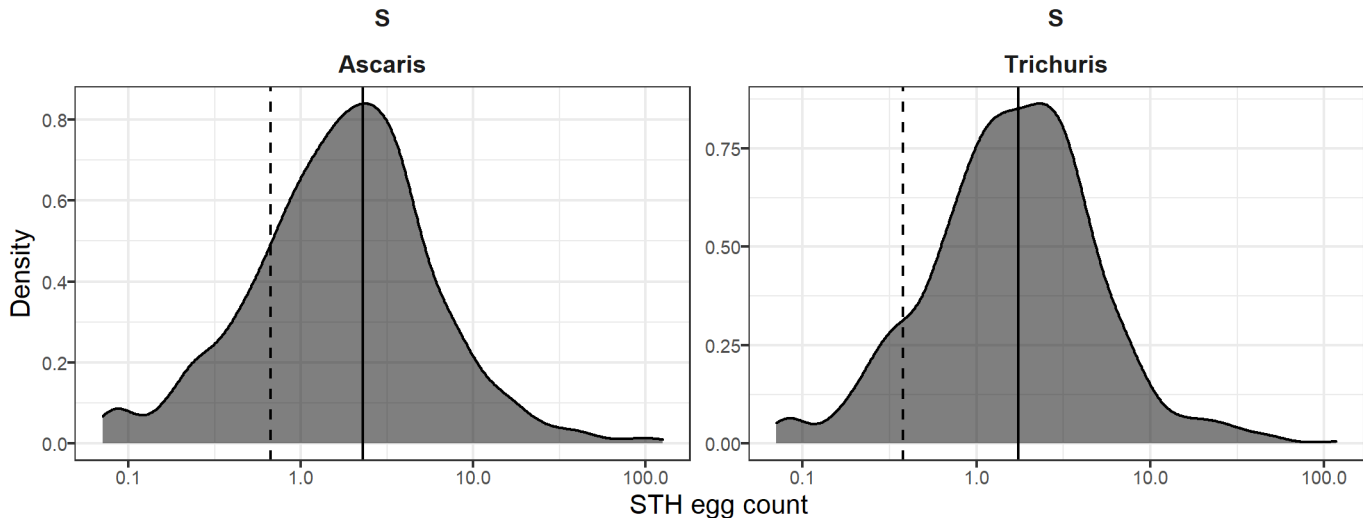
Animal (BacCow)

MH

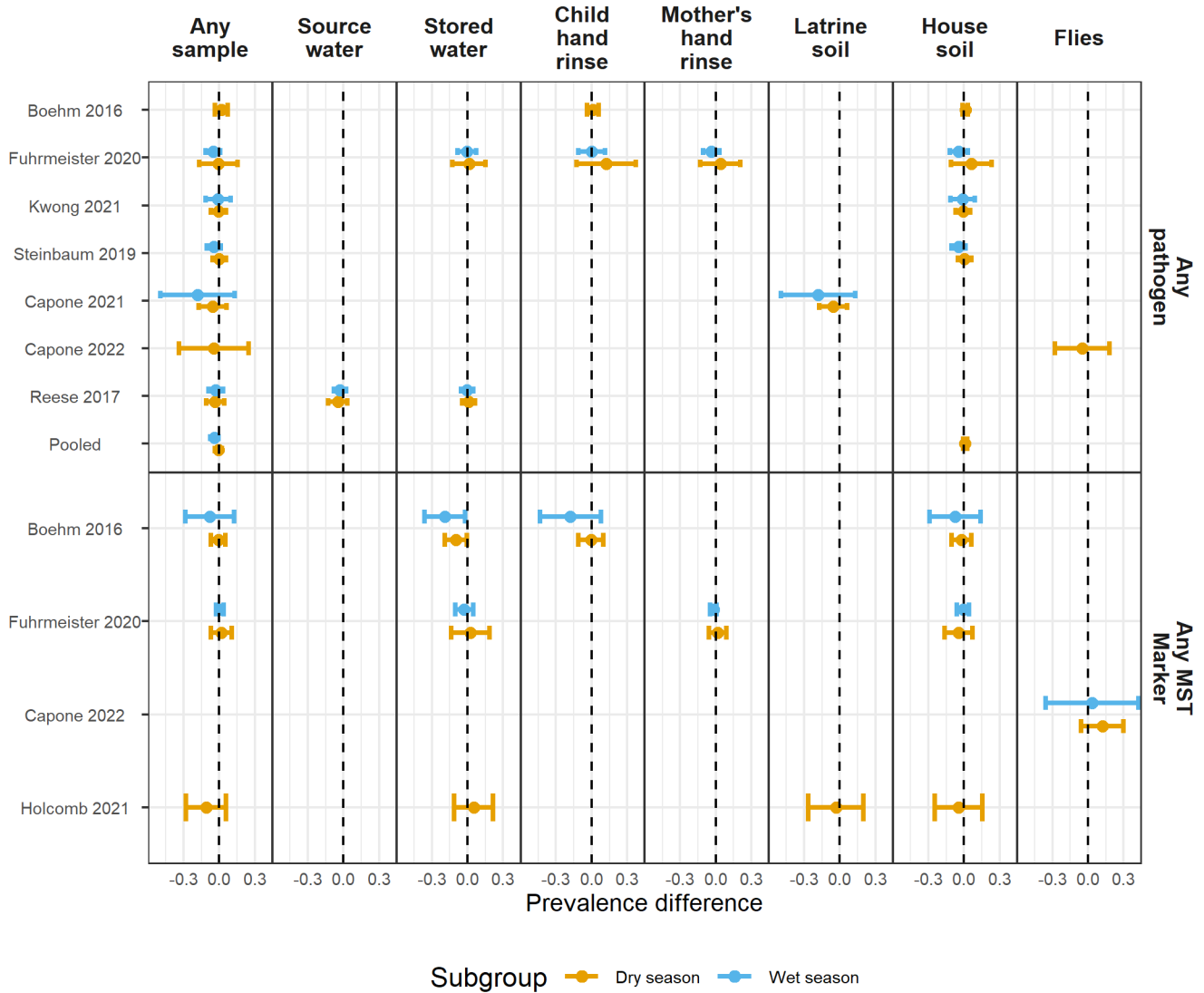
Animal (BacCow)



18

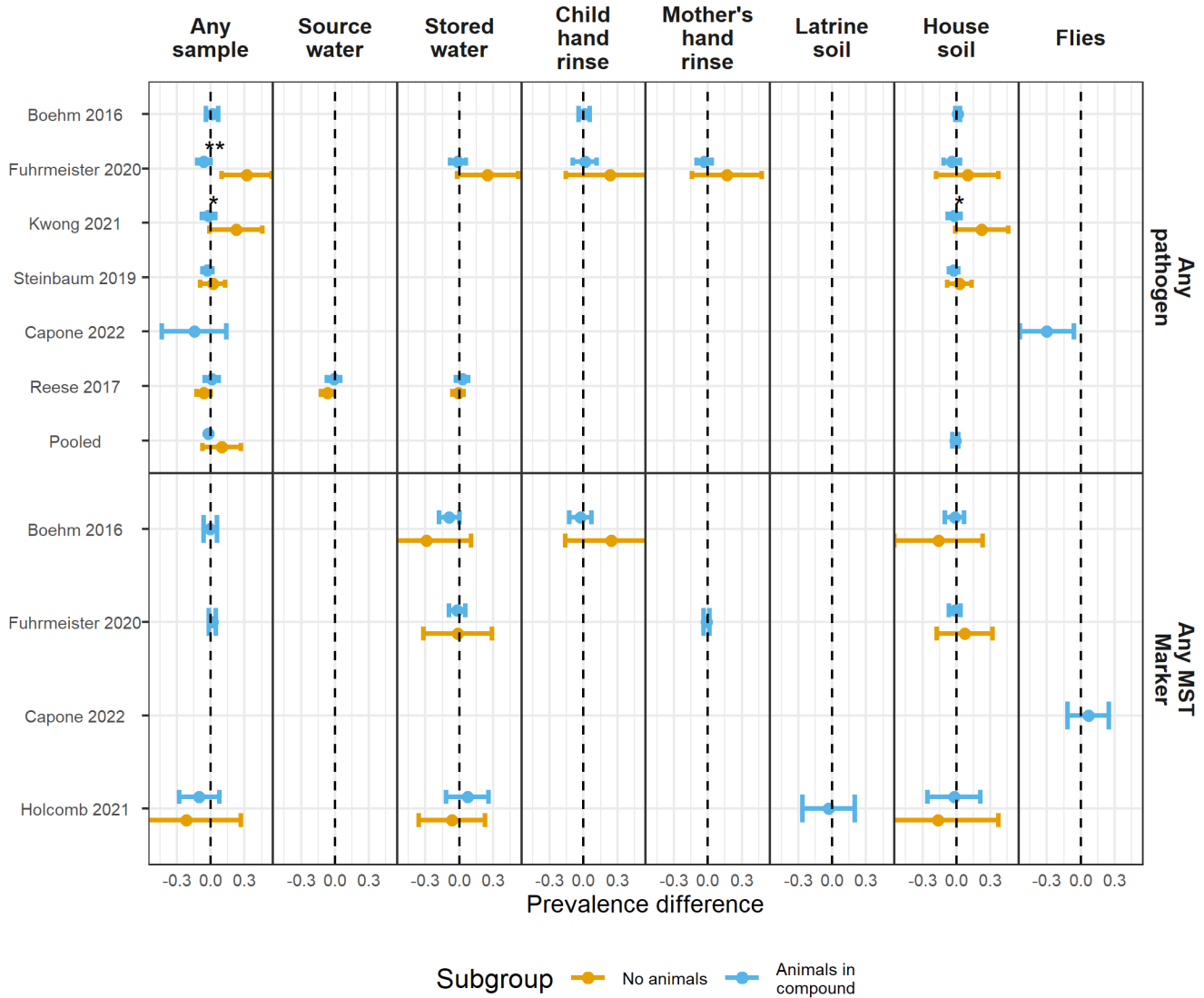


22 **Figure S3.** Distributions of abundance outcomes. The X-axes are displayed on the log-10 scale. Black vertical lines
 23 mark the means, and dashed lines mark the medians. Values below the limit of detection were imputed with with
 24 half the limit of detection and values below the limit of quantification were imputed with the midpoint between the
 25 limits of detections and quantification, leading to some bimodal distributions.



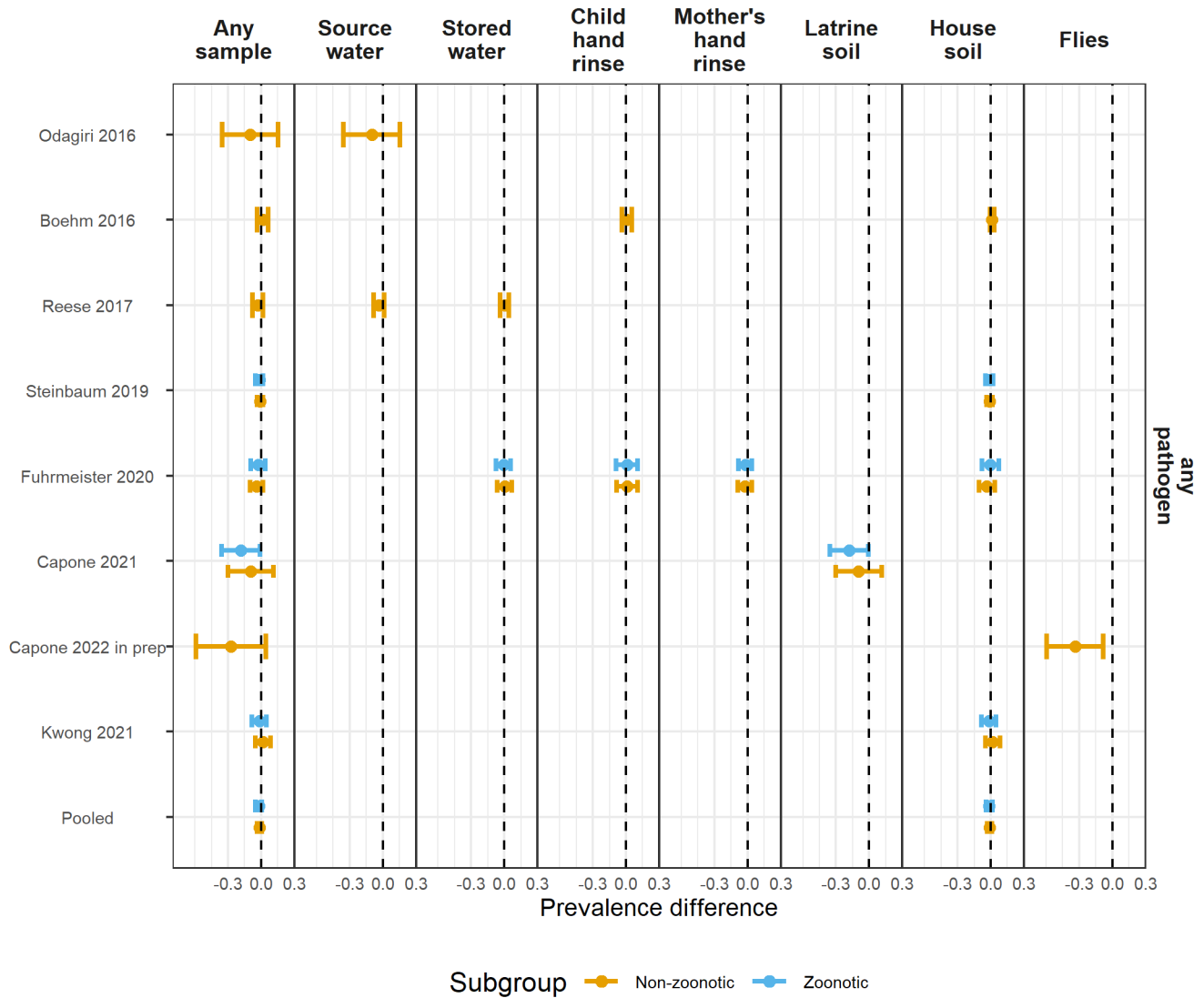
26

27 **Figure S4.** Forest plots of any enteropathogen prevalence differences or any MST prevalence differences between
 28 intervention and control arms, stratified by whether the sample was collected during the wet versus dry season
 29 (defined by the 6 months of highest average rainfall). Significant effect modification, as determined by the p-values
 30 on the regression model interaction term, is marked above points with asterisks (P < 0.05 = "*", P < 0.01 = "**", P <
 31 0.001 = "***"). Grey crossed points denote data that were too sparse to estimate a prevalence ratio (i.e., <10 positive
 32 observations).



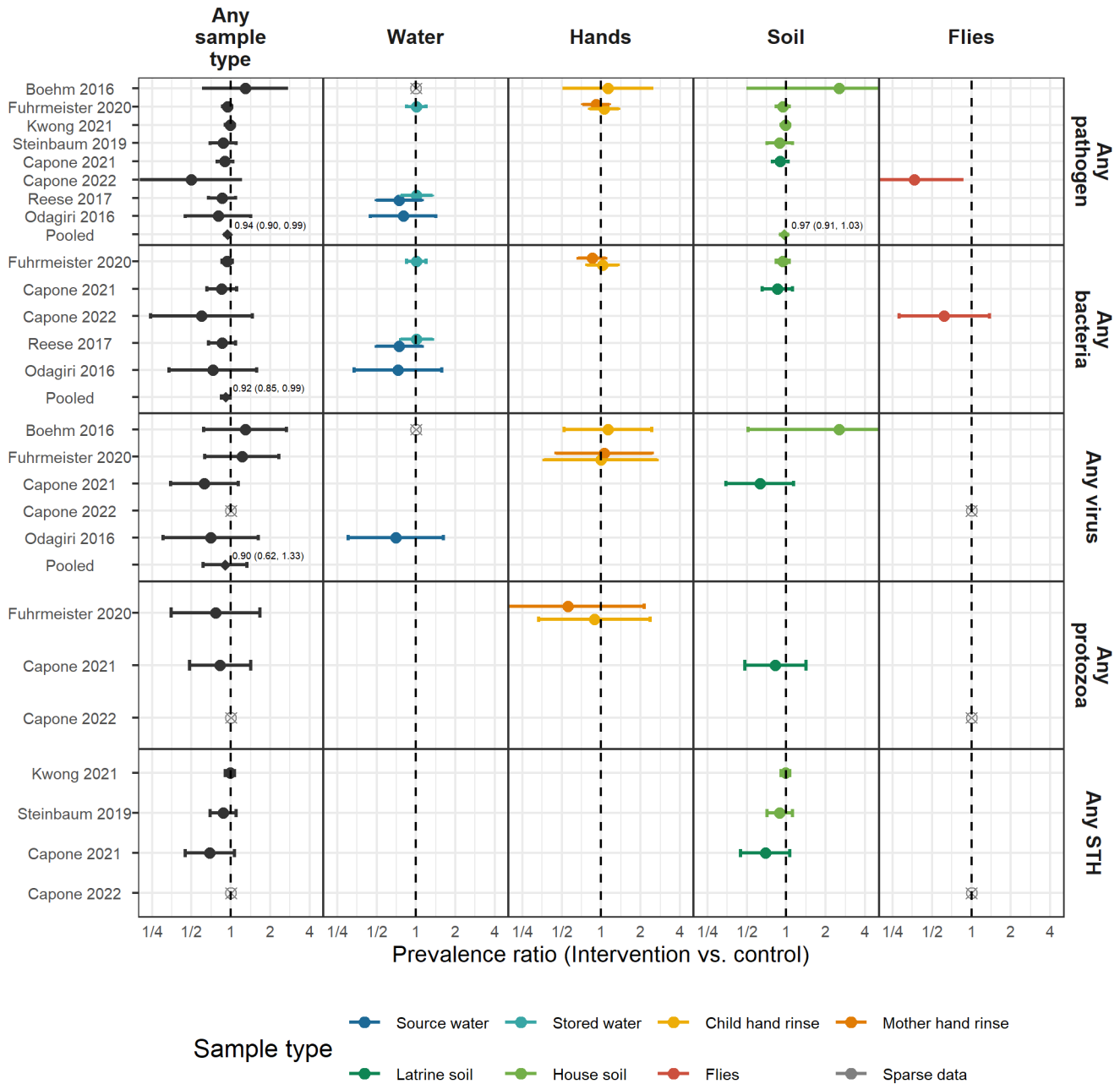
33

34 **Figure S5.** Forest plots of any enteropathogen prevalence differences or any MST prevalence differences between
 35 intervention and control arms, stratified by whether any animals were present in the compound. Significant effect
 36 modification, as determined by the p-values on the regression model interaction term, is marked above points with
 37 asterisks (P < 0.05 = "*", P < 0.01 = "**", P < 0.001 = "***"). Grey crossed points denote data that were too sparse to
 38 estimate a prevalence ratio (i.e., <10 positive observations).



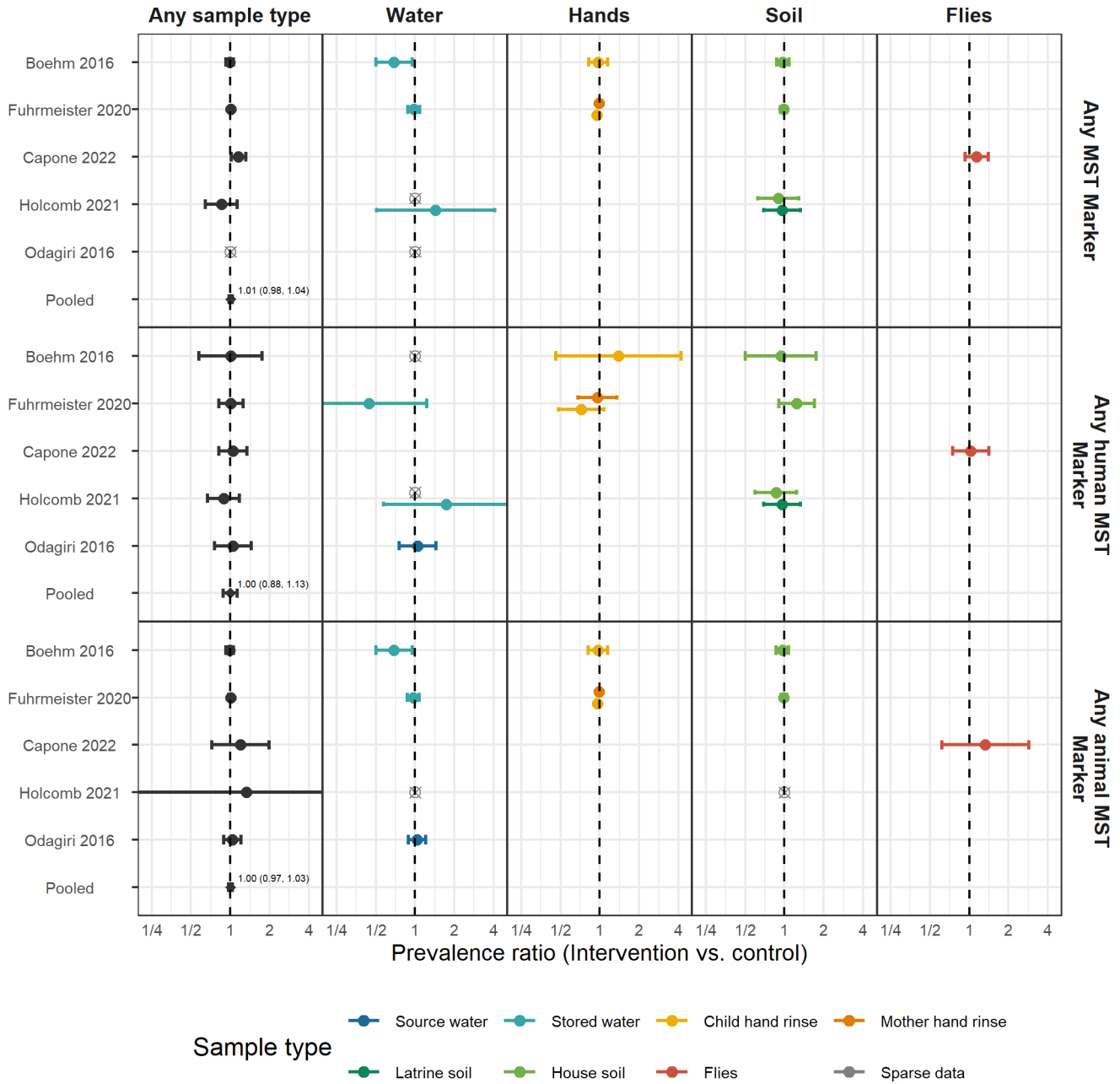
39

40 **Figure S6.** Forest plots of any enteropathogen prevalence differences or any MST prevalence differences between
 41 intervention and control arms, stratified by whether the pathogen is zoonotically transmitted. Grey crossed points
 42 denote data that were too sparse to estimate a prevalence ratio (i.e., <10 positive observations). Significant effect
 43 modification, as determined by the p-values on the regression model interaction term, is marked above points with
 44 asterisks (P < 0.05 = "*", P < 0.01 = "**", P < 0.001 = "***"). Grey crossed points denote data that were too sparse to
 45 estimate a prevalence ratio (i.e., <10 positive observations).



46

47 **Figure S7.** Forest plots of unadjusted intervention effects on the prevalence of any enteropathogen or type of
 48 enteropathogen (any bacteria, any virus, any protozoa and any STH) in different types of environmental samples.
 49 Point estimates and confidence intervals are printed next to pooled estimates. Grey crossed points denote data that
 50 were too sparse to estimate a prevalence ratio (i.e., <10 positive observations).

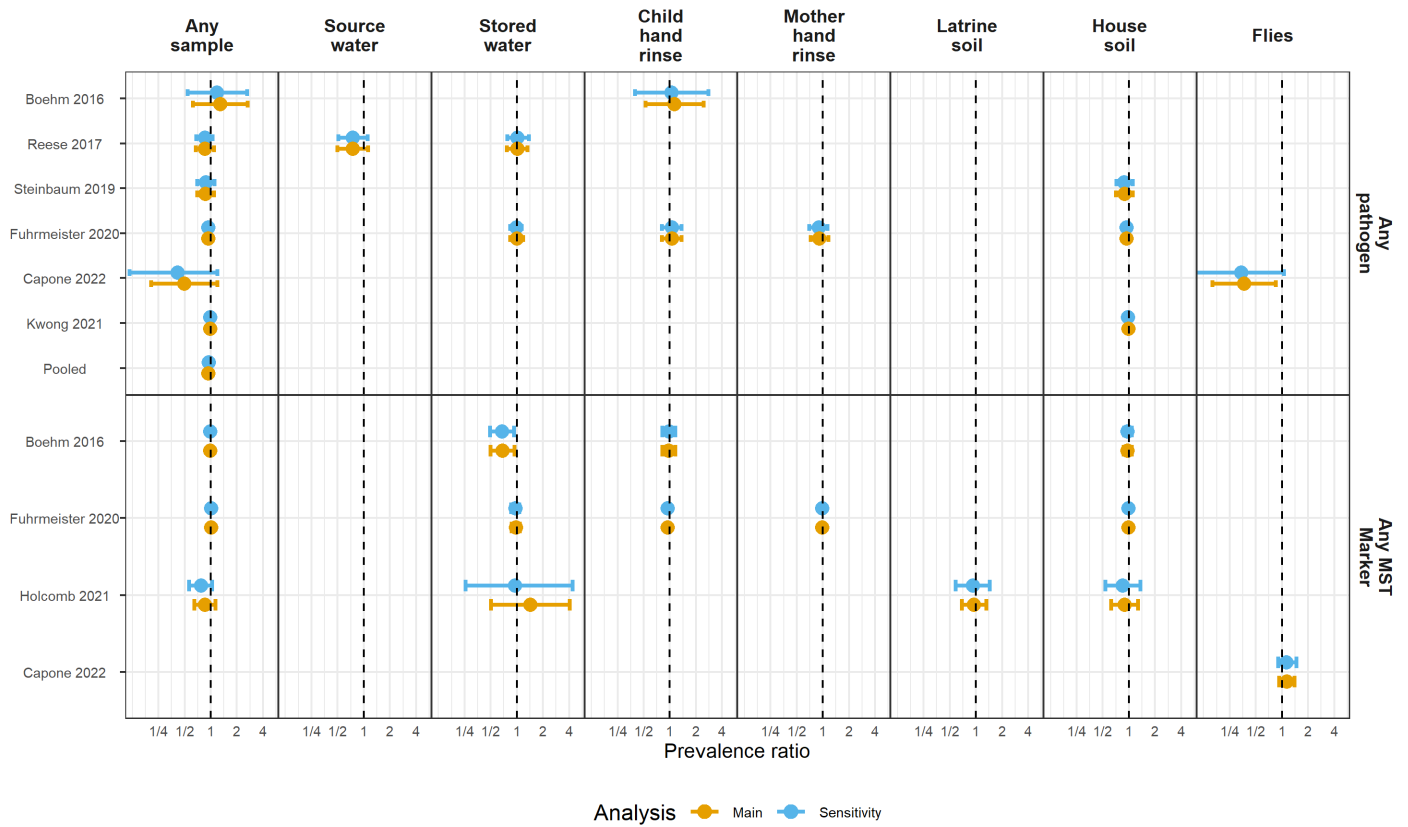


51

52 **Figure S8.** Forest plots of unadjusted intervention effects on the prevalence of any MST marker or type of MST
53 marker (human or animal MST markers) in different types of environmental samples. Point estimates and confidence
54 intervals are printed next to pooled estimates. Grey crossed points denote data that were too sparse to estimate a
55 prevalence ratio (i.e., <10 positive observations).

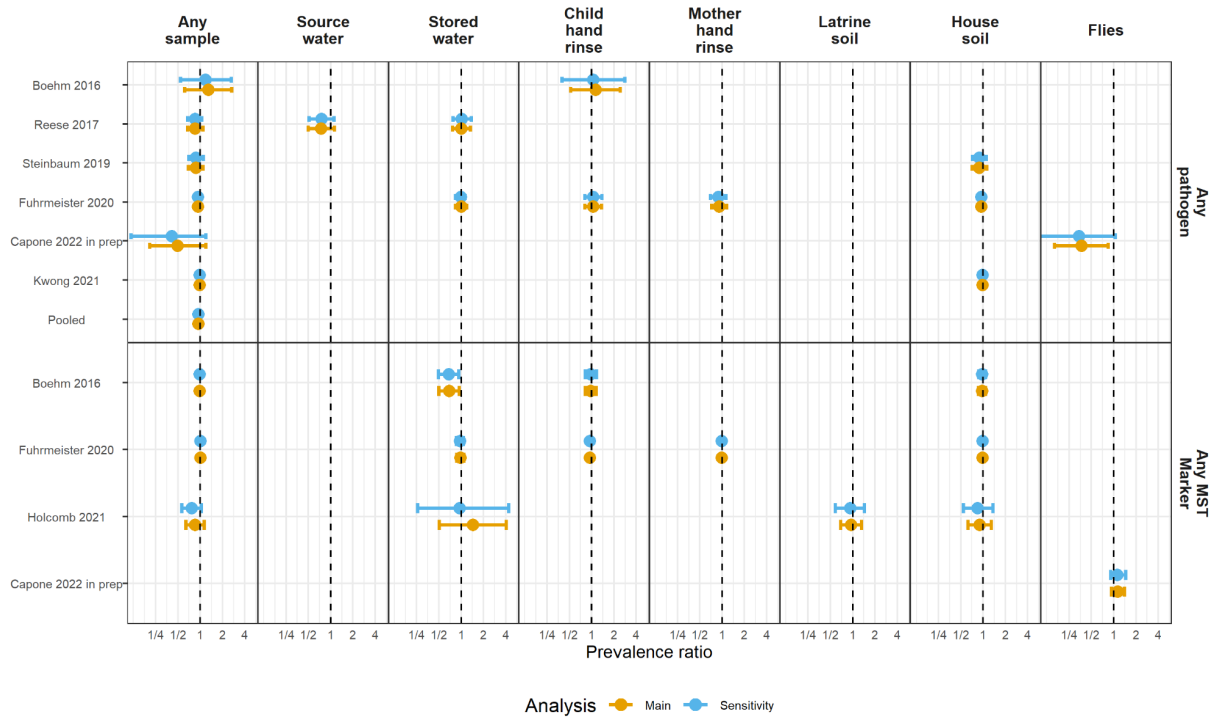
56

57



58

59 **Figure S9.** Forest plots of results from the primary analysis (orange) compared to a sensitivity analysis to the
60 covariate prescreening method (blue) for the outcomes of any pathogen detection (top row) and any MST marker
61 detection (bottom row) for different studies and sample types (columns). The sensitivity analysis used LASSO
62 penalized regressions to select covariates across 200 bootstrap iterations with replacement, with 95% confidence
63 intervals estimated with using the quantile method.



60

61

62

63

64

65

Figure S10. Forest plots of results from the primary analysis (orange) compared to a sensitivity analysis to the covariate prescreening method (blue) for the outcomes of any pathogen detection (top row) and any MST marker detection (bottom row) for different studies and sample types (columns). The sensitivity analysis used LASSO penalized regressions to select covariates across 200 bootstrap iterations with replacement, with 95% confidence intervals estimated with using the quantile method.

65 **Table S1. Systematic review search terms**

66 Search terms were combined with “OR” within columns and with “AND” across columns. We developed a search strategy from a two-step
 67 process. First, known key studies prior to the systematic review (WASH Benefits, Mapsan, Gram Vikas, Odisah [Table 1]) were examined for
 68 keywords and Medical Subject Heading (MeSH) terms relating to each of the following categories of terms comprising our search string:
 69 WASH interventions; microbial source tracking and environmental contamination; enteric infection; diarrhea; and child growth and
 70 development. Next, we performed an initial search using these terms and extracted other relevant terms and synonyms from relevant
 71 articles in the search results, resulting in the final list presented in this table.

Study design	WASH	Environmental markers	Child health
matched, trial, RCT, experiment, intervention, randomized, randomised, quasi-randomized, quasi-randomised, quasi-experimental, pseudo-randomized, pseudo-randomised, non-randomized controlled trials	Water, Sanitation, Hygiene, Handwashing, WSH, Sanitation, Water Supply, Sanitary Drainage, Toilet Facilities, Drinking Water, Hand Hygiene, Water Purification, Waste Water, disinfection	molecular source tracking, microbial source tracking, microbial transmission, diarrheal pathogen, diarrheal pathogens, diarrhoeal pathogen, diarrhoeal pathogens, fecal-oral, faecal-oral, entericpathogen, entericpathogens, ruminant, avian, Feces, Faeces, Faecal, Faecal, Fecally, Faecally	Entericinfection, Soil-transmitted helminth, Protozoan, Seroconversion, Fecal microbiology, Faecal microbiology, Fecal biomarker, Faecal biomarker, Intestinal Diseases, Parasitic, Seroconversion, Enteritis, Helminthiasis, Helminthiasis, Intestinal infection, Viral infection, Bacterial infection, Parasite infection, Parasitic infection, Helminth infection, Fecal sampling, Faecal sampling, Stool sampling, Stool collection, Diarrhea, Dysentery, Child growth faltering, Growth faltering, Child development, Length-for-age, Height-for-age, Weight-for-age, Head circumference, Waist circumference, Stunting, Stunted, Wasting, Wasted, Linear growth, Anthropometric measurement, Malnutrition, Undernourished, Undernutrition, Underweight, Growth Disorders, Childnutrition disorder, Wasting syndrome, Thinness, Growth velocity

72
 73 **Table S2. Pubmed search string**

74 [MH] are mesh headers and [TW] are text words. Search strings for other databases are available in the Open Science Framework analysis
 75 preregistration materials (<https://osf.io/8sgzn/>).

((matched [tw]) OR (trial [tw]) OR (RCT [tw]) OR (experiment [tw]) OR (intervention [tw]) OR (randomized [tw]) OR (randomised [tw]) OR (quasi-randomized [tw]) OR (quasi-randomised [tw]) OR (quasi-experimental [tw]) OR (pseudo-randomized [tw]) OR (pseudo-randomised [tw]) OR (“non-randomized controlled trials as topic” [mh])) AND ((Water [tw]) OR (Sanitation [tw]) OR (Hygiene [tw]) OR (Handwashing [tw]) OR (WSH [tw]) OR (“Sanitation” [mh]) OR (“Water Supply” [mh]) OR (“Drainage, Sanitary” [mh]) OR (Sanitary Drainage [tw]) OR (“Toilet Facilities” [mh]) OR (“Drinking Water” [mh]) OR (“Hand Hygiene” [mh]) OR (“Water Purification” [mh]) OR (“Waste Water” [mh]) OR (disinfect* [tw])) AND ((molecular source tracking [tw]) OR (microbial source tracking [tw]) OR (microbial transmission [tw]) OR (diarrheal pathogen [tw]) OR (diarrheal pathogens [tw]) OR (diarrhoeal pathogen [tw]) OR (diarrhoeal pathogens [tw]) OR (fecal-oral [tw]) OR (faecal-oral [tw]) OR (faecal-oral [tw]) OR (enteric pathogen [tw]) OR (enteric pathogens [tw]) OR (ruminant* [tw]) OR (avian* [tw]) OR (“Feces” [mh]) OR (Feces [tw]) OR (Faeces [tw]) OR (Faecal [tw]) OR (Faecal [tw]) OR (Fecally [tw]) OR (Faecally [tw])) AND (((Enteric infection* [tw]) OR (Soil-transmitted helminth* [tw]) OR (Protozoan* [tw]) OR (Seroconversion [tw]) OR (Fecal microbio* [tw]) OR (Faecal microbio* [tw]) OR (Fecal biomarker* [tw]) OR (Faecal biomarker* [tw]) OR (“Intestinal Diseases, Parasitic/epidemiology” [mh]) OR (“Seroconversion” [mh]) OR (Seroconversion [tw]) OR (“Enteritis/epidemiology” [mh]) OR (“Helminthiasis/complications” [mh]) OR (Helminthiasis [tw]) OR (Helminthiasis)OR (“Helminthiasis/epidemiology” [mh]) OR (“Helminthiasis/prevention and control” [mh]) OR (Intestinal infection* [tw]) OR (Viral infection* [tw]) OR (Bacterial infection* [tw]) OR (Parasite infection* [tw]) OR (Parasitic infection* [tw]) OR (Helminth infection* [tw]) OR (Fecal sampling [tw]) OR (Faecal sampling [tw]) OR (Bacterial

infection* [tw]) OR (Parasite infection* [tw]) OR (Parasitic infection* [tw]) OR (Helminth infection* [tw]) OR (Fecal sampling [tw]) OR (Faecal sampling [tw]) OR (Stool sampling [tw]) OR (Stool collection [tw])) OR ((Diarrh* [tw]) OR (Dysentery [tw]) OR ("Diarrhea/epidemiology" [mh]) OR ("Diarrhea/etiology" [mh]) OR ("Diarrhea/prevention and control" [mh]) OR ("Diarrhea, Infantile" [mh]) OR ("Dysentery" [mh])) OR (Child growth faltering [tw]) OR (Growth faltering [tw]) OR (Child development [tw]) OR (Length-for-age [tw]) OR (Height-for-age [tw]) OR (Weight-for-age [tw]) OR (Head circumference [tw]) OR (Waist circumference [tw]) OR (Stunt* [tw]) OR (Wasting [tw]) OR (Wasted [tw]) OR (Linear growth [tw]) OR (Anthropometric measurement* [tw]) OR (Maln* [tw]) OR (Undernourish* [tw]) OR (Undernutrition [tw]) OR (Underweight [tw]) OR ("Growth Disorders" [mh]) OR (Growth Disorders [tw]) OR ("Child nutrition disorders" [mh]) OR (Child nutrition disorder* [tw]) OR ("Malnutrition" [mh]) OR ("Wasting Syndrome" [mh]) OR (Wasting syndrome [tw]) OR ("Thinness" [mh]) OR (Thinness [tw]) OR (Growth velocity [tw]))

2 **Table S3. PRISMA Checklist**

Topic	No.	Item	Location where item is reported
TITLE			
Title	1	Identify the report as a systematic review.	Title
ABSTRACT			
Abstract	2	See the PRISMA for Abstracts checklist below	
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of existing knowledge.	Introduction paragraph 2
Objectives	4	Provide an explicit statement of the objective(s) or question(s) the review addresses.	Introduction paragraph 3
METHODS			
Eligibility criteria	5	Specify the inclusion and exclusion criteria for the review and how studies were grouped for the syntheses.	Methods, paragraph 1
Information sources	6	Specify all databases, registers, websites, organisations, reference lists and other sources searched or consulted to identify studies. Specify the date when each source was last searched or consulted.	Methods, paragraph 1, Fig. S1
Search strategy	7	Present the full search strategies for all databases, registers and websites, including any filters and limits used.	Tables S1-S2
Selection process	8	Specify the methods used to decide whether a study met the inclusion criteria of the review, including how many reviewers screened each record and each report retrieved, whether they worked independently, and if applicable, details of automation tools used in the process.	Methods, paragraph 1

Topic	No.	Item	Location where item is reported
Data collection process	9	Specify the methods used to collect data from reports, including how many reviewers collected data from each report, whether they worked independently, any processes for obtaining or confirming data from study investigators, and if applicable, details of automation tools used in the process.	Methods, paragraph 1
Data items	10a	List and define all outcomes for which data were sought. Specify whether all results that were compatible with each outcome domain in each study were sought (e.g. for all measures, time points, analyses), and if not, the methods used to decide which results to collect.	Methods, paragraphs 2-3
	10b	List and define all other variables for which data were sought (e.g. participant and intervention characteristics, funding sources). Describe any assumptions made about any missing or unclear information.	Methods, paragraph 2-3
Study risk of bias assessment	11	Specify the methods used to assess risk of bias in the included studies, including details of the tool(s) used, how many reviewers assessed each study and whether they worked independently, and if applicable, details of automation tools used in the process.	Methods, paragraph 7
Effect measures	12	Specify for each outcome the effect measure(s) (e.g. risk ratio, mean difference) used in the synthesis or presentation of results.	Methods, paragraph 4
Synthesis methods	13a	Describe the processes used to decide which studies were eligible for each synthesis (e.g. tabulating the study intervention characteristics and comparing against the planned groups for each synthesis (item 5)).	Methods, paragraph 1
	13b	Describe any methods required to prepare the data for presentation or synthesis, such as handling of missing summary statistics, or data conversions.	Methods, paragraph 4-7
	13c	Describe any methods used to tabulate or visually display results of individual studies and syntheses.	Figure captions
	13d	Describe any methods used to synthesize results and provide a rationale for the choice(s). If meta-analysis was performed, describe the model(s), method(s) to identify the presence and extent of statistical heterogeneity, and software package(s) used.	Methods, paragraph 5

Topic	No.	Item	Location where item is reported
	13e	Describe any methods used to explore possible causes of heterogeneity among study results (e.g. subgroup analysis, meta-regression).	Methods, paragraph 6
	13f	Describe any sensitivity analyses conducted to assess robustness of the synthesized results.	Methods, paragraph 7
Reporting assessment	bias	14 Describe any methods used to assess risk of bias due to missing results in a synthesis (arising from reporting biases).	Not applicable
Certainty assessment		15 Describe any methods used to assess certainty (or confidence) in the body of evidence for an outcome.	Not applicable
RESULTS			
Study selection	16a	Describe the results of the search and selection process, from the number of records identified in the search to the number of studies included in the review, ideally using a flow diagram.	Figure S1
	16b	Cite studies that might appear to meet the inclusion criteria, but which were excluded, and explain why they were excluded.	Results, paragraphs 1-2
Study characteristics	17	Cite each included study and present its characteristics.	Results, paragraphs 1-2, Table 1
Risk of bias in studies	18	Present assessments of risk of bias for each included study.	Table S4
Results of individual studies	19	For all outcomes, present, for each study: (a) summary statistics for each group (where appropriate) and (b) an effect estimate and its precision (e.g. confidence/credible interval), ideally using structured tables or plots.	Figures 1,2 S2-S3, S5-S8, Tables 2, S6-S9
Results of syntheses	20a	For each synthesis, briefly summarise the characteristics and risk of bias among contributing studies.	Not applicable
	20b	Present results of all statistical syntheses conducted. If meta-analysis was done, present for each the summary estimate and its precision (e.g. confidence/credible interval) and measures of statistical heterogeneity. If comparing groups, describe the direction of the effect.	Figures 1 ,2 S2-S3, S5-S8, Tables 2, S6-S9

Topic	No.	Item	Location where item is reported
Reporting biases	20c	Present results of all investigations of possible causes of heterogeneity among study results.	Results, paragraph 12
	20d	Present results of all sensitivity analyses conducted to assess the robustness of the synthesized results.	Results, paragraph 12
	21	Present assessments of risk of bias due to missing results (arising from reporting biases) for each synthesis assessed.	Not applicable
Certainty of evidence	22	Present assessments of certainty (or confidence) in the body of evidence for each outcome assessed.	Figures 1, 2 S2-S3, S5-S8, Tables 2, S6-S9
DISCUSSION			
Discussion	23a	Provide a general interpretation of the results in the context of other evidence.	Discussion, paragraphs 1-2
	23b	Discuss any limitations of the evidence included in the review.	Discussion, paragraphs 3-4
	23c	Discuss any limitations of the review processes used.	Discussion, paragraphs 3-4
	23d	Discuss implications of the results for practice, policy, and future research.	Discussion, paragraph 5
OTHER INFORMATION			
Registration and protocol	24a	Provide registration information for the review, including register name and registration number, or state that the review was not registered.	https://osf.io/8sgzn/
	24b	Indicate where the review protocol can be accessed, or state that a protocol was not prepared.	https://osf.io/8sgzn/
	24c	Describe and explain any amendments to information provided at registration or in the protocol.	Not applicable
Support	25	Describe sources of financial or non-financial support for the review, and the role of the funders or sponsors in the review.	Accompanying manuscript

Topic	No.	Item	Location where item is reported
Competing interests	26	Declare any competing interests of review authors.	Accompanying manuscript
Availability of data, code and other materials	27	Report which of the following are publicly available and where they can be found: template data collection forms; data extracted from included studies; data used for all analyses; analytic code; any other materials used in the review.	Accompanying manuscript

3

Topic	No.	Item	Reported?
TITLE			
Title	1	Identify the report as a systematic review.	Yes
BACKGROUND			
Objectives	2	Provide an explicit statement of the main objective(s) or question(s) the review addresses.	Yes
METHODS			
Eligibility criteria	3	Specify the inclusion and exclusion criteria for the review.	Yes
Information sources	4	Specify the information sources (e.g. databases, registers) used to identify studies and the date when each was last searched.	No
Risk of bias	5	Specify the methods used to assess risk of bias in the included studies.	Yes
Synthesis of results	6	Specify the methods used to present and synthesize results.	Yes
RESULTS			
Included studies	7	Give the total number of included studies and participants and summarise relevant characteristics of studies.	Yes

Topic	No.	Item	Reported?
Synthesis results	of	8 Present results for main outcomes, preferably indicating the number of included studies and participants for each. If meta-analysis was done, report the summary estimate and confidence/credible interval. If comparing groups, indicate the direction of the effect (i.e. which group is favoured).	Yes
DISCUSSION			
Limitations of evidence	of	9 Provide a brief summary of the limitations of the evidence included in the review (e.g. study risk of bias, inconsistency and imprecision).	Yes
Interpretation		10 Provide a general interpretation of the results and important implications.	Yes
OTHER			
Funding		11 Specify the primary source of funding for the review.	Yes
Registration		12 Provide the register name and registration number.	Yes

4
5

Table S4. Risk of bias based on modified Newcastle-Ottawa scale

Stars are given for low risk of bias in each category, up to a total of nine stars. Scoring details are in the footnotes.

Reference	Selection bias	Response bias	Follow-up bias	Misclassification bias	Outcome assessment	Outcome measurement	Bias in analysis	Total
	Is there evidence of selection bias, which refers to systematic differences between baseline characteristics of the groups that are compared? ^a	Is there evidence of response bias? ^b	Is there evidence of bias due to missing follow-up data? ^c	Is there risk of households not receiving the intervention being misclassified as having received it, or vice versa? ^d	Is there evidence of bias arising from how the outcome was assessed? ^e	Is there evidence of ascertainment bias? ^f	Is there evidence that analysis was not appropriately adjusted for clustering and/or confounding, if appropriate? ^g	Total number of stars (x/9 possible stars).
Clasen T, et al. Effectiveness of a rural sanitation programme on diarrhoea, soil-transmitted helminth infection, and child malnutrition in Odisha, India: a cluster-randomised trial. <i>Lancet Glob Health</i> . 2014.	*	* no, laboratory assessed and blinded	possible (86% of possible weeks are reported weeks)	* household-level interventions	**	*	** adjusted for clustering	8
Luby, S.P. et al.. Effects of water quality, sanitation, handwashing, and nutritional interventions on diarrhoea and child growth in rural Bangladesh: a cluster randomised controlled trial. <i>The Lancet Global Health</i> 2018	*	* no, laboratory assessed and blinded	* 94% complete FU	* household-level interventions	**	*	**	9

Table S4. Risk of bias based on modified Newcastle-Ottawa scale

Stars are given for low risk of bias in each category, up to a total of nine stars. Scoring details are in the footnotes.

Reference	Selection bias	Response bias	Follow-up bias	Misclassification bias	Outcome assessment	Outcome measurement	Bias in analysis	Total
Null, C. et al., Effects of water quality, sanitation, handwashing, and nutritional interventions on diarrhoea and child growth in rural Kenya: a cluster-randomised controlled trial. The Lancet Global Health 2018	*	* no, laboratory assessed and blinded	* <1% loss to FU	* household-level interventions	**	*	**	9
Reese, H. et al. Assessing longer-term effectiveness of a combined household-level piped water and sanitation intervention on child diarrhoea, acute respiratory infection, soil-transmitted helminth infection and nutritional status: a matched cohort study in rural Odisha, India. International journal of epidemiology 2019	selection bias is possible, as the study is not randomized and there are some baseline differences between intervention and control group	* no, laboratory assessed and blinded	substantial loss to FU	* household-level interventions	**	*	**	7

Table S4. Risk of bias based on modified Newcastle-Ottawa scale

Stars are given for low risk of bias in each category, up to a total of nine stars. Scoring details are in the footnotes.

Reference	Selection bias	Response bias	Follow-up bias	Misclassification bias	Outcome assessment	Outcome measurement	Bias in analysis	Total
Knee, J. et al. Effects of an urban sanitation intervention on childhood enteric infection and diarrhea in Maputo, Mozambique: A controlled before-and-after trial. eLife 2011	selection bias is possible, as the study is not randomized, but intervention and control groups were mostly balanced at baseline. Control households were more likely to have covered floors and higher quality walls and intervention groups had more people per household.	* no, laboratory assessed and blinded	substantial loss to FU	* household-level interventions	**	*	**	7

80 ^a RCTs receive 1 star, unless evidence of selection bias (e.g. randomisation procedures not followed). Meaningful differences between groups at baseline
81 in RCTs receive 0 stars. Rates of declining to participate >10% receive 0 stars. Non- or quasi-randomised studies receive 0 stars.
82 ^b If intervention recipient was not blinded to intervention status, 0 stars.
83 ^c <10% receives 1 star, greater than or equal to 10% receives 0 stars.
84 ^d Interventions delivered at the household/individual level receive 1 star. Interventions delivered at the community level that missed a substantial,
85 i.e. greater than or equal to 10%, proportion of the target population receive 0 stars, including when there is insufficient information to verify whether
86 this is the case. Interventions with substantial risk of contamination (control households receiving intervention) receive 0 stars.
87 ^e Parent / person recall (=0 stars). Fieldworker assessed (=1 star). Physician/microbiologically assessed (=2 stars)
88 ^f If outcome measurement staff were not blinded to intervention status, 0 stars.
89 ^g Scoring is based on losing stars (max. 2). Individual RCTs with baseline balance on covariates are unlikely to require adjustment (=2 stars). Cluster-RCTs
90 and non-randomised trials may require adjustment for clustering (-1 star if not done). RCTs or cRCTs may require adjustment for covariates, with
91 justification (-1 star if not done). Non-randomised studies require adjustment for covariates (-1 star if not done), but also adequate justification for
92 covariate selection (-1 star if not included), and there can be too few or too many covariates.

Table S5. Prevalence of pathogens by sample type tested in each study

Study	Sample	Target	Percent positive (n/N)	PR (95% CI)
Odagiri 2016	Source water	V. cholerae	31.7% (19/60)	0.73 (0.34, 1.57)
-	-	Adenovirus	8.3% (5/60)	0.25 (0.03, 2.19)
-	-	Rotavirus	23.3% (14/60)	0.75 (0.29, 1.93)
Boehm 2016	Stored water	Rotavirus	0.6% (3/493)	-
-	Child hand rinse	Rotavirus	6.1% (30/493)	-
-	House soil	Rotavirus	1.4% (7/496)	2.52 (0.51, 12.42)
Reese 2017	Source water	Shigella	10.7% (161/1499)	0.73 (0.46, 1.15)
-	-	V. cholerae	13% (36/276)	0.93 (0.46, 1.85)
-	Stored water	Shigella	10.1% (190/1874)	1.08 (0.77, 1.51)
-	-	V. cholerae	23.7% (100/422)	1.03 (0.66, 1.6)
Steinbaum 2019	House soil	Ascaris	13% (273/2107)	0.88 (0.68, 1.13)
-	-	Trichuris	6.9% (146/2107)	0.86 (0.6, 1.23)
Fuhrmeister 2020	Stored water	Pathogenic E. coli	38.6% (286/741)	1 (0.84, 1.19)
-	Child hand rinse	Pathogenic E. coli	34% (127/373)	-
-	-	Giardia	4.8% (15/311)	-
-	-	Norovirus	4.2% (14/337)	-
-	Mother's hand rinse	Pathogenic E. coli	24% (177/737)	-
-	-	Giardia	2.3% (14/602)	-
-	-	Norovirus	3.1% (21/684)	-
-	House soil	Pathogenic E. coli	61.3% (453/739)	0.94 (0.84, 1.06)
Capone 2021	Latrine soil	C. difficile	14.8% (13/88)	0.9 (0.32, 2.48)
-	-	Campylobacter	6.8% (6/88)	2.09 (0.4, 11.05)

Table S5. Prevalence of pathogens by sample type tested in each study

Study	Sample	Target	Percent positive (n/N)	PR (95% CI)
-	-	Pathogenic E. coli	56.8% (50/88)	0.89 (0.56, 1.42)
-	-	Salmonella	6.8% (6/88)	0.52 (0.1, 2.76)
-	-	Shigella	21.6% (19/88)	0.28 (0.1, 0.78)
-	-	V. cholerae	0% (0/88)	-
-	-	Yersinia	4.5% (4/88)	-
-	-	Ascaris	60.2% (53/88)	0.65 (0.41, 1.02)
-	-	Trichuris	17% (15/88)	0.92 (0.36, 2.33)
-	-	Cryptosporidium	8% (7/88)	0.78 (0.18, 3.36)
-	-	Entamoeba histolytica	1.1% (1/88)	-
-	-	Giardia	31.8% (28/88)	0.47 (0.21, 1.07)
-	-	Adenovirus	20.5% (18/88)	0.21 (0.06, 0.68)
-	-	Astrovirus	29.5% (26/88)	1.27 (0.67, 2.43)
-	-	Norovirus	2.3% (2/88)	-
-	-	Rotavirus	4.5% (4/88)	-
-	-	Sapovirus	0% (0/88)	-
Capone 2022 in prep	Flies	Campylobacter	1.2% (1/86)	-
-	-	Pathogenic E. coli	30.2% (26/86)	-
-	-	Shigella	2.3% (2/86)	-
-	-	V. cholerae	2.3% (2/86)	-
-	-	Ascaris	0% (0/86)	-
-	-	Trichuris	3.5% (3/86)	-
-	-	Giardia	4.7% (4/86)	-

Table S5. Prevalence of pathogens by sample type tested in each study

Study	Sample	Target	Percent positive (n/N)	PR (95% CI)
-	-	Adenovirus	4.7% (4/86)	-
-	-	Astrovirus	0% (0/86)	-
-	-	Norovirus	2.3% (2/86)	-
-	-	Pan enterovirus	0% (0/86)	-
-	-	Rotavirus	1.2% (1/86)	-
-	-	Sapovirus	0% (0/86)	-
Kwong 2021	House soil	Ascaris	62.1% (886/1426)	0.97 (0.87, 1.08)
-	-	Trichuris	56% (798/1426)	1.03 (0.91, 1.15)

Table S6. Prevalence of microbial source tracking markers by sample type tested in each study

Study	Sample	Target	Percent positive (n/N)	PR (95% CI)
Odagiri 2016	Source water	Animal (BacCow)	91.7% (55/60)	1.04 (0.89, 1.21)
-	-	Human (BacHum)	71.7% (43/60)	1.05 (0.76, 1.45)
Boehm 2016	Stored water	Avian (GFD)	9.3% (46/493)	0.71 (0.37, 1.36)
-	-	Ruminant (BacR)	21.9% (108/493)	0.62 (0.43, 0.9)
-	-	Human (HumM2)	0% (0/493)	-
-	Child hand rinse	Avian (GFD)	16.2% (80/493)	-
-	-	Ruminant (BacR)	54.2% (267/493)	-
-	-	Human (HumM2)	2.4% (12/493)	-
-	House soil	Avian (GFD)	33.3% (165/496)	0.98 (0.76, 1.27)
-	-	Ruminant (BacR)	66.7% (331/496)	0.98 (0.85, 1.12)
-	-	Human (HumM2)	8.9% (44/496)	0.94 (0.5, 1.75)
Fuhrmeister 2020	Stored water	Animal (BacCow)	68.5% (482/704)	0.97 (0.87, 1.08)
-	-	Human (HumM2)	2.6% (17/651)	0.44 (0.16, 1.23)
-	Child hand rinse	Animal (BacCow)	97.5% (356/365)	-
-	-	Human (HumM2)	21.9% (74/338)	-
-	Mother's hand rinse	Animal (BacCow)	96.7% (702/726)	-
-	-	Human (HumM2)	18.1% (118/651)	-
-	House soil	Animal (BacCow)	90.6% (572/631)	0.99 (0.94, 1.04)
-	-	Human (HumM2)	20.1% (127/631)	1.24 (0.91, 1.7)
Holcomb 2021	Source water	Avian (GFD)	0% (0/41)	-
-	-	Human (HF183)	2.4% (1/41)	-
-	-	Human (M. smithii)	0% (0/41)	-

Table S6. Prevalence of microbial source tracking markers by sample type tested in each study

Study	Sample	Target	Percent positive (n/N)	PR (95% CI)
-	Stored water	Avian (GFD)	1.1% (1/94)	-
-	-	Human (HF183)	14.9% (14/94)	1.72 (0.57, 5.18)
-	-	Human (M. smithii)	0% (0/94)	-
-	Latrine soil	Avian (GFD)	3.3% (2/60)	-
-	-	Human (HF183)	50% (30/60)	0.88 (0.51, 1.52)
-	-	Human (M. smithii)	45% (27/60)	0.74 (0.36, 1.55)
-	House soil	Avian (GFD)	3.6% (3/83)	-
-	-	Human (HF183)	42.2% (35/83)	0.81 (0.49, 1.34)
-	-	Human (M. smithii)	24.1% (20/83)	1.3 (0.62, 2.73)
Capone 2022 in prep	Flies	Animal (BacCow)	12.8% (11/86)	-
-	-	Dog (BacCan)	30.2% (26/86)	-
-	-	Human (BacHum)	72.1% (62/86)	-

Table S7.

Unadjusted and adjusted results by study, sample type, and aggregated variables for pathogen targets (any pathogen, any bacteria, any viruses, any protozoa, any STH).

Study	Target	Sample	Positive, Intervention	Negative, Intervention	Positive, Control	Negative, Control	Total observations	Unadjusted Prevalence Ratio	Unadjusted p-value	Adjusted Prevalence Ratio	Adjusted p-value
Capone 2022 in prep	Any pathogen	Any sample	7	13	20	17	57	PR=0.65 (95% CI: 0.33, 1.28)	0.21	PR=0.5 (95% CI: 0.21, 1.19)	0.12
Capone 2021	Any pathogen	Any sample	37	6	43	2	88	PR=0.9 (95% CI: 0.78, 1.03)	0.13	PR=0.9 (95% CI: 0.78, 1.03)	0.13
Fuhrmeister 2020	Any pathogen	Any sample	314	136	348	123	921	PR=0.94 (95% CI: 0.87, 1.02)	0.17	PR=0.94 (95% CI: 0.87, 1.02)	0.13
Steinbaum 2019	Any pathogen	Any sample	206	979	173	707	2,065	PR=0.88 (95% CI: 0.7, 1.11)	0.29	PR=0.87 (95% CI: 0.7, 1.09)	0.24
Reese 2017	Any pathogen	Any sample	185	792	238	825	2,040	PR=0.85 (95% CI: 0.66, 1.08)	0.18	PR=0.86 (95% CI: 0.68, 1.09)	0.21
Boehm 2016	Any pathogen	Any sample	19	229	15	234	497	PR=1.27 (95% CI: 0.6, 2.68)	0.53	PR=1.28 (95% CI: 0.62, 2.66)	0.5
Odagiri 2016	Any pathogen	Any sample	12	18	15	15	60	PR=0.8 (95% CI: 0.45, 1.42)	0.45		
Reese 2017	Any pathogen	Source water	68	588	122	747	1,525	PR=0.74 (95% CI: 0.49, 1.12)	0.15	PR=0.74 (95% CI: 0.5, 1.12)	0.16
Odagiri 2016	Any pathogen	Source water	12	18	15	15	60	PR=0.8 (95% CI: 0.45, 1.42)	0.45		
Fuhrmeister 2020	Any pathogen	Stored water	138	218	148	237	741	PR=1.01 (95% CI: 0.85, 1.2)	0.93	PR=1 (95% CI: 0.84, 1.19)	0.97
Reese 2017	Any pathogen	Stored water	134	786	147	860	1,927	PR=1 (95% CI: 0.75, 1.32)	0.99	PR=1.01 (95% CI: 0.77, 1.34)	0.94
Boehm 2016	Any pathogen	Stored water	2	243	1	245	491	Not estimated		Not estimated	
Kwong 2021	Any pathogen	House soil	363	125	687	221	1,396	PR=0.98 (95% CI: 0.91, 1.06)	0.67	PR=0.98 (95% CI: 0.91, 1.06)	0.68
Fuhrmeister 2020	Any pathogen	House soil	217	144	236	142	739	PR=0.96 (95% CI: 0.86, 1.08)	0.53	PR=0.94 (95% CI: 0.84, 1.06)	0.31

Table S7.

Unadjusted and adjusted results by study, sample type, and aggregated variables for pathogen targets (any pathogen, any bacteria, any viruses, any protozoa, any STH).

Study	Target	Sample	Positive, Intervention	Negative, Intervention	Positive, Control	Negative, Control	Total observations	Unadjusted Prevalence Ratio	Unadjusted p-value	Adjusted Prevalence Ratio	Adjusted p-value
Steinbaum 2019	Any pathogen	House soil	209	1,000	173	725	2,107	PR=0.9 (95% CI: 0.72, 1.13)	0.35	PR=0.89 (95% CI: 0.71, 1.11)	0.31
Boehm 2016	Any pathogen	House soil	5	242	2	247	496	PR=2.52 (95% CI: 0.51, 12.42)	0.26	PR=2.52 (95% CI: 0.51, 12.42)	0.26
Capone 2021	Any pathogen	Latrine soil	37	6	43	2	88	PR=0.9 (95% CI: 0.78, 1.03)	0.13	PR=0.9 (95% CI: 0.78, 1.03)	0.13
Capone 2022 in prep	Any pathogen		8	23	25	30	86	PR=0.57 (95% CI: 0.28, 1.15)	0.12	PR=0.37 (95% CI: 0.16, 0.85)	0.02
Fuhrmeister 2020	Any pathogen		75	113	72	116	376	PR=1.04 (95% CI: 0.8, 1.35)	0.76	PR=1.05 (95% CI: 0.81, 1.37)	0.69
Fuhrmeister 2020	Any pathogen		96	266	110	267	739	PR=0.91 (95% CI: 0.72, 1.15)	0.43	PR=0.92 (95% CI: 0.72, 1.16)	0.47
Boehm 2016	Any pathogen		16	231	14	232	493	PR=1.14 (95% CI: 0.52, 2.48)	0.75	PR=1.13 (95% CI: 0.52, 2.44)	0.76
Capone 2022 in prep	Any bacteria	Any sample	7	13	17	20	57	PR=0.76 (95% CI: 0.38, 1.54)	0.45	PR=0.6 (95% CI: 0.24, 1.46)	0.26
Capone 2021	Any bacteria	Any sample	28	15	35	10	88	PR=0.84 (95% CI: 0.64, 1.1)	0.2	PR=0.85 (95% CI: 0.65, 1.11)	0.24
Fuhrmeister 2020	Any bacteria	Any sample	306	144	340	131	921	PR=0.94 (95% CI: 0.86, 1.03)	0.18	PR=0.94 (95% CI: 0.86, 1.02)	0.14
Reese 2017	Any bacteria	Any sample	185	792	238	825	2,040	PR=0.85 (95% CI: 0.66, 1.08)	0.18	PR=0.86 (95% CI: 0.68, 1.09)	0.21
Odagiri 2016	Any bacteria	Any sample	8	22	11	19	60	PR=0.73 (95% CI: 0.34, 1.57)	0.42		
Reese 2017	Any bacteria	Source water	68	588	122	747	1,525	PR=0.74 (95% CI: 0.49, 1.12)	0.15	PR=0.74 (95% CI: 0.5, 1.12)	0.16
Odagiri 2016	Any bacteria	Source water	8	22	11	19	60	PR=0.73 (95% CI: 0.34, 1.57)	0.42		

Table S7.

Unadjusted and adjusted results by study, sample type, and aggregated variables for pathogen targets (any pathogen, any bacteria, any viruses, any protozoa, any STH).

Study	Target	Sample	Positive, Intervention	Negative, Intervention	Positive, Control	Negative, Control	Total observations	Unadjusted Prevalence Ratio	Unadjusted p-value	Adjusted Prevalence Ratio	Adjusted p-value
Fuhrmeister 2020	Any bacteria	Stored water	138	218	148	237	741	PR=1.01 (95% CI: 0.85, 1.2)	0.93	PR=1 (95% CI: 0.84, 1.19)	0.97
Reese 2017	Any bacteria	Stored water	134	786	147	860	1,927	PR=1 (95% CI: 0.75, 1.32)	0.99	PR=1.01 (95% CI: 0.77, 1.34)	0.94
Fuhrmeister 2020	Any bacteria	House soil	217	144	236	142	739	PR=0.96 (95% CI: 0.86, 1.08)	0.53	PR=0.94 (95% CI: 0.84, 1.06)	0.31
Capone 2021	Any bacteria	Latrine soil	28	15	35	10	88	PR=0.84 (95% CI: 0.64, 1.1)	0.2	PR=0.85 (95% CI: 0.65, 1.11)	0.24
Capone 2022 in prep	Any bacteria		8	23	21	34	86	PR=0.68 (95% CI: 0.32, 1.41)	0.3	PR=0.62 (95% CI: 0.28, 1.38)	0.24
Fuhrmeister 2020	Any bacteria		64	122	63	124	373	PR=1.02 (95% CI: 0.78, 1.35)	0.88	PR=1.02 (95% CI: 0.78, 1.35)	0.88
Fuhrmeister 2020	Any bacteria		81	281	96	279	737	PR=0.87 (95% CI: 0.68, 1.13)	0.3	PR=0.85 (95% CI: 0.67, 1.09)	0.2
Capone 2022 in prep	Any virus	Any sample	0	20	4	33	57	Not estimated		Not estimated	
Capone 2021	Any virus	Any sample	16	27	22	23	88	PR=0.76 (95% CI: 0.46, 1.25)	0.28	PR=0.63 (95% CI: 0.35, 1.14)	0.13
Fuhrmeister 2020	Any virus	Any sample	17	330	14	338	699	PR=1.23 (95% CI: 0.63, 2.4)	0.54	PR=1.22 (95% CI: 0.63, 2.34)	0.56
Boehm 2016	Any virus	Any sample	19	229	15	234	497	PR=1.27 (95% CI: 0.6, 2.68)	0.53	PR=1.28 (95% CI: 0.62, 2.66)	0.5
Odagiri 2016	Any virus	Any sample	7	23	10	20	60	PR=0.7 (95% CI: 0.3, 1.62)	0.4		
Odagiri 2016	Any virus	Source water	7	23	10	20	60	PR=0.7 (95% CI: 0.3, 1.62)	0.4		
Boehm 2016	Any virus	Stored water	2	243	1	245	491	Not estimated		Not estimated	

Table S7.

Unadjusted and adjusted results by study, sample type, and aggregated variables for pathogen targets (any pathogen, any bacteria, any viruses, any protozoa, any STH).

Study	Target	Sample	Positive, Intervention	Negative, Intervention	Positive, Control	Negative, Control	Total observations	Unadjusted Prevalence Ratio	Unadjusted p-value	Adjusted Prevalence Ratio	Adjusted p-value
Boehm 2016	Any virus	House soil	5	242	2	247	496	PR=2.52 (95% CI: 0.51, 12.42)	0.26	PR=2.52 (95% CI: 0.51, 12.42)	0.26
Capone 2021	Any virus	Latrine soil	16	27	22	23	88	PR=0.76 (95% CI: 0.46, 1.25)	0.28	PR=0.63 (95% CI: 0.35, 1.14)	0.13
Capone 2022 in prep	Any virus		0	31	5	50	86	PR=0 (95% CI: 0, 0)	0	PR=0 (95% CI: 0, 0)	0
Fuhrmeister 2020	Any virus		7	162	7	161	337	PR=0.99 (95% CI: 0.37, 2.69)	0.99	PR=0.99 (95% CI: 0.37, 2.69)	0.99
Fuhrmeister 2020	Any virus		11	331	10	332	684	PR=1.1 (95% CI: 0.47, 2.57)	0.83	PR=1.06 (95% CI: 0.45, 2.46)	0.9
Boehm 2016	Any virus		16	231	14	232	493	PR=1.14 (95% CI: 0.52, 2.48)	0.75	PR=1.13 (95% CI: 0.52, 2.44)	0.76
Capone 2022 in prep	Any protozoa	Any sample	0	20	3	34	57	Not estimated		Not estimated	
Capone 2021	Any protozoa	Any sample	15	28	19	26	88	PR=0.83 (95% CI: 0.48, 1.42)	0.49	PR=0.83 (95% CI: 0.48, 1.42)	0.49
Fuhrmeister 2020	Any protozoa	Any sample	12	293	16	291	612	PR=0.75 (95% CI: 0.35, 1.65)	0.48	PR=0.77 (95% CI: 0.35, 1.67)	0.5
Capone 2021	Any protozoa	Latrine soil	15	28	19	26	88	PR=0.83 (95% CI: 0.48, 1.42)	0.49	PR=0.83 (95% CI: 0.48, 1.42)	0.49
Capone 2022 in prep	Any protozoa		0	31	4	51	86	Not estimated		Not estimated	
Fuhrmeister 2020	Any protozoa		7	147	8	149	311	PR=0.89 (95% CI: 0.33, 2.38)	0.82	PR=0.89 (95% CI: 0.33, 2.38)	0.82
Fuhrmeister 2020	Any protozoa		5	296	9	292	602	PR=0.56 (95% CI: 0.14, 2.13)	0.39	PR=0.56 (95% CI: 0.14, 2.13)	0.39
Capone 2022 in prep	Any STH	Any sample	0	20	3	34	57	Not estimated		Not estimated	

Table S7.

Unadjusted and adjusted results by study, sample type, and aggregated variables for pathogen targets (any pathogen, any bacteria, any viruses, any protozoa, any STH).

Study	Target	Sample	Positive, Intervention	Negative, Intervention	Positive, Control	Negative, Control	Total observations	Unadjusted Prevalence Ratio	Unadjusted p-value	Adjusted Prevalence Ratio	Adjusted p-value
Capone 2021	Any STH	Any sample	20	23	34	11	88	PR=0.62 (95% CI: 0.43, 0.89)	0.01	PR=0.69 (95% CI: 0.45, 1.07)	0.1
Steinbaum 2019	Any STH	Any sample	206	979	173	707	2,065	PR=0.88 (95% CI: 0.7, 1.11)	0.29	PR=0.87 (95% CI: 0.7, 1.09)	0.24
Kwong 2021	Any STH	House soil	363	125	687	221	1,396	PR=0.98 (95% CI: 0.91, 1.06)	0.67	PR=0.98 (95% CI: 0.91, 1.06)	0.68
Steinbaum 2019	Any STH	House soil	209	1,000	173	725	2,107	PR=0.9 (95% CI: 0.72, 1.13)	0.35	PR=0.89 (95% CI: 0.71, 1.11)	0.31
Capone 2021	Any STH	Latrine soil	20	23	34	11	88	PR=0.62 (95% CI: 0.43, 0.89)	0.01	PR=0.69 (95% CI: 0.45, 1.07)	0.1
Capone 2022 in prep	Any STH		0	31	3	52	86	Not estimated		Not estimated	

95

96

97

Table S8.

Unadjusted and adjusted results by study, sample type, and aggregated variables for MST targets (any MST, any general MST, any human MST, any animal MST).

Study	Target	Sample	Positive, Intervention	Negative, Intervention	Positive, Control	Negative, Control	Total observations	Unadjusted Prevalence Ratio	Unadjusted p-value	Adjusted Prevalence Ratio	Adjusted p-value
Capone 2022 in prep	Any MST Marker	Any sample	20	0	32	5	57	PR=1.16 (95% CI: 1.02, 1.32)	0.03	PR=1.16 (95% CI: 1.02, 1.32)	0.03
Holcomb 2021	Any MST Marker	Any sample	41	28	44	17	130	PR=0.82 (95% CI: 0.62, 1.09)	0.18	PR=0.86 (95% CI: 0.65, 1.13)	0.27
Fuhrmeister 2020	Any MST Marker	Any sample	421	26	438	29	914	PR=1 (95% CI: 0.97, 1.04)	0.8	PR=1.01 (95% CI: 0.97, 1.04)	0.7
Boehm 2016	Any MST Marker	Any sample	220	28	222	27	497	PR=0.99 (95% CI: 0.93, 1.06)	0.88	PR=0.99 (95% CI: 0.93, 1.06)	0.76
Odagiri 2016	Any MST Marker	Any sample	30	0	28	2	60	Not estimated			
Holcomb 2021	Any MST Marker	Source water	1	21	0	19	41	Not estimated		Not estimated	
Odagiri 2016	Any MST Marker	Source water	30	0	28	2	60	Not estimated			
Holcomb 2021	Any MST Marker	Stored water	9	39	6	40	94	PR=1.44 (95% CI: 0.51, 4.08)	0.5	PR=1.44 (95% CI: 0.51, 4.08)	0.5
Fuhrmeister 2020	Any MST Marker	Stored water	230	119	256	119	724	PR=0.97 (95% CI: 0.87, 1.07)	0.52	PR=0.97 (95% CI: 0.88, 1.08)	0.63
Boehm 2016	Any MST Marker	Stored water	57	188	82	164	491	PR=0.7 (95% CI: 0.51, 0.96)	0.03	PR=0.69 (95% CI: 0.5, 0.95)	0.02
Holcomb 2021	Any MST Marker	House soil	21	18	26	18	83	PR=0.91 (95% CI: 0.6, 1.38)	0.66	PR=0.89 (95% CI: 0.62, 1.28)	0.54

Table S8.

Unadjusted and adjusted results by study, sample type, and aggregated variables for MST targets (any MST, any general MST, any human MST, any animal MST).

Study	Target	Sample	Positive, Intervention	Negative, Intervention	Positive, Control	Negative, Control	Total observations	Unadjusted Prevalence Ratio	Unadjusted p-value	Adjusted Prevalence Ratio	Adjusted p-value
Fuhrmeister 2020	Any MST Marker	House soil	283	38	297	36	654	PR=0.99 (95% CI: 0.93, 1.05)	0.7	PR=0.99 (95% CI: 0.93, 1.05)	0.66
Boehm 2016	Any MST Marker	House soil	180	67	187	62	496	PR=0.97 (95% CI: 0.87, 1.08)	0.59	PR=0.97 (95% CI: 0.87, 1.08)	0.58
Holcomb 2021	Any MST Marker	Latrine soil	21	9	22	8	60	PR=0.95 (95% CI: 0.69, 1.32)	0.78	PR=0.95 (95% CI: 0.69, 1.32)	0.78
Capone 2022 in prep	Any MST Marker		27	4	42	13	86	PR=1.14 (95% CI: 0.93, 1.39)	0.2	PR=1.14 (95% CI: 0.93, 1.39)	0.2
Fuhrmeister 2020	Any MST Marker		174	11	182	1	368	PR=0.95 (95% CI: 0.91, 0.98)	0.01	PR=0.95 (95% CI: 0.91, 0.98)	0.01
Fuhrmeister 2020	Any MST Marker		346	14	359	9	728	PR=0.99 (95% CI: 0.96, 1.01)	0.26	PR=0.99 (95% CI: 0.96, 1.01)	0.29
Boehm 2016	Any MST Marker		145	102	148	98	493	PR=0.98 (95% CI: 0.82, 1.16)	0.78	PR=0.97 (95% CI: 0.82, 1.15)	0.74
Capone 2022 in prep	Any human MST Marker	Any sample	17	3	30	7	57	PR=1.05 (95% CI: 0.82, 1.34)	0.71	PR=1.05 (95% CI: 0.82, 1.34)	0.71
Holcomb 2021	Any human MST Marker	Any sample	41	28	43	18	130	PR=0.84 (95% CI: 0.63, 1.12)	0.24	PR=0.89 (95% CI: 0.67, 1.18)	0.41
Fuhrmeister 2020	Any human MST Marker	Any sample	124	313	133	330	900	PR=0.99 (95% CI: 0.8, 1.22)	0.91	PR=1.01 (95% CI: 0.82, 1.25)	0.92
Boehm 2016	Any human MST Marker	Any sample	26	222	26	223	497	PR=1 (95% CI: 0.57, 1.75)	0.99	PR=1 (95% CI: 0.57, 1.76)	0.99

Table S8.

Unadjusted and adjusted results by study, sample type, and aggregated variables for MST targets (any MST, any general MST, any human MST, any animal MST).

Study	Target	Sample	Positive, Intervention	Negative, Intervention	Positive, Control	Negative, Control	Total observations	Unadjusted Prevalence Ratio	Unadjusted p-value	Adjusted Prevalence Ratio	Adjusted p-value
Odagiri 2016	Any human MST Marker	Any sample	22	8	21	9	60	PR=1.05 (95% CI: 0.76, 1.45)	0.78		
Holcomb 2021	Any human MST Marker	Source water	1	21	0	19	41	Not estimated		Not estimated	
Odagiri 2016	Any human MST Marker	Source water	22	8	21	9	60	PR=1.05 (95% CI: 0.76, 1.45)	0.78		
Holcomb 2021	Any human MST Marker	Stored water	9	39	5	41	94	PR=1.72 (95% CI: 0.57, 5.18)	0.33	PR=1.72 (95% CI: 0.57, 5.18)	0.33
Fuhrmeister 2020	Any human MST Marker	Stored water	5	310	12	324	651	PR=0.44 (95% CI: 0.16, 1.23)	0.12	PR=0.44 (95% CI: 0.16, 1.23)	0.12
Boehm 2016	Any human MST Marker	Stored water	0	245	0	246	491	Not estimated		Not estimated	
Holcomb 2021	Any human MST Marker	House soil	20	19	26	18	83	PR=0.87 (95% CI: 0.57, 1.32)	0.5	PR=0.86 (95% CI: 0.6, 1.24)	0.42
Fuhrmeister 2020	Any human MST Marker	House soil	68	243	59	261	631	PR=1.19 (95% CI: 0.87, 1.61)	0.28	PR=1.24 (95% CI: 0.91, 1.7)	0.18
Boehm 2016	Any human MST Marker	House soil	21	226	23	226	496	PR=0.92 (95% CI: 0.5, 1.71)	0.79	PR=0.94 (95% CI: 0.5, 1.75)	0.84
Holcomb 2021	Any human MST Marker	Latrine soil	21	9	22	8	60	PR=0.95 (95% CI: 0.69, 1.32)	0.78	PR=0.95 (95% CI: 0.69, 1.32)	0.78
Capone 2022 in prep	Any human MST Marker		24	7	38	17	86	PR=1.12 (95% CI: 0.83, 1.51)	0.46	PR=1.02 (95% CI: 0.75, 1.41)	0.88

Table S8.

Unadjusted and adjusted results by study, sample type, and aggregated variables for MST targets (any MST, any general MST, any human MST, any animal MST).

Study	Target	Sample	Positive, Intervention	Negative, Intervention	Positive, Control	Negative, Control	Total observations	Unadjusted Prevalence Ratio	Unadjusted p-value	Adjusted Prevalence Ratio	Adjusted p-value
Fuhrmeister 2020	Any human MST Marker		30	142	44	122	338	PR=0.66 (95% CI: 0.44, 0.99)	0.04	PR=0.72 (95% CI: 0.48, 1.07)	0.11
Fuhrmeister 2020	Any human MST Marker		58	268	60	265	651	PR=0.96 (95% CI: 0.68, 1.37)	0.84	PR=0.96 (95% CI: 0.68, 1.35)	0.82
Boehm 2016	Any human MST Marker		7	240	5	241	493	PR=1.39 (95% CI: 0.46, 4.2)	0.56	PR=1.39 (95% CI: 0.46, 4.2)	0.56
Capone 2022 in prep	Any animal MST Marker	Any sample	12	8	17	20	57	PR=1.31 (95% CI: 0.78, 2.17)	0.3	PR=1.2 (95% CI: 0.72, 1.99)	0.48
Holcomb 2021	Any animal MST Marker	Any sample	3	66	2	59	130	PR=1.33 (95% CI: 0.18, 9.59)	0.78	PR=1.33 (95% CI: 0.18, 9.59)	0.78
Fuhrmeister 2020	Any animal MST Marker	Any sample	419	26	437	28	910	PR=1 (95% CI: 0.97, 1.04)	0.91	PR=1 (95% CI: 0.97, 1.04)	0.8
Boehm 2016	Any animal MST Marker	Any sample	219	29	221	28	497	PR=0.99 (95% CI: 0.93, 1.06)	0.88	PR=0.99 (95% CI: 0.93, 1.06)	0.74
Odagiri 2016	Any animal MST Marker	Any sample	28	2	27	3	60	PR=1.04 (95% CI: 0.89, 1.21)	0.65		
Holcomb 2021	Any animal MST Marker	Source water	0	22	0	19	41	Not estimated		Not estimated	
Odagiri 2016	Any animal MST Marker	Source water	28	2	27	3	60	PR=1.04 (95% CI: 0.89, 1.21)	0.65		
Holcomb 2021	Any animal MST Marker	Stored water	0	48	1	45	94	Not estimated		Not estimated	

Table S8.

Unadjusted and adjusted results by study, sample type, and aggregated variables for MST targets (any MST, any general MST, any human MST, any animal MST).

Study	Target	Sample	Positive, Intervention	Negative, Intervention	Positive, Control	Negative, Control	Total observations	Unadjusted Prevalence Ratio	Unadjusted p-value	Adjusted Prevalence Ratio	Adjusted p-value
Fuhrmeister 2020	Any animal MST Marker	Stored water	229	113	253	109	704	PR=0.96 (95% CI: 0.86, 1.07)	0.43	PR=0.97 (95% CI: 0.87, 1.08)	0.57
Boehm 2016	Any animal MST Marker	Stored water	57	188	82	164	491	PR=0.7 (95% CI: 0.51, 0.96)	0.03	PR=0.69 (95% CI: 0.5, 0.95)	0.02
Holcomb 2021	Any animal MST Marker	House soil	2	37	1	43	83	Not estimated		Not estimated	
Fuhrmeister 2020	Any animal MST Marker	House soil	281	30	291	29	631	PR=0.99 (95% CI: 0.94, 1.05)	0.82	PR=0.99 (95% CI: 0.94, 1.04)	0.72
Boehm 2016	Any animal MST Marker	House soil	178	69	186	63	496	PR=0.96 (95% CI: 0.86, 1.08)	0.53	PR=0.96 (95% CI: 0.86, 1.08)	0.51
Holcomb 2021	Any animal MST Marker	Latrine soil	2	28	0	30	60	Not estimated		Not estimated	
Capone 2022 in prep	Any animal MST Marker		12	19	18	37	86	PR=1.18 (95% CI: 0.7, 2)	0.53	PR=1.33 (95% CI: 0.62, 2.86)	0.47
Fuhrmeister 2020	Any animal MST Marker		174	8	182	1	365	PR=0.96 (95% CI: 0.93, 1)	0.03	PR=0.96 (95% CI: 0.93, 1)	0.03
Fuhrmeister 2020	Any animal MST Marker		344	15	358	9	726	PR=0.98 (95% CI: 0.96, 1.01)	0.17	PR=0.98 (95% CI: 0.96, 1.01)	0.19
Boehm 2016	Any animal MST Marker		144	103	147	99	493	PR=0.98 (95% CI: 0.82, 1.16)	0.78	PR=0.97 (95% CI: 0.82, 1.15)	0.7

Table S9.

Baseline covariates by study. Note that Odigari et al. 2016 is not included as data shared from this study were from village water sources and did not have associated covariates from individual households; therefore all estimates for this study are unadjusted.

	Boehm 2016	Reese 2017	Steinbaum 2019	Fuhrmeister 2020	Holcomb 2021	Capone 2021	Capone 2022 in prep.	Kwong 2021
Number of rooms in the household								
Household wealth								
Low	125 (25.2%)	28 (11.6%)	861 (40.9%)	153 (25.6%)	45 (27.6%)	22 (25.0%)	14 (24.6%)	355 (25.4%)
Medium-low	124 (24.9%)	51 (21.1%)	439 (20.8%)	145 (24.3%)	46 (28.2%)	23 (26.1%)	18 (31.6%)	343 (24.6%)
Medium-high	125 (25.2%)	39 (16.1%)	402 (19.1%)	147 (24.6%)	35 (21.5%)	22 (25.0%)	12 (21.1%)	351 (25.1%)
High	123 (24.7%)	65 (26.9%)	403 (19.1%)	152 (25.5%)	37 (22.7%)	21 (23.9%)	13 (22.8%)	347 (24.9%)
Missing	0 (0%)	59 (24.4%)	2 (0.1%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Number of people in the household								
<5	271 (54.5%)	17 (7.0%)	612 (29.0%)	335 (56.1%)	38 (23.3%)	0 (0%)	0 (0%)	783 (56.1%)
5-8	199 (40.0%)	171 (70.7%)	1149 (54.5%)	224 (37.5%)	44 (27.0%)	7 (8.0%)	3 (5.3%)	528 (37.8%)
5-8	199 (40.0%)	171 (70.7%)	1149 (54.5%)	224 (37.5%)	44 (27.0%)	7 (8.0%)	3 (5.3%)	528 (37.8%)
>8	27 (5.4%)	54 (22.3%)	245 (11.6%)	38 (6.4%)	81 (49.7%)	81 (92.0%)	54 (94.7%)	85 (6.1%)
Missing	0 (0%)	0 (0%)	101 (4.8%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Number of rooms in the household								
1-2	0 (0%)	0 (0%)	0 (0%)	0 (0%)	98 (60.1%)	61 (69.3%)	41 (71.9%)	0 (0%)

Table S9.

Baseline covariates by study. Note that Odigari et al. 2016 is not included as data shared from this study were from village water sources and did not have associated covariates from individual households; therefore all estimates for this study are unadjusted.

.	Boehm 2016	Reese 2017	Steinbaum 2019	Fuhrmeister 2020	Holcomb 2021	Capone 2021	Capone 2022 in prep.	Kwong 2021
>3	0 (0%)	0 (0%)	0 (0%)	0 (0%)	65 (39.9%)	27 (30.7%)	16 (28.1%)	0 (0%)
Missing	497 (100%)	242 (100%)	2107 (100%)	597 (100%)	0 (0%)	0 (0%)	0 (0%)	1396 (100%)
Improved roof								
0	8 (1.6%)	0 (0%)	693 (32.9%)	8 (1.3%)	0 (0%)	0 (0%)	0 (0%)	23 (1.6%)
1	489 (98.4%)	0 (0%)	1414 (67.1%)	589 (98.7%)	0 (0%)	0 (0%)	0 (0%)	1373 (98.4%)
Missing	0 (0%)	242 (100%)	0 (0%)	0 (0%)	163 (100%)	88 (100%)	57 (100%)	0 (0%)
Father in agriculture								
0	332 (66.8%)	126 (52.1%)	0 (0%)	419 (70.2%)	0 (0%)	0 (0%)	0 (0%)	952 (68.2%)
1	165 (33.2%)	89 (36.8%)	0 (0%)	178 (29.8%)	0 (0%)	0 (0%)	0 (0%)	444 (31.8%)
Missing	0 (0%)	27 (11.2%)	2107 (100%)	0 (0%)	163 (100%)	88 (100%)	57 (100%)	0 (0%)
Land owned								
0	0 (0%)	97 (40.1%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
1	0 (0%)	117 (48.3%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Missing	497 (100%)	28 (11.6%)	2107 (100%)	597 (100%)	163 (100%)	88 (100%)	57 (100%)	1396 (100%)
Acres of land owned								
Mean (SD)	0.110 (0.128)	Missing	Missing	0.150 (0.206)	Missing	Missing	Missing	0.142 (0.212)

Table S9.

Baseline covariates by study. Note that Odigari et al. 2016 is not included as data shared from this study were from village water sources and did not have associated covariates from individual households; therefore all estimates for this study are unadjusted.

	Boehm 2016	Reese 2017	Steinbaum 2019	Fuhrmeister 2020	Holcomb 2021	Capone 2021	Capone 2022 in prep.	Kwong 2021
Median [Min, Max]	0.0700 [0.0100, 1.23]	Missing	Missing	0.0800 [0.0100, 2.10]	Missing	Missing	Missing	0.0800 [0.0100, 3.15]
Missing	13 (2.6%)	242 (100%)	2107 (100%)	21 (3.5%)	163 (100%)	88 (100%)	57 (100%)	62 (4.4%)
Maternal education								
No education	85 (17.1%)	0 (0%)	0 (0%)	86 (14.4%)	6 (3.7%)	0 (0%)	0 (0%)	207 (14.8%)
Incomplete Primary	0 (0%)	83 (34.3%)	1095 (52.0%)	0 (0%)	38 (23.3%)	0 (0%)	0 (0%)	0 (0%)
Primary	180 (36.2%)	30 (12.4%)	511 (24.3%)	183 (30.7%)	14 (8.6%)	0 (0%)	0 (0%)	449 (32.2%)
Secondary	232 (46.7%)	70 (28.9%)	499 (23.7%)	328 (54.9%)	41 (25.2%)	0 (0%)	0 (0%)	740 (53.0%)
More than secondary	0 (0%)	11 (4.5%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Missing	0 (0%)	48 (19.8%)	2 (0.1%)	0 (0%)	64 (39.3%)	88 (100%)	57 (100%)	0 (0%)
Maternal age								
Mean (SD)	23.7 (5.18)	Missing	26.4 (6.32)	23.7 (5.08)	Missing	Missing	Missing	24.0 (5.03)
Median [Min, Max]	23.0 [15.0, 42.0]	Missing	25.5 [14.9, 47.9]	23.0 [15.0, 41.0]	Missing	Missing	Missing	24.0 [15.0, 43.0]
Missing	0 (0%)	242 (100%)	11 (0.5%)	0 (0%)	163 (100%)	88 (100%)	57 (100%)	2 (0.1%)
Improved wall								
0	78 (15.7%)	0 (0%)	2019 (95.8%)	197 (33.0%)	41 (25.2%)	16 (18.2%)	10 (17.5%)	369 (26.4%)
1	419 (84.3%)	0 (0%)	88 (4.2%)	400 (67.0%)	122 (74.8%)	72 (81.8%)	47 (82.5%)	1027 (73.6%)

Table S9.

Baseline covariates by study. Note that Odigari et al. 2016 is not included as data shared from this study were from village water sources and did not have associated covariates from individual households; therefore all estimates for this study are unadjusted.

.	Boehm 2016	Reese 2017	Steinbaum 2019	Fuhrmeister 2020	Holcomb 2021	Capone 2021	Capone 2022 in prep.	Kwong 2021
Missing	0 (0%)	242 (100%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Improved floor								
0	461 (92.8%)	0 (0%)	1999 (94.9%)	524 (87.8%)	4 (2.5%)	1 (1.1%)	1 (1.8%)	1253 (89.8%)
1	36 (7.2%)	0 (0%)	108 (5.1%)	73 (12.2%)	159 (97.5%)	87 (98.9%)	56 (98.2%)	143 (10.2%)
Missing	0 (0%)	242 (100%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Electricity								
0	234 (47.1%)	34 (14.0%)	1958 (92.9%)	246 (41.2%)	3 (1.8%)	4 (4.5%)	2 (3.5%)	584 (41.8%)
1	263 (52.9%)	202 (83.5%)	147 (7.0%)	351 (58.8%)	160 (98.2%)	84 (95.5%)	55 (96.5%)	812 (58.2%)
Missing	0 (0%)	6 (2.5%)	2 (0.1%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Animal ownership								
Mean (SD)	0.956 (0.206)	0.423 (0.495)	0.899 (0.302)	0.955 (0.208)	0.896 (0.307)	0.966 (0.183)	0.982 (0.132)	0.968 (0.177)
Median [Min, Max]	1.00 [0, 1.00]	0 [0, 1.00]	1.00 [0, 1.00]	1.00 [0, 1.00]	1.00 [0, 1.00]	1.00 [0, 1.00]	1.00 [0, 1.00]	1.00 [0, 1.00]
Missing	1 (0.2%)	29 (12.0%)	0 (0%)	1 (0.2%)	0 (0%)	0 (0%)	0 (0%)	1 (0.1%)