

The Curse of Bad Geography: Stagnant Water, Diseases, and Children’s Human Capital

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Abstract

Waterborne diseases lead to over 6 billion diarrheal episodes per year, with most of the burden on children in low-income countries. We employ hydrological engineering principles to construct a novel measure of stagnant water, crucial to the spread of these diseases. Using a difference-in-differences approach, we estimate the causal effect of stagnant water on the health and cognitive skills of Tanzanian children. A 10 percentage point increase in stagnant water increases local diarrhea incidence rates among children by 30%. Our results also show an immediate reduction in the cognitive abilities of affected children, measured by standardised test scores. The effects on health and cognition are exacerbated by high temperatures and population density, but are completely mitigated by access to safe water and sanitation. We find that two degrees Celsius of global warming could triple the burden of waterborne diseases, but that disease awareness in high-risk locations remains low, which could motivate targeted information campaigns. Our results show how stagnant water exposure in areas with inadequate water and sanitation may result in millions of children failing to reach their cognitive potential.

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1 Introduction

Waterborne diseases rank as the second most common type of disease in the world and lead to more than 6 billion diarrheal episodes per year (Murray et al., 2020), with most of the health burden on children in low-income countries (Prüss et al., 2002).¹ Despite the persistently high prevalence of waterborne diseases and the evidence on long-term impact of childhood health shocks (Currie and Almond, 2011), we have limited knowledge on the consequences of childhood exposure to risk factors of waterborne diseases. The aim of this paper is to evaluate the effect of exposure to an important risk factor of waterborne diseases – stagnant water – on child health and cognitive skills.

Waterborne diseases spread through physical contact with pathogen-contaminated food or water that infect the gastrointestinal system.² Stagnant water enables the spread of waterborne pathogens through the fecal-oral route, and is hence a key risk factor of waterborne diseases, especially in areas with low access to safe sanitation (Bridle, 2021; Prüss-Üstün et al., 2004). Although infection is completely preventable with investments in improved water and sanitation, an estimated 829,000 people – mainly young children – die from diarrhea every year due to inadequate access to clean water and sanitation (Prüss-Ustün et al., 2014; WHO, 2022). The economic historical literature suggests that most of the decline in child mortality in high-income countries in the early 20th century can be attributed to investments in safe sanitation and drinking water (Alsan and Goldin, 2019; Cutler and Miller, 2005). Yet, recent RCTs on sanitation investments in low-income countries show mixed results (Schmidt, 2015), and recent mapping efforts indicate large geographic variation in disease burden even within countries (Reiner et al., 2020). This raises the question of whether exposure to stagnant water may explain this difference, and what effect this exposure has on children’s human capital.

We develop a novel method to predict stagnant water shocks based on established hydrological engineering principles. We use variation in rainfall, evaporation, topography, and soil infiltration from high-resolution satellite data to simulate surface water flow over 90 m grid cells across mainland Tanzania over the period 2010-2017. The treatment variable, *Waterborne Disease Potential* (WDP), is defined as the share of the local area covered by stagnant water in the 8 weeks prior to a child being surveyed. We are the first to produce a high-resolution, time-varying measure of stagnant water occurrence at this geographic and temporal scale, which enables us to match exposure to treatment to a nationally representative survey data on children’s health and cognitive skills. In order to causally estimate the effect of WDP on children’s health and learning we use repeated cross-sectional household survey data and a difference-in-differences identification strategy. For health outcomes, we use the *Demographic and Health Surveys*, specifically three waves of georeferenced data from 1999, 2010 and 2015, with which we estimate effects on short-run health symptoms, such as diarrhea and fever, for children aged 0-5 years. For learning outcomes, we use

¹This statistic also includes food-borne illnesses, since the responsible pathogens spread through both contaminated water and food.

²For example, cholera causes acute diarrhea and is caused by the intake of cholera bacteria, and the Rota virus causes diarrhea and abdominal pain. This is in contrast to mosquito-born malaria which is contracted in the bloodstream from a parasite and typically not classified as a waterborne disease.

data from the Uwezo surveys that provide standardised test scores for school children near annually 2011-2017.

We find that a 10 percentage point increase in WDP leads to a 30% increase in diarrhea incidence, but find no effects on other health outcomes, which suggests that the negative impact of stagnant water is due to waterborne diseases. Access to high-quality sanitation and improved water sources completely mitigates this effect. The effect of stagnant water shocks on diarrhea are larger in warmer and more densely populated locations, which poses challenges with ongoing trends in global warming and urbanisation. We also find that awareness of how to mitigate waterborne diseases does not increase with stagnant water exposure, even in cross-sectional regressions. While suggestive, this information gap may explain why sanitation RCTs have found limited uptake.

Regarding learning, we find that exposure to a 10pp increase in WDP over the past two months lowers average test scores by 7% of a standard deviation. However, this result masks considerable heterogeneity: effect sizes are approximately twice as high in urban compared to rural environments. The negative effect on learning is robust to heterogeneous and dynamic treatment effects ([de Chaisemartin and D'Haultfoeuille, 2022](#)), and is not explained by other confounding factors such as malaria, the disruptive effects of floods, or child labour. We find evidence that the channels through which waterborne diseases affect test scores is a combination of school absence and cognitive impairment. Similar to our child health outcomes, we find that results are larger and more precisely estimated in urban and warmer places.

Following these insights, we produce a back-of-the envelope climate change multiplier of how the burden of waterborne diseases is affected by global warming. Using the latest climate projection data for East Africa ([Ayugi et al., 2021](#)), we estimate that 2°C of global warming may lead to a three-fold increase in the combined severity and frequency of stagnant water shocks and consequently, waterborne disease outbreaks.

Our findings have a number of important policy implications. First, stagnant water shocks can be prevented by improved drainage, water management and urban planning. Second, due to low awareness, stagnant water shocks may be mitigated by short-range forecasts and information campaigns about their potential harm. In the short run, health consequences can be mitigated by targeted treatment in the form of oral rehydration salts, and in the long run by improved targeting of comprehensive investment in water and sanitation.

This paper makes a significant contribution to the measurement of risk factors of waterborne diseases, with three specific advantages over existing methods. First, there is a general lack of the frequency and spatial resolution on stagnant water necessary for large-scale panel data analysis. Satellite data tends to suffer from missing data following increased cloud coverage during wet seasons, and is primarily used for long-term occurrence analysis ([Pekel et al., 2016](#)).³ Meanwhile, available flood maps represent spatially constrained rare events, while our measure is able to capture the intensive margin of day-to-day exposure to stagnant water.

³There is also a potential endogeneity issue to using observed surface water occurrence, since locations may differ in their capability of diverting stagnant water in ways that correlate with our outcomes of interest. In contrast, the within-location variation of our measure is driven by arguably exogenous meteorological variation, primarily rainfall on upstream catchments, which permits a causal interpretation of our estimates.

Second, our treatment variable is specifically tailored to measure the risk of waterborne diseases. Most of the public health literature on waterborne diseases has investigated the association between weather events, such as intense rainfall and high temperatures, and disease outbreaks (Levy et al., 2016). A large and growing economics literature relies on variation in rainfall to instrument for income and health shocks in developing countries (e.g. Maccini and Yang, 2009; Shah and Steinberg, 2017). Using rainfall as an instrument for disease risk, one would risk violating the exclusion restriction, since it would likely affect many other channels that could affect children's health and learning outcomes (Sarsons, 2015; Mellon, 2022). By using variation in stagnant water as our treatment variable, and separately controlling for local rainfall, we are able to disentangle the effects of stagnant water from the potentially confounding effects of weather.

A third contribution with our stagnant water measure is that since our prediction algorithm uses meteorological data, it can readily be plugged in to a short-range forecasting model to predict future disease outbreaks, used in climate change impact analysis, and applied to other settings thus increasing the scope for future external validity.

We contribute to a large economic literature on how shocks, in utero or early life, affect children's human capital and economic outcomes. Existing studies show that children's development is impacted by exposure to rainfall (or drought) shocks (Maccini and Yang, 2009; Shah and Steinberg, 2017), floods (Rosales-Rueda, 2018), parasites (Bleakley, 2007; Miguel and Kremer, 2004), and shocks in general (Currie and Almond, 2011). These studies tend to estimate shocks on a relatively wide early-life window (e.g. the year of birth) and find large long-run effects. We contribute to this strand of literature by investigating how exposure to stagnant water, at a well-specified time and within small spatial windows, affects contemporary health and learning outcomes. This paper is the first to causally address the effect of waterborne diseases on children's human capital accumulation with nationally representative survey data, and at two key stages of development: health shocks in young children, and the effect on cognition for school-aged children and adolescents.

Furthermore, we draw from a growing literature on how climate change will affect public health in the future, mostly focusing on the direct impacts of temperature on mortality (Carleton et al., 2022). Importantly, waterborne diseases are affected by the dynamic interaction of both temperature and rainfall. For example, Levy et al. (2018) in a recent review of the climate and waterborne diseases literature report a need to identify combined dyamic effects of temperature and precipitation. Indeed, the most recent IPCC report stated that climate change will increase the incidence of water-borne diseases with "high confidence", and particularly in regions with limited safe wa-ter access (International Panel of Climate Change, 2022).⁴ We contribute to this literature first by quantifying how a warmer climate will lead to increased *severity* from stagnant water shocks, and then how a future increase in rainfall intensity will increase the *frequency* of these shocks, using the latest climate projections for East Africa (Ayugi et al., 2021) and future rainfall variability (Li et al., 2021).

Recent randomised controlled trials on sanitation investments have found small or no effects on

⁴See the IPCC 2022 Technical Summary B.4.5-6, page 50.

both diarrheal disease and related mortality, sometimes due to low uptake rates (Schmidt, 2015).⁵ This stands in contrast to historical research from the United States and Sweden which shows that investment in safe water and sanitation reduced or eradicated mortality due to waterborne diseases (Alsan and Goldin, 2019; Cutler and Miller, 2005; Knutsson, 2020), and RCTs in water treatment which typically find high reductions in both diarrhea and child mortality (Kremer et al., 2022). We contribute to this literature by analysing to what extent predetermined access to improved water and sanitation mitigates the effects of stagnant water. Consistent with, Duflo et al. (2015) and Alsan and Goldin (2019), we find that water and sanitation investments have to be of sufficiently high quality to effectively mitigate the increase in diarrhea incidence.

Lastly, given the importance of cognitive skills for economic growth (Hanushek and Woessmann, 2012), this paper contributes to a broader literature on geographic determinants of development⁶ by showing how geographically determined exposure to stagnant water affects children's performance. Our contribution lies in introducing a novel measure of waterborne diseases risk and showing how this affects children's human capital today at the micro level, which in turn may explain some of the subnational variation in development in low-income countries. In addition, the consistently stronger effects in urban settings suggest that waterborne diseases may not necessarily be inhibited by general economic development, but may instead be exacerbated if economic development leads to uncontrolled urbanisation without complementary investments in sanitation.

The rest of this paper is organised as follows: Section 2 provides background on the role of stagnant water for waterborne diseases and the current health burden of these diseases in developing countries. Section 3 summarises our data sources and key variables, while Section 4 explains our empirical strategy. Section 5 presents our results, Section 6 provides a battery of robustness checks and Section 7 investigates potential mechanisms. Finally, Section 8 provides a climate change impact analysis while Section 9 concludes the discussion and presents our policy recommendations.

2 Background

2.1 Waterborne diseases: the role of stagnant water

Waterborne diseases are adverse health conditions caused by pathogens that are transmitted primarily through water or food. These pathogens include bacteria, viruses and worms, and common diseases are cholera, typhoid fever, and dysentery.⁷ Symptoms vary depending on the cause, but by far the most common are diarrhea and other gastrointestinal issues, such as abdominal pain (Magana-Arachchi and Wanigatunge, 2020). For most types of waterborne diseases, the incubation

⁵But for an exception, see Duflo et al. (2015) who finds that sanitation investments, when integrated into a comprehensive water and sanitation program, led to a reduction in diarrheal episodes by 30-50%.

⁶Some of the most notable contributions are Gallup and Sachs (2000) who estimate the economic burden of malaria, Easterly and Levine (2003) who investigate the role of exposure to germs on contemporary development, Nunn and Puga (2012) who focus on the role of ruggedness on income today, and Alsan (2015) who estimate the effect of Tsetse fly on contemporary development.

⁷See Table B.1 for a summary of some of the most commonly occurring diseases and symptoms, by type of pathogen.

period is a few days. For example, cholera takes between 2 hours to 5 days for a person to show symptoms after first ingesting contaminated food or water (Azman et al., 2199). Symptoms can last from a few days (e.g. most viruses) to years (parasitic worms), but most common is days or a few weeks (Percival et al., 2014), and disease outbreaks tend to occur in a matter of weeks after the first infection.

Waterborne pathogens occur naturally in aquatic environments, but tend to multiply under favorable conditions, such as in stagnant, warm and polluted water (Bridle, 2021). Stagnant water, in contrast to flowing water, enables the formation of biofilms, the clustering of waterborne pathogens into communities that enable exponential growth and survival (Wingender and Flemming, 2011), which can develop in a matter of hours or days (Ling et al., 2018).

Stagnant water becomes an especially important transmission link in crowded environments with poor access to sanitation, such as urban slums. For most waterborne diseases, the faecal-oral channel, where contaminated human feces spread to water or food, is the most important channel behind outbreaks (Magana-Arachchi and Wanigatunge, 2020). That is, waterborne pathogens survive from ingestion to feces, and can in this way, when sanitation is poor, enter new water or agricultural environments. The formation of stagnant water through surface flooding enables a direct transmission link between an unsanitary environment and human exposure (Prüss-Üstün et al., 2004).⁸ Thus, the spread of waterborne disease is inextricably linked to Water, Hygiene and Sanitation Practices (WASH) and improving these can break the faecal-oral transmission channel.

Most empirical work on the role of climatic drivers of waterborne diseases is in the form of time series-analyses and case reports, and typically find seasonal correlations between heavy rainfall and high temperatures and local disease outbreaks (Levy et al., 2016). Effect sizes tend to be larger in warm areas, as this is often necessary to induce pathogen growth, and areas that have been dry, as this leads to a buildup of pollutants and fecal matter (Kraay et al., 2020). However, few papers use variation in exposure to stagnant water, our main hypothesised transmission mechanism, explicitly. Case studies have relied on variation in river levels as a proxy for stagnant water and analysed impacts on diarrhoeal incidence at the local community level (Alexander et al., 2018; Hashizume et al., 2008), finding that this is more informative than rainfall alone. A recent working paper analysed the effect of rainfall on a cholera outbreak in Dar-Es Salaam in 2015 and found that most of the effect of rainfall was in locations most at risk of floods (Picarelli et al., 2017).

2.2 The waterborne diseases burden: persistence and inequalities

Waterborne diseases rank as the second to fourth most debilitating disease in low-income countries depending on the measure used, but typically only represent a minor share of the disease burden in high-income countries. This difference is believed to be partly due to low access to safe sanitation and water, and it is estimated that 829,000 people die from a lack of safely managed drinking water each year (UN, 2022).

⁸See the diagram in Figure B.1 for a complete overview of transmission mechanisms of waterborne diseases through the faecal-oral channel.

Despite this, low-income countries such as Tanzania has made great strides in reducing childhood diarrhoea mortality, with the mortality rate declining by 89% from 1980 to 2015 ([Masanja et al., 2019](#)). This is mostly attributed to the introduction of oral rehydration salts (ORS), while only 3% of the reduction is attributed to improved access to safe water and sanitation. The consequences of this is that waterborne diseases remain as prevalent as ever, as seen in [Figure B.2](#). This is largely because ORS, in contrast to safe water and sanitation, do not prevent waterborne diseases but only mitigate the worst consequences. Repeated exposure to diarrheal episodes may affect gastrointestinal functioning, and increase the risk of malnutrition and stunting. Moreover, exposure to diarrhoeal diseases in early life is associated with lower cognitive ability in later life, even independent of stunting ([Niehaus et al., 2002](#); [Pinkerton et al., 2016](#)). Hence, there is a great urgency to address not only the consequences of waterborne diseases on mortality but also their repeated occurrence.

While waterborne diseases are particularly disruptive for children under five, older children are also at risk. Prevalence varies smoothly with age, with a minimum occurring typically around the age of 15-20 years, as [Figure B.3](#) shows. Thus, we would also expect school-going children in the ages of 6 and above to also be affected by stagnant water shocks increasing the risk of contracting waterborne diseases.

In addition, there is large geographic variation in the waterborne disease burden also within low-income countries, which can partly be explained by the fact that local climate and geography play an important role for waterborne disease outbreak. [Reiner et al. \(2020\)](#) find that high-prevalence areas in Tanzania carry a three times greater burden than low-prevalence areas. In this regard, Tanzania is representative of many other low-income countries that face a continued persistence in waterborne diseases and an uneven distribution within the country.

For more context on waterborne disease burden in Tanzania specifically, see [Appendix C. I](#) and for a recent history on prevention and treatment strategies in Tanzania, see [Appendix C. II](#).

3 Data

3.1 Geographic data

To generate our treatment variable, “waterborne disease potential” (WDP), we construct a high-resolution hydrological model to simulate the flow of surface water across all of Tanzania. Our aim is to create a treatment variable with the highest possible geographic resolution that is feasible enough for large-scale simulation, using harmonized high-quality data as input to hydrological and hydraulic model components. To this end, we use the latest available global gridded datasets at the highest possible resolution, as shown in [Figure 1](#). For topographic data, used as input for the hydraulic flow component, we use data from the Shuttle Radar Topography Mission ([Farr et al., 2007](#)). Rainfall and evaporation data, which allows water to enter and leave the model, are extracted from ERA5 reanalysis data. ERA5 is a dataset produced by the European Centre for Medium-Range Weather Forecasts using a combination of weather station and satellite data, coupled to a physi-

cal weather forecasting model, to generate a consistent, high-resolution long-term record of past weather and climate conditions (Hersbach et al., 2018). One advantage this has over historical data from rainfall measurement stations is that it will be less sensitive to measurement error stemming from a lack of rainfall station infrastructure, which might be correlated with local economic development. Finally, to account for differences in soil infiltration, we use the ISRIC 2.0 global soil database (Poggio et al., 2021), which contains high-resolution data on soil composition across the globe. Appendix A. I contains detailed descriptions of each of these data sources, while Section 4.1 describes the methodology we use to simulate stagnant water across Tanzania over time.

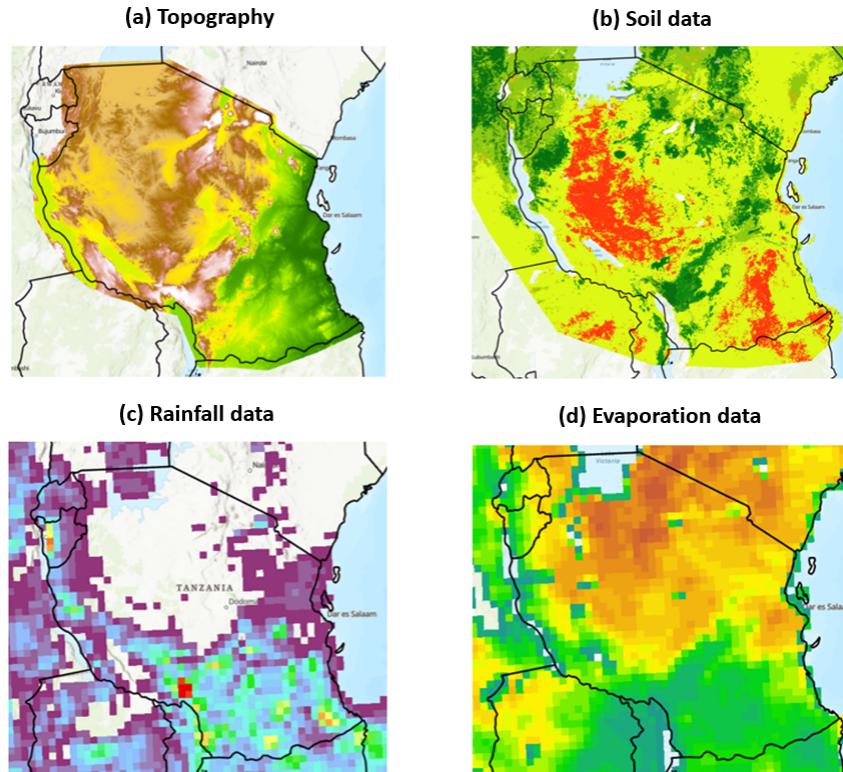


Figure 1
Data sources used to construct the WDP measure

Note: The hydrological model uses as input four high-resolution gridded geographic datasets: (a) topographic data from SRTM, (b) soil infiltration data from the ISRIC 2.0 soil database, and (c) hourly rainfall and (d) potential evapotranspiration from ERA-5 reanalysis data.

In order to investigate treatment heterogeneity in terms of local climate, we use historical annual mean precipitation and temperature from the WorldClim v2 database (Fick and Hijmans, 2017), covering the period 1970-2000.

3.2 Demographic and Health Surveys (DHS)

To explore health-related outcomes we use the Demographic and Health Surveys (DHS), specifically the three waves with georeferenced data: 1999, 2010 and 2015⁹.

⁹The DHS Program is funded by USAID and has collected survey data with a particular focus on children and women's health since 1984. For more information, visit <https://dhsprogram.com/>

DHS are repeated cross-sectional surveys representative at the national level, with two important features for our analysis. First, DHS provides spatial coordinates of villages (so-called 'clusters'), although for confidentiality reasons the coordinates are randomly shifted 0-5 km. Since village locations will differ across waves but we want to exploit the within-location variation in WDP over time we assign each household to a 50x50 km static grid cell. The cells are large enough to capture the variation in the share of stagnant water over time, but arguably small enough to control for most of the unobserved geographical variation.

The second advantage of DHS data is that it reports on health issues that children may have had in the last *two weeks* leading up to the survey date. We can therefore accurately capture short-term changes in health. DHS collects information on children's recent health issues (for example, "has your child had diarrhea in the past two weeks?") as well as basic anthropometric features such as height for age and weight for age. Importantly, since the children surveyed in DHS are below five years old, they are likely more sensitive to waterborne diseases than older children of school age ([Khalil et al., 2018](#)). Assuming that the risk of contracting waterborne disease decreases with age, we can interpret these results as upper-bound estimates for children of school-going age.

3.3 Uwezo surveys

To analyse learning outcomes we use the Uwezo surveys. These are collected by the NGO *Twazeza* and are large-scale assessments of school-age children in Kenya, Uganda and Tanzania. The surveys are administered to schools, communities and households, and are repeated cross-sections. We utilise surveys from the survey waves in the years 2011, 2013, 2014, 2015 and 2017, and all survey waves are representative at the district level. In our paper, we employ the household surveys for children's test scores, and school surveys for absence.¹⁰ For each household, there is information on household wealth and assets, as well as basic socio-economic information such as the child's age, year of schooling, whether they are currently enrolled, mother's age and mother's schooling.

There are three features of the Uwezo surveys that are crucial for our purpose of studying the effect of stagnant water on learning. First, all school-aged children (aged 6-16) in the surveyed household are tested in basic Mathematics, English and Kiswahili by being administered a random sample of question cards in each subject. This allows us to measure downstream effects on learning and cognitive ability, in addition to first stage effects on health. Another advantage is that test scores are collected for all children of school-age in the household which makes us able to capture the effects also on those children who drop out of school due to e.g. ill health. Lastly, the surveys are administered by Twazeza. The NGO works largely independently of the Tanzanian government/authority, and the tests have no bearing on the school grades of the children or the evaluation of teacher performance. Hence, there is little reason to believe that parents or schools would influence performance during the tests, which may be an issue when using test scores from school-administered tests.¹¹

¹⁰For more information, visit <https://uwezotanzania.or.tz/>

¹¹For a more extensive background on the education system in Tanzania and the current status of Tanzanian children's learning, see [Appendix C. III](#).

Depending on how well the child answers the question sets, the children get allocated a score: The scores are discrete and are 1-5 for reading in English and Kiswahili, and 1-6 for Mathematics, except in wave 2015 where scores are given between 1 and 9. For example, scores in reading from 1 to 5 are based on whether the child can recognise letters (1), recognise words, read a paragraph or read a short story (5)¹². We have no reason to think that the effects would be different for verbal and quantitative abilities, and hence we construct our main outcome as the mean age-standardised test score across the three subjects. We standardise each child's test score by wave to have mean zero and standard deviation one, and then take the average and standardise again such that

$$\text{Mean standardised test scores} = \frac{\text{Avg Score} - \text{Mean}(\text{Avg Score})}{\text{SD}(\text{Avg Score})} \quad (1)$$

where *Avg Score* is the average for each child across the three subjects.¹³ We thus end up with a continuous measure of children's current performance and learning which is comparable across waves. In our results, unless otherwise stated we refer to the mean standardised test score as "test scores". Test scores are missing for 8% of our sample which we exclude from our analysis. If these differ systematically from the non-missing values this could bias our result. For example, if the test scores are missing because children did not get tested as they were ill, we may underestimate the effect of WDP on test scores.¹⁴

The second important feature of the Uwezo surveys is that we similar to DHS know the exact survey date for the vast majority of observations. The third important feature is that the data is georeferenced at the *ward*-level instead of at the village-level, which means that we can use ward-level fixed effects directly, instead of imposing a grid cell structure as we do for the DHS data¹⁵. To summarise, for health outcomes using the Demographic and Health surveys we create 50^2 km grid cells that contain villages from different waves, while for the Uwezo surveys we have data at the level of *wards*, whose boundaries are fixed over time.

¹²In Mathematics, scores are based on whether the children can count, recognise numbers, order numbers, add, subtract or multiply.

¹³That is, for each child and $\text{Subject} \in \{\text{English}, \text{Kiswahili}, \text{Maths}\}$ we standardise each score by wave:

$$\text{Subject}_{\text{std}} = \frac{\text{Score} - \text{Mean}(\text{Score})}{\text{SD}(\text{Score})}$$

where mean and standard deviation, SD, are wave-specific. We then take the mean of the standardised subject-specific variables and standardise, again by wave, to construct *Avg Score*:

$$\text{Avg Score} = \frac{\text{English}_{\text{std}} + \text{Kiswahili}_{\text{std}} + \text{Maths}_{\text{std}}}{3}$$

¹⁴Assuming that children too sick to take the test would have performed relatively worse, excluding these children would attenuate our results to zero. We test if WDP predicts the degree of missing values in test scores (for each separate subject) with our main model specification but we find no systematic correlation.

¹⁵The average ward area is 253 square kilometres, and the wards are of similar size as e.g. UK parliamentary constituencies.

4 Empirical strategy

4.1 Modelling waterborne disease potential

We are interested in estimating the causal effect of unsafe water on children’s health and learning. Since disease incidence likely correlates with unobserved geographic factors, we require exogenous variation in exposure to unsafe water in order to recover a causal estimate. A naïve approach would be to simply use variation in rainfall to proxy for this risk, as extreme rainfall events are associated with disease outbreaks (Levy et al., 2016; Lo Iacono et al., 2017). However, this is problematic due to two reasons. First, local stagnant water is often the result of rainfall on *upstream* catchments rather than just on the local area. Second, local rainfall can affect children’s health and learning through multiple pathways not necessarily related to waterborne diseases (Mellon, 2022). To improve upon the rainfall measure, we use well-established hydrological modelling principles and high-resolution geospatial data to construct a nationwide hydrological model, which we then use to simulate the overland flow of water and the formation of stagnant water pools. Since it is the stagnant water that enables the growth of waterborne pathogens (Eisenberg et al., 2013; Leclerc et al., 2002), and we want to use variation in this measure as our treatment, we remove any permanent water bodies and free-flowing water from the simulation output.

As input to the model, we use the global gridded geographic datasets described in [Section 3.1](#) and [Appendix A. I](#). Simulations are run at a spatial resolution of 90 m, which is orders of magnitude smaller than the ward level, while being feasible enough to enable simulation over the time scale of months at the country level.¹⁶ For more details on the computational scheme, [Appendix A. II](#) provides a schematic of the input data and algorithm used to model stagnant water.

We measure exposure to stagnant water (WDP) at the local level: for DHS we average over a circle with a radius of 10 km¹⁷ where the centroid is the village coordinate, and for Uwezo surveys we compute the average over each ward. We define WDP for each location and survey year, $S_{l,y} \in [0, 1]$, as the time average of the share of location l’s area, A_l , covered by stagnant water $A_{S,t}$ at day t over the past n days from the date of the visit:

$$S_{l,y} = \frac{\sum_{t=1}^n \frac{A_{S,t}}{A_l}}{n} \quad (2)$$

[Figure 2](#), top row, shows the spatial distribution and resolution of the georeferenced locations in the DHS and Uwezo data. In the bottom row, we provide a quasi-three dimensional visualisation of the output from the hydrological model for an arbitrary point in time, as a first validation that our measure captures that stagnant water pools tend to form in low-lying areas prone to accumulating

¹⁶The runtime of hydrodynamic simulations are very sensitive to the level of resolution and typically with the *cube* of the spatial resolution. However, a recent evaluation of large-scale 2D hydrodynamic simulations for several European rivers show that resulting flooded areas and water levels are insensitive to variations in spatial resolution, once it is finer than 100 m (Dazzi et al., 2021; Falter et al., 2013, 2016). This is important as it allows us to feasibly simulate the formation of stagnant water across a whole country such as Tanzania over time.

¹⁷The choice of radius length is partly to account for the fact that the village coordinate is shifted 0-5 km, and partly to create an area close to the average ward size.

water. A more intense red colour denotes a higher value of $S_{l,y}$. Here we can see that stagnant water has been trapped in the lowlands next to the Lake Victoria coast, and in the valleys of a group of mountains.

In our main specification, we compute WDP as the time average over the last *eight weeks* prior to the survey date for each observation. We choose eight weeks mainly for two reasons. First, by limiting the analysis to within eight weeks, we hope to capture the short-run dynamics of disease outbreaks^{[18](#)} and decrease the likelihood of our measure capturing other confounding factors, such as effects on agricultural productivity. Second, since we want to estimate the effects on children's learning and we do not necessarily want to capture the acute effects of illness, we look back over eight weeks to include children who got sick and then recovered^{[19](#)}. In later robustness checks, we later relax this and find effects of similar magnitude and significance over the whole range of exposure over the last 1 to 12 weeks, which is the available extent of our data on stagnant water.

4.2 Validating Waterborne Disease Potential

To verify that our measure actually captures variation in surface water cross-sectionally and over time, we perform two validation exercises. First, using satellite data spanning 1989-2019 we find that WDP at the ward level is in accordance with satellite data, especially for non-permanent surface water, which is exactly what we want to capture with our treatment variable. The model performs best for the treated locations^{[20](#)} which sees the largest dynamic variation in stagnant water, where our measure explains 18 to 39 per cent of the variation in satellite-measured surface water for transient water. This reassures us that our measure accurately captures the spatial variation in exposure to stagnant water across Tanzania.

To validate that our treatment also captures the *dynamic* variation within each location we rely on reported data at the household level from DHS. Conditional on location fixed effects, we find that exposure to WDP reduces the time to the nearest source of surface water, such that 100% stagnant water coverage would mean close to zero distance to the nearest source of surface water. See [Appendix A. III](#) for the cross-sectional validation using satellite data and [Appendix A. IV](#) for the dynamic validation using DHS data, as well as for a detailed methodology of the validation procedure.

4.3 DiD model specification

We estimate the following difference-in-differences model for outcome Y of individual i residing in location l , surveyed in calendar month m in the year y :

$$Y_{ilm} = \alpha_l^1 + \alpha_y^2 + \alpha_m^3 + \delta S_{ly} + \gamma R_{ay} + X'_{ily}\beta + \varepsilon_{ilm} \quad (3)$$

¹⁸ Waterborne pathogens can grow and infect humans within days and even hours under ideal conditions, see [Appendix B. II](#) for more details.

¹⁹ We think it is unlikely that children under acute illness were subjects to the test, even if they took the test in their home. We also do not find any association between WDP and missing data on test scores

²⁰ Defined as those who switch in and out of treatment when using a binary indicator.

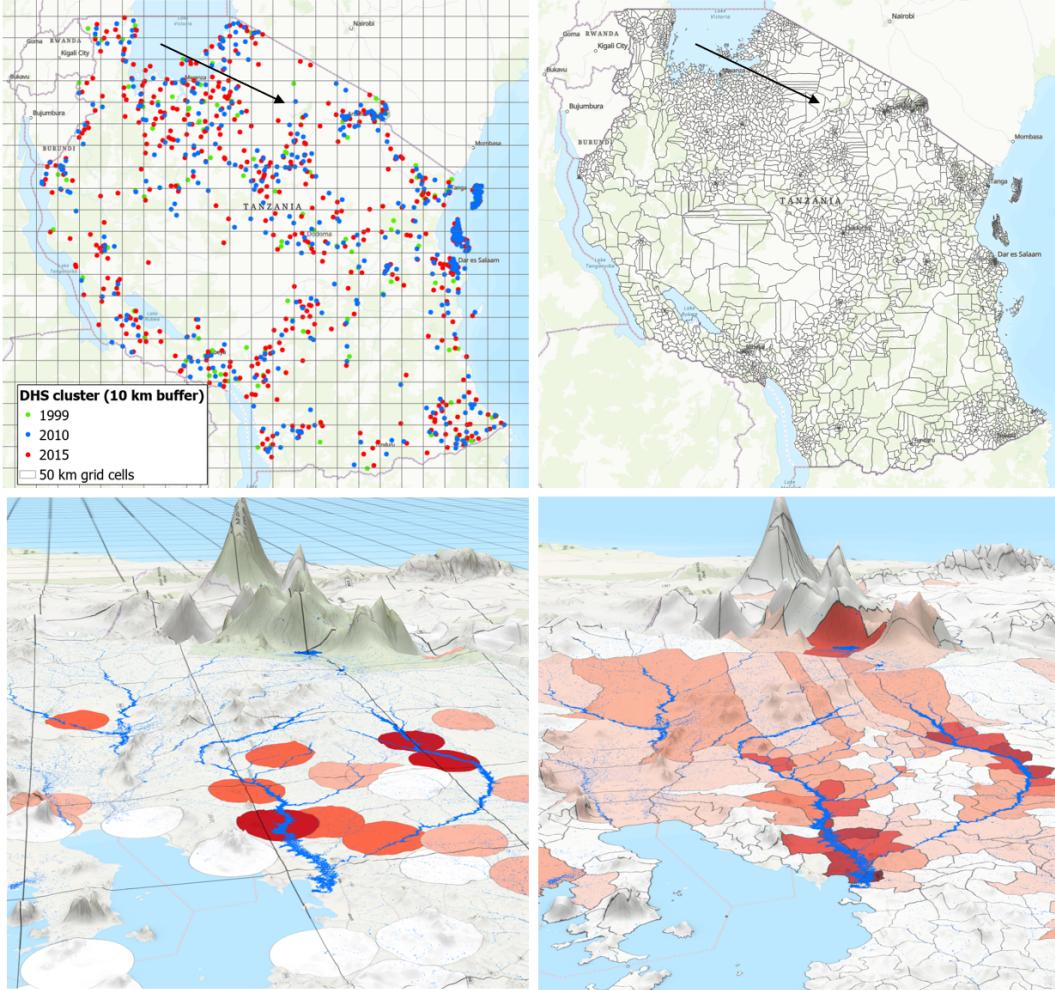


Figure 2
WDP with DHS and Uwezo surveys

Note: This figure displays the output of our simulation which contains our main treatment variable, WDP, in one particular time step. Here we also highlight the difference in output between DHS (left) and Uwezo (right). In the top row, we display the available local area information we have. For DHS clusters we have different locations across waves thus necessitating the $50 \times 50 \text{ km}^2$ gridcells. For the Uwezo surveys, we have time-invariant wards. The bottom row shows the output for both types of spatial information for a subset of Tanzania (roughly indicated as the view along the black arrow with Lake Victoria towards the bottom of the figure). For DHS, treatment will be a weighted average of WDP intensity within the gridcell. For Uwezo, treatment is always given as a function of ward area.

where δ is the main coefficient of interest: $S_{ly} \sim (0, 1)$ is our WDP measure in each ward in the eight weeks prior to the date of the survey. Hence, δ captures the causal effect of an individual i being exposed to a higher likelihood of waterborne diseases in their location l in year y . Since the WDP share always lies between 0 and 1, the value of the coefficient presented in results below is the treatment effect when the stagnant water share is 100% of the local area – an unrealistic scenario. Thus, for interpretation of the results we refer to the coefficient divided by ten, reflecting a 10pp increase in WDP²¹.

Next, as the share of stagnant water over time is potentially affected by local rainfall, and local rainfall may also affect the outcome, we include the last eight weeks of rainfall at the local area

²¹While this is relatively uncommon, it is far from extreme. In the DHS data, we find this to occur in 5% of the sample, which is a far greater frequency than that of a devastating flood or other natural disasters.

level for each wave, R_{ly} , in our baseline specification. We control for time-invariant unobserved differences across wards with ward fixed effects, α_l^1 , as well as survey-year fixed effects α_y^2 (equivalent to survey wave) and calendar month fixed effects α_m^3 to absorb seasonality effects, which have been shown to be informative of waterborne diseases (Reiner et al., 2020).

Lastly, we also include a vector of controls in X on the individual level (gender, age, whether the mother has secondary education or higher, the mother's age, and an index for household wealth). We impute missing values within these variables as the sample mean.

To explore potential heterogeneities we also interact the variable of interest, S_{ly} with indicators for e.g. sanitation quality, an urban-rural indicator and other relevant heterogeneity dimensions. Formally, we estimate the triple DiD:

$$Y_{ilmy} = \alpha_w + \alpha_y + \alpha_m^3 + \delta_1 S_{ly} + \delta_2 WASH_{ily} + \delta_3 S_{ly} \times WASH_{ily} + \gamma R_{ly} + X'_{ilmy} \beta + \varepsilon_{ilmy} \quad (4)$$

where WASH is a binary indicator of sanitation status, such as whether the household has a toilet. Here, we are interested in δ_1 , δ_2 and δ_3 . The parameter δ_1 is the effect of WDP on our outcome when WASH=0, δ_2 is the direct effect on Y_{iwy} from WASH. The key contribution of this model is δ_3 , which is the coefficient on the interaction term. This estimate will give us the contribution to the outcome of the WASH variable through the effect of WDP. We will carefully discuss potential sources of endogeneity in this interaction variable, but we nonetheless believe these results provide important, if sometimes only causally suggestive, evidence.

4.4 Descriptive statistics

[Table 1](#) provides summary statistics of the DHS and Uwezo surveys, providing information on the individual and household level in Panel A and for the local area level in Panel B. For DHS, we have information on 253 grid cells and on average 420 clusters (villages or neighbourhoods) per wave. There is large variation in household wealth and sanitation, and 78% of clusters are rural. Overall, WDP has a strong mode at zero, where the average level is 1.7%, but there is large variation (see also [Figure D.1](#) in [Appendix D](#) for a graphical representation of the distribution).

For the Uwezo surveys in the bottom half of [Table 1](#), we include our main outcome, standardised test scores. The test scores are standardised with mean zero and standard deviation one by wave, thus the total sample has a slightly lower mean. Children are on average 11.1 years old, and 46% are girls. Households are relatively large with seven people on average. Mothers have a similar education to other national surveys (see [Appendix C. III](#)) where 24% have at least a secondary education. We also provide a wealth index based on normalised principal components of household assets, which is normalised to have mean zero and standard deviation one, by each wave. The components in the wealth index are the type of wall, whether the household has a radio, television, bicycle, motorbike, cattle, or electricity. Next, we see that 73% of households have any type of toilet. In panel B we provide ward-level characteristics on the 3876 wards. 84% of the wards are rural. In our sample, a ward includes approximately 20 households sampled per ward, but there is

a large variation in both the number of households and villages sampled.

Table 1
Summary statistics: DHS and Uwezo

	Mean	SD	Min	Max
<i>Demographic and Health surveys</i>				
<i>A. Household and child characteristics</i>				
Birth Order	3.75	2.544	1	17
Girl	0.50	0.500	0	1
Age	1.93	1.416	0	4
Mother's age	29.3	7.108	15	49
Mother total fertility	4.12	2.588	1	17
Household wealth index	2.88	1.382	1	5
Sanitation - none	0.23	0.420	0	1
Sanitation - unimproved	0.46	0.498	0	1
Sanitation - shared	0.056	0.230	0	1
Sanitation - improved	0.26	0.438	0	1
<i>B. Cluster/Grid Cell characteristics</i>				
WDP [0.1]	0.017	0.0522	0	0.512
Local precipitation (cm)	0.0081	0.0101	0	0.223
Urban share of clusters	0.22	0.414	0	1
Num. villages/wave	493.6	146.5	176	608
Num. households/wave	324.4	21.98	303	351
Grid cells: 253				
Obs: 21,471				
<i>Uwezo surveys</i>				
<i>A. Household and child characteristics</i>				
Test score (std)	-0.020	0.997	-4.481	4.239
Woman	0.46	0.499	0	1
Age	11.1	2.792	6	16
Mother's Age	36.3	5.903	16	60
Mother's Edu \geq Sec	0.24	0.408	0	1
Wealth (index)	-0.0064	1.584	-2.230	24.58
Children in household	3.32	1.873	1	20
<i>B. Ward characteristics</i>				
WDP [0.1]	0.013	0.0361	0	0.812
Local precipitation (cm)	4.48	6.437	0	226.9
Rural ward	0.84	0.370	0	1
Ward area (sq.km.)	255.9	599.3	0.111	11437.0
Wards: 3876				
Schools: 15,428				
Obs: 386,005				

Note: Summary statistics of mean, standard deviation, minimum and maximum of each variable. Panel A displays statistics across individuals, while Panel B displays statistics at the local area level.

To get an overview of the spatial distribution of our treatment measure we also present [Figure 3](#). While this is only representative of the time periods covering the Uwezo surveys, the distribution of treatment intensity compares favourably to the historical spatial distribution of cholera incidence, which has shown a higher incidence in the Lake Victoria region, south-east, and north-west part of the country, where we also find greater likelihood of stagnant water forming ([Nkoko et al., 2011](#)). The distribution of stagnant water in [Figure 3](#) is also similar to the distribution from a recent ef-

fort to model the global subnational distribution of diarrheal diseases using Bayesian geostatistical methods (Reiner et al., 2020), see Figure B.4 for a map of this data. There is a large variation in the ward area, which correlates negatively with population density. We also see that WDP (measured as the average over waves) tends to concentrate in smaller wards.²² There is relatively little correlation between distance to rivers and WDP, which is precisely what we would expect since WDP is a function of *stagnant* and not free-flowing water.

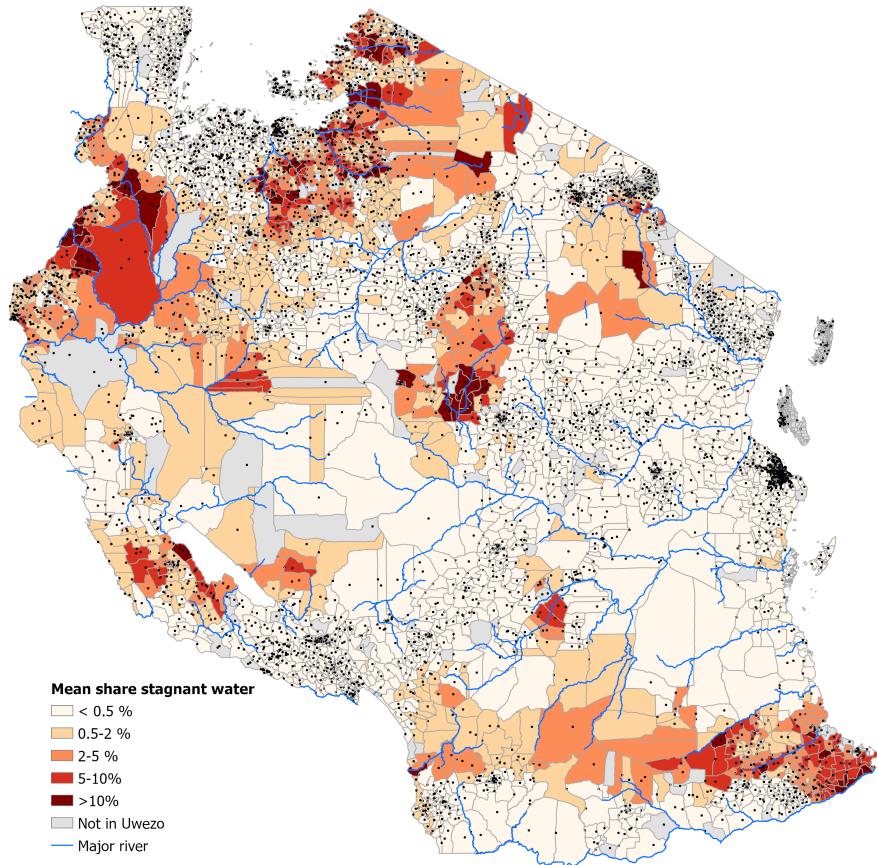


Figure 3
Spatial distribution of WDP

Note: This map shows the geographic distribution of wards in Tanzania, together with population density (each dot represents 10,000 people), major rivers, and the mean share of stagnant water, our main treatment measure, in each ward in our sample period. Note that the mean share of stagnant water is only representative of the specific months and years represented in our Uwezo survey waves, which is the period for which we ran our hydrological model, and is not indicative of the long-term mean values for Tanzania.

5 Results

5.1 Effects of waterborne disease potential on health

In the first set of results we test whether the channel we expect WDP to act through is indeed waterborne diseases and if there are effects on health. To explore the health mechanism we make use of three waves (1999, 2010, 2015) of the Demographic and Health Surveys.

²²See Appendix D for a longer discussion.

We first explore how well the measure we call WDP captures waterborne disease incidence. While DHS does not test for waterborne diseases specifically, they measure important physical attributes of children, such as weight for height, and ask about recent health issues and symptoms of ill health. We make use of these variables to test our main hypothesis that WDP affects waterborne disease incidence. We also conduct placebo checks on the effect of WDP on other health channels. In [Table 2](#) we estimate our main DiD specification. In Panel A, each column represents a different outcome which we estimate on the full sample. The outcomes in the first two columns are outcomes most plausibly affected by waterborne diseases: diarrhea, which is the symptom most closely associated with waterborne disease, and weight for age (since severe diarrhea tends to affect weight). The following four columns in Panel A are outcomes that should not be affected by the waterborne disease to the same degree: Fever, cough, anemia and height. Fever might e.g. be positive if our results are driven by increased malaria incidence, or other mosquito-born illnesses such as dengue and Rift Valley fever, as fever is one of the main short-term symptoms of malaria. Cough is mainly associated with respiratory disease, while anemia and height are both long-run outcomes that should not be affected by WDP since this measure is defined as the change in stagnant water in the past few weeks. All health outcomes are equal to one if the child has had the health issue at any point in the past two weeks, while weight, anemia and height are tested at the date of the survey.

We find a large and statistically significant effect on the probability that the child has had diarrhea: A ten per cent WDP increases the probability of the child having had diarrhea recently by 2.75%. Alternatively, a one standard deviation increase in WDP increases the probability that a child has had diarrhea in the past two weeks by 1.4 percentage points, which is an increase of 11% relative to the mean diarrhea incidence. The coefficient on weight is negative as expected but not statistically significant. The remaining outcomes that represent placebo checks are reassuringly estimated close to zero and statistically insignificant..

As further placebo checks, in Panel B of [Table 2](#) we estimate the effect of WDP on diarrhea related to how likely it is that the child received contaminated water. In columns 1-2 we estimate the effect of WDP on diarrhea and split the sample by whether the child is breastfeeding. We hypothesise that children who are breastfeeding are less likely to have received food or water from other sources which should minimise the risk of contracting diarrhea from waterborne diseases. In these estimations we restrict the sample to children 24 months and younger, since breastfeeding at an older age is rare. We estimate that for children who do *not* currently breastfeed, 10% of WDP increases the probability of the child having had diarrhea recently by 5.1pp (one sd of WDP increases diarrhea by 2.6pp). For children who breastfeed the coefficient is small and statistically insignificant. While breastfeeding is not randomised across children and these mothers or children likely differ in more than this aspect, the large difference in the effect of WDP is highly suggestive of a link between WDP, water and diarrhea, and consistent with public health advice to breastfeed to reduce diarrheal risk ([Keskin et al., 2017](#); [Bhandari et al., 2003](#)).

In columns 3-4, we perform the same estimation of WDP on diarrhea but instead divide the sample by whether the child has been given plain water in the past 24 hours. As is consistent with WDP leading to an increase in the likelihood of water to contain pathogens, children who

Table 2
The effect of WDP on health, and by rehydration source

	(1)	(2)	(3)	(4)	(5)	(6)
<i>Panel A. Health and physical outcomes</i>						
	WBD					
	Diarrhoea	W.Height	Fever	Cough	Anemia	Height
WDP	0.297*** (0.0910)	-5.744 (5.653)	-0.00808 (0.130)	0.0386 (0.130)	0.0889 (0.0742)	14.07 (16.32)
Mean DV	0.13	88.49	0.22	0.21	0.40	92.12
Obs.	19,399	18,152	19,468	19,469	21,108	18,783
Clusters	252	252	252	252	252	252
<i>Panel B. Health and water</i>						
	Diarrhoea				Fever	
	Not Breastfed	Breastfed	Not water	Water	Not water	Water
WDP	0.512*** (0.139)	0.149 (0.130)	0.0275 (0.188)	0.324*** (0.106)	0.183 (0.346)	0.000968 (0.194)
Mean DV	0.11	0.14	0.13	0.13	0.22	0.22
Obs.	7,596	11,800	2,366	11,003	2,367	11,019
Clusters	251	250	195	250	195	250

Note: Standard errors in parentheses clustered on DHS cluster/village level. WDP is the share of the area covered in stagnant water in the eight weeks preceding date of survey. * p<0.01 ** p<0.05 *** p<0.1. In panel A: Columns 1-2 displays outcomes plausibly affected by waterborne disease. In column 1, the dependent variable = 1 if child has suffered from diarrhea the past two weeks, and column 2 weight for age as percentile of wave median. Columns 3-5 are placebo outcomes of health issues less associated with waterborne disease: Fever, cough and anemia (note anemia = 1 for mild to severe measure, thus the high mean). Column 6 records height in cm, also less likely to be affected by current waterborne disease. All health issues (diarrhea, fever and cough = 1 if child has had in past two weeks). In Panel B, the first four columns record the effect of WDP on diarrhea with different subsamples: By whether the child is breastfed (columns 1-2, only \leq 24 months children used in this sample), and whether the child was given plain water in the past 24 hours. Columns 5-6 estimates the effect on fever from WDP when dividing the results by whether the child has been given water. Cell, Wave, Calendar month fixed effects and sum of past eight weeks precipitation used in all estimations. Individual level controls include birth order, multiple birth, gender, age, mother's age, total fertility of mother, toilet type.

have been given water recently have a 30pp higher likelihood of having had diarrhea recently and this effect is statistically significant. For the children who have not been given water recently, we estimate a coefficient of -0.08, and it is not statistically significant. Here too, the results suggest a direct link between WDP, water and the likelihood of contracting diarrhea. As a final placebo check, in columns 5-6 we again run the same specification and split the sample by whether the child was given water, but change the outcome to whether the child has had a fever recently. If the effect on fever looks similar, it might suggest a general issue with health in the community, or some interaction with the probability of contracting malaria. However, for both subsamples of children we find that the coefficient is small and not statistically different from zero. We take this as further support that WDP accurately measures risk factors in water becoming contaminated with waterborne pathogens.

5.1.1 Heterogeneity by access to improved water and sanitation

Next, we turn to what extent WASH practices can mitigate or worsen the spread of waterborne diseases from stagnant water. Existing literatures both in economics and public health emphasise the importance of the faecal-oral route in spreading waterborne diseases ([Troeger et al., 2018](#); [Magana-Arachchi and Wanigatunge, 2020](#)), and in concordance with these, we find that access to high-quality sanitation completely mitigates the effects of stagnant water.

We classify household toilet types reported in the DHS into four categories: No facilities, Unimproved sanitation, Shared facilities, and Improved sanitation, based on the UN Sanitation ladder classification ([Kvarnström et al., 2011](#)).²³ We estimate the effect of WDP on diarrhea in one estimation, interacting WDP with each category of the sanitation ladder, letting the lowest rung of sanitation – no facilities and open defecation – represent the baseline. As in previous analyses of WASH practices, toilet types are not randomly allocated to the household. However, given the existing evidence on the importance of the faecal-oral channel in spreading waterborne diseases, and our estimated results on the link between WDP and diarrhea, we expect a direct link between sanitation and waterborne diseases. Furthermore, we are mostly interested in the interaction effect between sanitation quality and time-varying exposure to waterborne disease, which is arguably a more exogenous measure of the effect of sanitation quality on waterborne diseases.

The estimation results are summarised in [Figure 4](#). Since both the existing literature and our previous results suggest that the WASH practices channel differ by urban and rural contexts, and this correlates with access to improved sanitation, we also split the sample by urban or rural status. As expected, we see that WDP increases the probability of the child having had diarrhea recently for children living in both urban and rural areas, although the effect is both larger and more precisely estimated for urban areas. The direct effect of the sanitation ladder on diarrhea is precisely estimated to be close to zero for all types of toilets. In contrast, we estimate negative effects for all but one of the three interaction terms between WDP and each rung on the sanitation ladder. For the full sample, it is only households on the highest rung of the ladder where we can precisely estimate a mitigation effect of WASH practices from WDP. The coefficient suggests that households who have these sanitation facilities can completely offset the increased risk from WDP. This is consistent with existing evidence on the link between sanitation and diarrhea, which suggests that not any sanitation but mainly *high-quality* facilities are important to stop the faecal-oral channel of the spread of waterborne diseases ([Troeger et al., 2018](#); [Magana-Arachchi and Wanigatunge, 2020](#)).

Furthermore, we see that the main negative effect of WDP and the mitigation with improved sanitation originate from urban areas, which is consistent with observational literature and historical case studies. The large heterogeneity between wards and households with higher and lower types of sanitation also provides an explanation for why only analysing the urban sample yields an imprecisely estimated effect, since there are large heterogeneities. For urban wards, we also see that the step from no facilities to 'unimproved sanitation' also contributes to mitigating the negative

²³To better understand the differences between these categories, [Figure E.1](#) in [Appendix E](#) illustrates some examples.

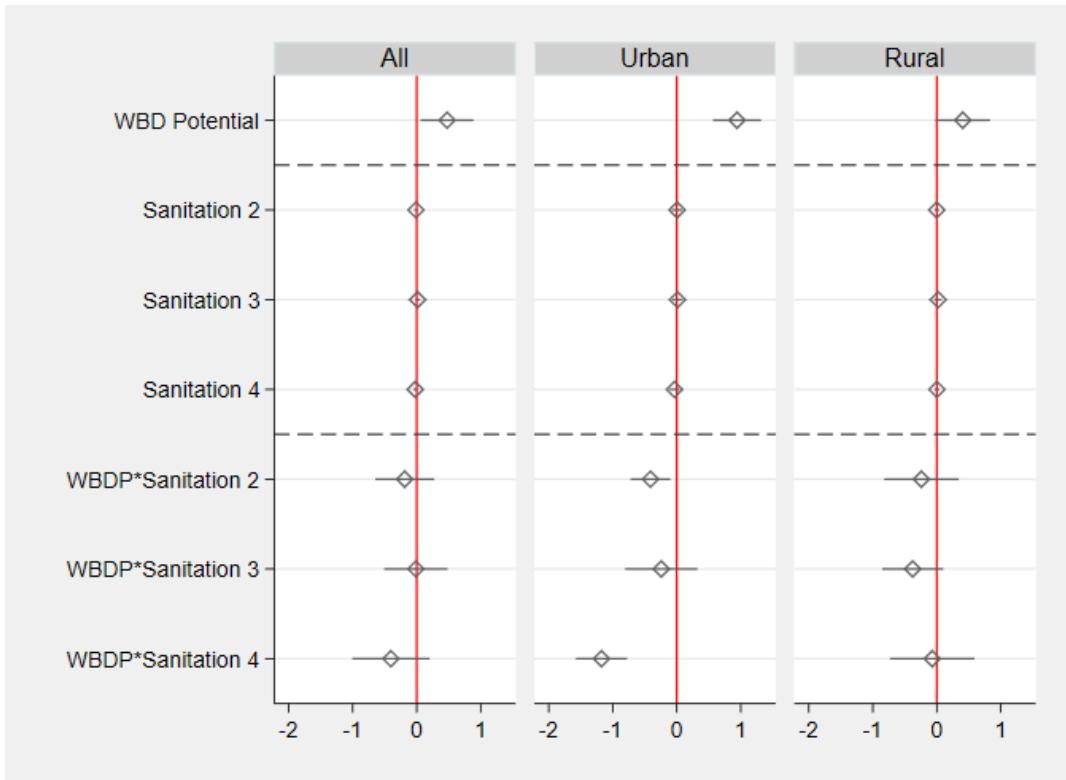


Figure 4
UN Sanitation ladder: Effect of WDP on diarrhea

Note: The three subgraphs represent separate triple-difference estimations of our main treatment, WDP, interacted with sanitation ladder and the effect on the probability the child has had diarrhea the past two weeks. The sanitation ladder is a categorical variable representing four types of sanitation facilities: No facilities, Unimproved sanitation, Shared facilities, Improved sanitation. Here, the baseline is households with the first sanitation ladder category: No facilities. The left subgraph presents the results using the whole sample of DHS children, while the remaining two divide the sample by whether the household lives in an urban or rural area (whether the DHS cluster is classified as urban or rural). See [Figure E.1](#) for more explanation of the sanitation categories.

effect of WDP, but less so than the high-quality facilities. Interestingly, the effect of the third category, shared facilities, is estimated with the most imprecision. This may reflect that shared facilities may be a poor indicator of better sanitation as there is large heterogeneity in how well they meet the sanitation needs of households ([Magana-Arachchi and Wanigatunge, 2020](#)). Indeed, we find that sharing a toilet with other households increases the risk of contracting WBD when WDP is high, which is reported in [Table E.2](#).

Lastly, we interact WDP with a binary indicator for whether the household has access to an improved source of water, as defined by the WHO. We find that access attenuates the effect on diarrhea, but only for urban areas, where we also found that high-quality sanitation matters more. The results are reported in [Table 3](#). This implies that access to safe water is especially important in environments with a high presence of waterborne pathogens, such as densely populated areas afflicted by a stagnant water shock.

Table 3**Effect of WDP on diarrhea incidence, by sanitation quality and access to an improved water source**

Sample:	Dependent: Diarrhea		
	All	Urban	Rural
	(1)	(2)	(3)
WDP	0.597** (0.291)	2.112*** (0.344)	0.370 (0.329)
WDP × Sanitation ladder	-0.254*** (0.091)	-0.376*** (0.141)	-0.175 (0.142)
WDP × Improved water	-0.052 (0.202)	-1.364*** (0.308)	0.179 (0.280)
Observations	12,384	2,930	9,454
Clusters	237	97	230

Note: This table reports the effect of WDP on diarrhea, interacted with the sanitation ladder (from 1 to 5) and an improved water source dummy. Standard errors in parentheses are clustered at the DHS grid-cell level. * p<0.01 ** p<0.05 *** p<0.1. WDP is the average share of the area covered by stagnant water in the eight weeks preceding the date of the survey.

5.1.2 Additional heterogeneity in effects on health

To better understand the effect we estimate, we next conduct a heterogeneity analysis both by key individual and location characteristics.

Urban and rural: We next analyse if there are differences between urban and rural areas since most existing literature find that there are more severe disease effects in urban areas that are densely populated ([Alsan and Goldin, 2019](#); [Troeger et al., 2018](#)), and given the results on the sanitation ladder and urban-rural heterogeneity on diarrhea in [Figure 4](#). [Table E.3](#) reports the results split by whether the surveyed location is urban or rural. We find that the effect is about 50% larger for urban areas²⁴. This suggest that there is a higher probability of contracting waterborne diseases in urban areas following a stagnant water shock, even despite the fact that urban households tend to have better access to improved water and sanitation and are wealthier overall.

Climate: The public health literature has identified an empirical relationship between high temperatures and outbreaks of waterborne diseases ([Levy et al., 2016](#)). We investigate this heterogeneity by the average temperature over the past two weeks, since all health symptoms in the DHS are reported for this period specifically. [Figure H.1](#), top panel, reports the results by temperature level, using a window of +/- 1 degree Celsius. While the differences between most levels are not significant, there is an overall trend towards greater effect sizes for locations with higher average temperatures in the past two weeks, which is consistent both with empirical findings and the biological mechanism of pathogen growth potential.

Gender: Existing literature shows that flooding affects children's outcomes differently for boys

²⁴Running the same specification with an interaction term yields a weakly significant difference between urban and rural locations.

and girls ([Maccini and Yang, 2009](#)), and in India, the mortality rate for diarrheal diseases for girls have been approximately 50% higher than for boys ([Morris et al., 2011](#)). Similarly, as [Table E.5](#) reports, we find a substantially larger effect of WDP on girls than for boys, with the effect size being more than twice as high for girls. However, the difference between the two is not statistically significant.

Age: Heterogeneity results for age are reported in [Figure E.2](#). We find substantial age heterogeneity, where the effect for the age group 0-1 years is close to 0, and the highest effect is found for the age group 1-2 years. This is consistent with a ramping up of diarrhea infection rates when the child transitions out of breastfeeding ([Bhandari et al., 2003](#)). For age groups 2 up to 5 the effects are still positive, than smaller, and there is no sign that the effect attenuates with age, once the child is older than 2 years. This has important implications for our later results with test scores, as it suggests that exposure to stagnant water should have an impact also on children older than five.

5.2 Effects of waterborne disease potential on children's cognition

We next present our main results, the effect of WDP on learning as revealed by standardised test scores. As discussed in [Section 4.3](#), in our main specification WDP is the average share of ward area covered by stagnant water in the eight weeks preceding the survey and test date. Test scores are standardised averages of the child's score in English, Maths and Swahili.

Table 4
Effect of WDP on test scores

	(1)	(2)	(3)	(4)	(5)	(6)
<i>Dependent: Test score (std)</i>						
WDP	-1.624*** (0.323)	-1.476*** (0.251)	-1.079*** (0.379)	-0.776** (0.360)	-0.750** (0.349)	-0.893** (0.359)
Obs.	368,204	368,155	368,202	368,202	368,202	368,153
Clusters	3,842	3,842	3,840	3,840	3,840	3,840
Covs	✓					✓
Ward FE		✓		✓	✓	✓
Wave FE			✓	✓	✓	✓
Month FE				✓	✓	✓

Note: This table reports the effect of WDP on test scores. Standard errors in parentheses are clustered at the ward level. * p<0.01 ** p<0.05 *** p<0.1. WDP is the average share of ward covered in stagnant water in the eight weeks preceding the date of the survey. Covariates include child's gender and age, and mother's age and secondary education, a wealth index, local past eight weeks of ward precipitation. Ward is the treatment unit of observation. Wave is the year of the survey and waves included are 2011, 2013, 2014, 2015 and 2017. Month is the calendar month of treatment.

[Table 4](#) displays the estimates of the effect of WDP on test scores. We successively add more controls and fixed effects to an initial parsimonious specification. Throughout columns 1-6, all estimates are negative and statistically significant. In column 1, we report a pooled regression with

no controls and the estimated coefficient implies that when WDP is 10%, children's test scores are reduced by 0.14 standard deviations. In column 2 we add household-level controls, and then through columns 3-5 add ward, wave and calendar month fixed effects. Both ward and wave fixed effects reduce the coefficient size substantially, although calendar month does not affect the coefficient much, suggesting the estimate is not very sensitive to seasonal variation. Column 6 is our preferred specification: We include household and individual covariates, ward fixed effects, wave fixed effects and month fixed effects. Compared to our specification without covariates, the estimate is only slightly larger, and since the covariates explain a meaningful share of the variation in test scores, this should make it unlikely that selection on unobservables is biasing our estimate upwards ([Oster, 2019](#)). Our results imply that a 10pp increase in WDP reduce children's test scores by 0.074 standard deviations, or alternatively that a one standard deviation increase in WDP reduces test scores by 0.028 standard deviations. Compared to the observational difference in column 1, this effect is reduced by almost half. This suggests there are important time-invariant or ward-invariant unobservable characteristics which influence how WDP affects test scores.

We hypothesise that our results reflect a higher incidence of waterborne diseases among tested children, which affects their capacity to learn. However, we cannot here distinguish between whether the child performs worse because they have been absent from school, or they are ill or just recovering from illness when they take the test and thus performing worse than they would otherwise. In [Section 6](#) we further explore how sensitive our results are to the time dimension of the definition of WDP.

One of our contributions is the ability to capture the effect of waterborne disease potential in the areas where stagnant water settles, as opposed to the from local precipitation. This is important, since rainfall can affect both income and child labor demand through an increase in agricultural productivity, and may thus have an ambiguous net effect on children's cognitive skills through competing channels such as school absence and improved nutritional status ([Shah and Steinberg, 2017](#); [Maccini and Yang, 2009](#); [Rosales-Rueda, 2018](#)). In our main estimation we define and include a variable *Local Rainfall* which is equal to the sum (in m) of rain in the eight weeks preceding the survey date.²⁵ However, local precipitation may drive the estimated effect nonetheless. To address this concern we next run our main specification both with and without WDP and local precipitation to estimate how both variables affect children's test scores.

[Table F.2](#) summarises the result of this exercise. Column 1 shows the correlation between precipitation and WDP. We find no statistically significant relationship between local rainfall in the past eight weeks and our stagnant water measure. This likely indicates that rainfall that contributes to stagnant water stems mostly from upstream catchments, and is consistent with the null finding on the relationship between rainfall and cholera in Tanzania ([Traerup et al., 2011](#)).²⁶ Columns 2

²⁵We use eight weeks to mirror the time window used to construct the treatment variable.

²⁶Interestingly [Traerup et al. \(2011\)](#) finds that almost all cholera outbreaks tend to occur during the rainy season, but conditional on seasonal indicators, rainfall shows no significant relationship with cholera. This is consistent with a setting where local stagnant water will depend on upstream rainfall, and hence show a seasonal pattern, while being mostly independent of local rainfall, controlling for the seasonal effects which correlate with both upstream and local rainfall.

and 3 use WDP and precipitation as separate explanatory variables in regressions on test scores, and column 4 repeats our main specification from column 6 in [Table 4](#). We find no statistically significant effect of local rainfall on test scores, while the effect of WDP remains largely unchanged with and without the inclusion of rainfall as a control variable. We are thus reassured that our results are driven by WDP and not local rainfall, however, we include it as a control variable throughout our analyses.

For completeness, we run the same exercise with diarrhea as an outcome, since this data aggregates stagnant water in a slightly different manner and covers a different time period. [Table F.3](#) summarises the results. Again, we find no significant relationship between local rainfall in the past eight weeks and WDP, and there is also no significant effect of local rainfall on diarrhea, consistent with ([Traerup et al., 2011](#)).

5.2.1 Heterogeneity in effects on cognition

To better understand the effect we estimate, we next conduct a heterogeneity analysis both by key individual characteristics and by type of ward.

Urban and rural: [Table F.4](#) reports the effect of WDP on test scores separately for rural and urban wards. The effect on test scores is more precisely estimated for rural wards, but the estimates are consistently negative and not statistically different from one another. This is consistent with our health findings, which suggest that there is a higher probability of contracting waterborne diseases in urban areas following a stagnant water shock.

Climate: For Uwezo, we investigate heterogeneity by average temperature over the past eight weeks, to include children who got sick and subsequently recovered. [Figure H.1](#), middle panel, reports the results by temperature level, using a window of +/- 1 degree Celsius. While the differences between most levels are not significant, similar to the results for diarrhea there is an overall trend towards greater effect sizes for locations with higher average temperatures in the past two weeks, which is consistent both with empirical findings and the biological mechanism of pathogen growth potential.

Gender: Existing literature shows that flooding affects children's outcomes differently for boys and girls ([Maccini and Yang, 2009](#)). As summarised in ??, we estimate negative effects overall and the coefficients of WDP on test scores for boys and girls are never close to statistically significantly different from one another.

Age: For age, we find similar effects for the age groups 7-8 up to 13-14, whereas the effects for ages 15-16 are somewhat closer to zero, though these differences are not statistically significant. These results are summarised in [Figure F.1](#). Consistent with the age heterogeneity we find in our health results, this suggests that higher age is not necessarily protective against waterborne diseases, at least not until mid-adolescence (age 15-16). In addition, event studies using the estimator from [de Chaisemartin and D'Haultfoeuille \(2022\)](#) suggest that younger children may be particularly vulnerable to persistence of the short-term effect. In the event study, we redefine WDP into a binary

variable indicating whether or not it exceeds 5%,²⁷ and restrict the treated cohorts to those who are only treated once in our sample. [Figure F.7](#) reports event studies for the age groups 6-9, 10-12 and 13-16 years respectively, which are slightly more aggregated to account for the loss of precision from using this estimator.²⁸ For the children aged 6-9, the results suggest that the effect survives at least one, and possibly two or more, waves into the future.²⁹ In contrast, for children in the age groups 10-12, while we identify a relatively large negative effect for the year when they are exposed to stagnant water, it then seems to recover in the subsequent periods. For the oldest children, 13-16 years, we instead find only a small and insignificant effect during the year of exposure, and again little signs of persistence into subsequent periods.

5.3 Effects of waterborne disease potential on school absence

Can the negative effects on learning be explained by an increase in absence from school? To analyse this, we investigate whether a stagnant water shock leads to an increase in absence. For this purpose, we use the school datasets of the Uwezo survey data, which contains a headcount of present children in the classrooms across different grades.³⁰ [Figure G.1](#) shows a histogram of this data. From these data we construct our absence measure, a measure from 0 to 1 which indicates the share of enrolled children that are absent at the survey date. [Figure 5](#) shows the results for absence, by whether the ward is urban or rural and by temperature in the past eight weeks. We find a small but insignificant positive overall effect on absence. However, this masks considerable heterogeneity. The effect on absence is large and significant for urban wards and warm wards, while for rural wards it is only significant for warm wards. This is largely consistent with the mechanism of waterborne diseases, and the results from similar heterogeneity analyses for the effects on children's health and cognition.

To reality-check the large effects we find for absence in urban wards, we can compare the effects of WDP on diarrhea for the oldest and hence most comparable children in DHS for urban locations (aged 4-5 years). Here, we find an effect size of 0.58, which is comparable in magnitude to the effect size for absence for school children at around 1.17, with considerable overlap in the confidence intervals. [Figure H.1](#), third panel, further breaks down the effect on absence by the level of temperature in the past 8 weeks, and shows that the effect size tends to increase with temperature.

²⁷Following our robustness checks in [Section 6](#).

²⁸This age categorization provides roughly equal-sized groups, since there are few children aged 6 and 16 in our sample.

²⁹Since the time between our survey waves is typically between 1 to 2 years, the length of one period in the event studies can roughly be interpreted as 1.5 years.

³⁰School datasets are collected in conjunction with surveyed villages, and like the other surveys are cross-sectional. Across our waves we have 15,428 school observations.

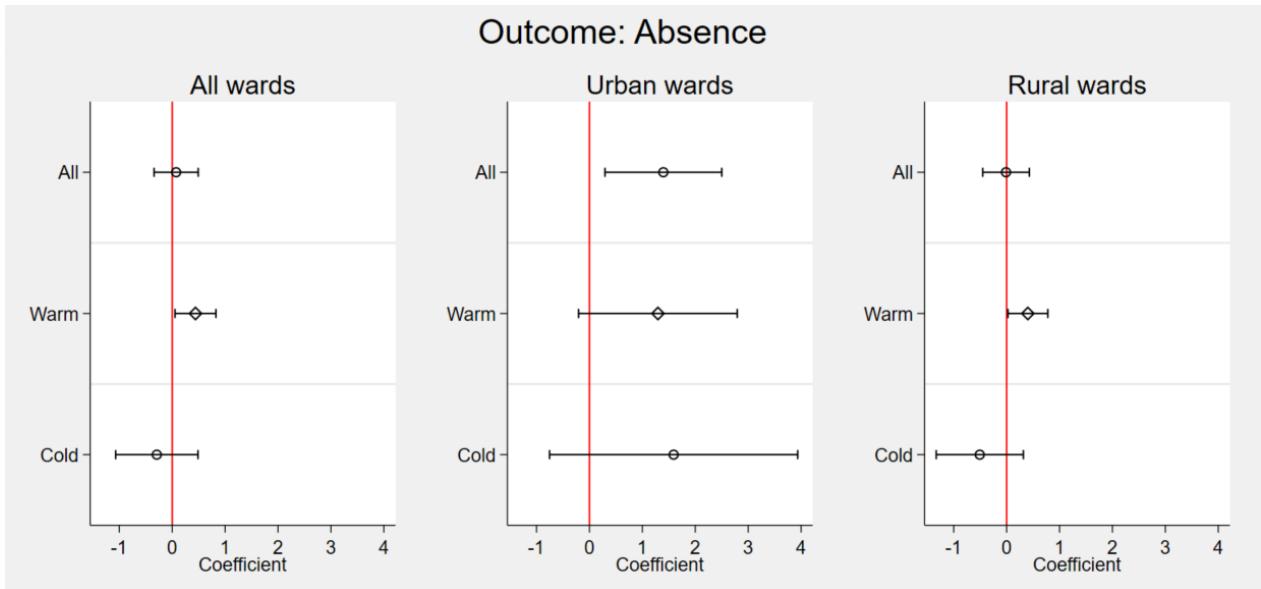


Figure 5
Effects on absence

Note: This figure shows the effect of WDP on absence by ward type and temperature in the last 8 weeks. Coefficients and 95% confidence intervals are displayed.

We next perform a mediation analysis to see to what degree absence can help explain the negative effects of WDP.³¹ Table G.1 reports the results of the mediation analysis. First, from columns 1 and 4, we find that the negative effects on test scores also hold when we collapse the data to the school-level, although they are only significant when we weight the regressions by the number of observed children in the household data³². Including absence as a control variable, as is done in columns 2 and 5, the effect of WDP is largely unchanged. This suggests that most of the effect of WDP on test scores is not due to absence itself. However, adding the interaction of WDP and absence, as is done in columns 3 and 6, removes a significant share of the effect of WDP. This suggests that absence largely moderates the effect of WDP on test scores. In other words, only in schools where WDP leads to an increase in absence among school children do test scores drop, but this is not due to absence itself. The most relevant alternative explanation is cognitive impairment, either from falling sick due to waterborne diseases, or due to a direct effect of stagnant water on cognition itself. Since treatment is confined to the past 8 weeks, it seems reasonable that absence would not be the main driver of the reduction in test scores. However, absence itself may well lead to impacts in the longer-run, as found by Cattan et al. (2023), and we primarily interpret the effects on absence as an additional learning outcome.

³¹ Technically, absence is also an outcome of exposure, and hence a “bad control”. However, it could still be valuable to see to what degree the effect of stagnant water exposure runs through absence.

³² Note that due to missing data and schools excluded due to measurement error we will not have the same sample as in the main analysis. However, the results are still largely comparable to our main results.

6 Robustness checks

Non-linearities in treatment effects: In our main specification we estimate the effect of a continuous share of WDP (which can take any value between zero and one) and estimate this on test scores. This assumes a linear relationship between WDP and test scores. In this section, we explore how strong an assumption that imposes. We start by first redefining WDP to be a binary treatment by creating a dummy which is equal to one for the wards in waves where WDP is greater than 5% (see [Table 1](#)). Including this in our main specification instead of our continuous variable, we again estimate the effect on test scores. As summarised in [Table F.8](#) in [Appendix F. II](#) we find that treatment leads to -0.10 standard deviations lower thresholds.³³ This is larger but comparable to our main result of -0.7 standard deviations, consistent with the fact that 5% is a relatively severe or rare shock.

Next, we estimate the effect on our main WDP measure but also include a squared term. As displayed in [Table F.9](#), the linear term of the effect of WDP on test scores increases from .74 standard deviations in our main specification to now being -1.33, and still statistically significant. The coefficient for the squared term, although it is statistically insignificant, is large and positive (1.52), which implies decreasing marginal effects of WDP on test scores. While this may seem counter-intuitive, since more stagnant water would lead to a greater probability of a disease outbreak which could potentially create positive feedback loops, attenuation happens mostly for treatment values near 1, indicating a fully water-covered ward, which never occurs in practice. One would also expect that, as the share of stagnant water grows, contaminated surface water becomes more diluted, thereby decreasing the probability of an outbreak. Several studies have shown that the probability of outbreaks of diarrhea due to heavy rainfall is typically higher following a dry period than a wet period, which would generate a smaller but less contaminated amount of surface water, suggesting that rainfall dynamics affect not only the quantity but also the quality of surface water ([Levy et al., 2016](#)).

To further investigate how the intensity of our treatment affects children's test scores, we create additional dummies of WDP and include these dummies in the same estimation (WDP 1-5%, 5-10%, 10-15%, 15-20% and above 20%). These results are presented in [Figure F.3](#). We find no statistically significant effect of WDP for values less than 5%. Reassuringly, there is a clear negative trend with an increase in the treatment magnitude, and the coefficients are negative for all other binned values of WDP, although only statistically significant for WDP between 10-20%. Imprecision increases when $\text{WDP} \geq 15\%$, consistent with these being rare events (see e.g. [Figure D.1](#))

Randomisation inference: We conduct randomisation inference to evaluate to what extent our statistically significant findings could be based on spurious results ([Hsiang et al., 2014; Young, 2019](#)). This is a methodology with which to conduct a data-driven inference, and is a powerful exercise to evaluate sensitivity to spatial correlation, non-random exposure to exogenous shocks ([Borusyak and Hull, 2022](#)), and the systematic assignment of placebo treatments in non-treated periods, commonly used in difference-in-differences specifications. We run our main specification

³³All results in this section can be found in [Appendix F. II](#).

from [Equation \(3\)](#), but randomly permute the treatment within wards, such that for every permutation, WDP values from a randomly selected year is assigned. The fact that for each iteration, all treatment values are drawn from the same year will take into account any spatial correlation between wards, and provide a data-driven way to estimate p-values that are adjusted for spatial correlation^{[34](#)}. The expectation is that we should find no effect on test scores with reshuffled treatment unless our results are driven by spurious regressions, i.e. the distribution of estimated coefficients should be centred around zero. We run these placebo regressions 500 times and find no evidence supporting that our results are due to spurious regressions (see [Figure F.4 Appendix F. II](#)).

Heterogeneous and dynamic treatment effects: As we rely on variation in treatment timing, such that some locations are treated in different years than others, it is possible that our estimates are biased due to heterogeneous treatment effects ([Goodman-Bacon, 2021](#)). In our setting, the likeliest source of such heterogeneity is that children exposed to higher waterborne disease potential are sicker and more vulnerable, putting them on a permanently more negative trajectory compared to non-treated children. If this trajectory persists and affects children across waves (i.e. years) a comparison against such wards would attenuate our effect to zero. Notably, since the vast majority of children in our sample are never treated, we do not expect such effects to dominate.

However to assess this issue formally, we implement the estimator developed by [de Chaisemartin and D'Haultfoeuille \(2018\)](#) (denoted DCDH). From the potential other estimators we implement this estimator since our setting is non-staggered, in that wards can go from treated to untreated and back ([de Chaisemartin and D'Haultfoeuille, 2022](#)). Moreover, since the correct estimators for continuous treatments where treatment is non-staggered are not well-established as of yet, we redefine our treatment, WDP, to a binary variable. This implies a ward becomes treated if the share of the stagnant water of the ward area exceeds 5%. [Figure F.5](#) summarises these results. The estimate of the effect of WDP on test scores with two-way fixed effects (TWFE) is -0.095 standard deviations while the estimate with the DHDC estimator is -0.144, suggesting that heterogeneous treatment effects may play a role, although this estimate is also less precise, and the differences between the two estimates is insignificant. We also extend the estimator to account for dynamic effects (as according to [de Chaisemartin and D'Haultfoeuille, 2022](#)) and although more imprecisely estimated our treatment effect is not significantly different from the previously tried estimators. We thus conclude dynamic treatment effects have at most a limited impact on our estimates using TWFE.

Pretrends and the parallel trends assumption: Typically, an event study, such as reported in [Figure F.6](#), can be used to assess the similarity of pretrends for treated and non-treated groups. However, due to the low power of this test with the estimator we use, we do a second test of pretrends where we make use of more of the sample, although this necessitates testing pretrends not immediately prior to treatment in all cases. To do this, we again use the binary indicator of $WDP \geq 5\%$, and only include wards that were never treated or wards that were treated only in the

³⁴One advantage of randomisation inference over Conley standard errors adjusted for spatial correlation is that the former does not rely on assumptions about the correct distance cutoff values and functional forms of the spatial correlation structure.

period 2014-2017 (“later-treated”). This allows us to test whether later-treated and never-treated wards followed different trends in the pre-treatment period between 2011 and 2013, the first two waves in our data. [Table F.10](#) reports the results of this exercise. In the first column, we first verify that we still identify a significant treatment effect for the later-treated at -0.284. The second column reports the results of the pretrends test. We find a coefficient near zero with much greater precision than in the event study specification. The lower 95% confidence interval bound of this estimate is -0.09, which is relatively small compared to the effect size, and it is thus unlikely that differential trajectories in the pre-treatment period can explain the observed treatment effects.

However, we expand this analysis to include our full sample, and in [Table F.11](#) report our main results together with a set of additional fixed effects to flexibly control for shocks and individual trends that may have affected treated locations differently. First, we add district-level \times wave FE to absorb any current shocks that may occur with stagnant water shocks, such as rainfall or water available for irrigation that can affect the local economy. The results remain unchanged. We then analyze whether areas more prone to stagnant water shocks may have seen differential trajectory. We define *High WDP* as an indicator of whether the ward at any point observes, which captures locations that have a high systematic risk of stagnant water exposure. Including high WDP \times wave FE would absorb any shocks that affect more and less stagnant water-prone areas differentially. Again, we find that the treatment effect is largely unchanged. Finally, we allow for each ward to follow different trends over time. If anything this only strengthens the treatment effect, but the difference to previously estimated treatment effects is insignificant.

Sensitivity to choice of time window: In our main specification we define WDP to be the average share of stagnant water which covers the ward area over the past eight weeks. In [Figure F.8](#) we vary the number of weeks included in this average and re-estimate our main result, the effect of WDP on test scores. We find that the coefficient of the treatment is remarkably stable across weeks included in the treatment, and statistically significant throughout. To better understand which weeks drive this result, we next compute a treatment variable which measures the stagnant water share by week, discretely. That is, in one estimation we only include the stagnant water share in the third week since the date of the survey, and in the next, we only include the stagnant water share in the fourth week since the date of the survey. The estimation results are summarised in [Figure F.9](#). Here we see that there is a large initial effect which is measured with the most precision in week one, but the statistically significant effect remains up to eight weeks. This means there is likely a high serial correlation between these definitions of our treatment since the same pool of water can linger across weeks. Together, the results suggest that our estimated effect on test scores is the strongest for the weeks closer to the survey date but persists for the period observed. Importantly, we also see that the effect is measured also in the week immediately following the date of the survey, which is consistent with studies on outbreaks and the microbiological literature suggesting waterborne pathogens can contaminate and spread quickly.

Given that the results show high autocorrelation of WDP over time, one concern is that our model captures a phenomenon in the future, which we then estimate effects for due to the correlation with past events. Such would be the case if past WDP captures that children expect more

favourable environmental conditions or events in the future in a way which affects test scores. If this is the case, *future* WDP (in terms of the date children are surveyed) should better predict the change in test scores than past events. However, due to the high correlation (95%) between weeks, models including weekly WDP in the same estimation are likely to induce problems associated with multicollinearity. [Figure F.10](#) displays two attempts to disentangle the potential role of future WDP while reducing the influence of multicollinearity: We measure the change in WDP over time or coarsely bin WDP across time. These results are measured with less precision than our main results, but past WDP better explains the reduction in test scores than future WDP (see [Appendix F. II](#) for more detailed explanation.)

7 Awareness and alternative explanations

7.1 Awareness

We next analyse to what extent the effects of stagnant water on children's health and learning be explained by a lack of awareness or preparedness. We employ data from the DHS on reported community health problems, potential behavioural responses such as breastfeeding and drinking of plain water, and sanitation quality as outcomes. We run regressions with and without gridcell fixed effects. The regressions without gridcell fixed effects can be interpreted as cross-sectional regressions, where we exploit the geographic variation across Tanzania, instead of the within-location variation over time. This allows us to see to what degree communities in high- versus low-risk areas are aware of the risk factor of stagnant water, while the regressions with gridcell fixed effects allow us to see how awareness and behaviour responds to a stagnant water shock over the short term. Of course, we would not expect sanitation quality to change in the short term, but still include this in the analysis for completeness.

Table 5 reports our results. For our results without gridcell fixed effects, we find that exposure to WDP has a *negative* effect on stated importance of diarrheal diseases. This negative effect is most likely explained by the fact that malaria is considered the most important problem in these areas, as discussed in [Section 7.2](#), and with grid cell fixed effects there is no significant effect. We find that mothers breastfeed *less* in areas more prone to stagnant water, but again that there is no effect once including gridcell fixed effects. This indicates that there is overall less breastfeeding in more vulnerable areas, and that breastfeeding does not increase during periods of increased stagnant water, despite its protective effects. In a similar vein, giving plain water is not less common in areas more prone to stagnant water, and in fact increases in stagnant water only leads to higher consumption of plain water, despite the. Lastly, we do not find that sanitation quality is higher in areas more prone to stagnant water despite its mitigating effects. If anything, quality seems lower, though this difference is not significant. As expected, we also find no change in sanitation quality when including gridcell FE, as it is unrealistic that sanitation investments would be affected by a shock lasting only eight weeks.

Our interpretation of these results is that there is a significant gap between the risks and ways

to mitigate waterborne diseases, and what households actually do. This suggests that effective information campaigns on the benefits of breastfeeding and importance of water treatment may induce households to change their behaviour and lower the risk of waterborne diseases. In the long run, given that we do not find evidence of past sanitation investments having targeted at-risk areas, our findings can be informative on how to improve the targeting of sanitation investment given the robustness of our results from WDP.

Table 5
Effects of WDP on waterborne diseases awareness and preparedness

	(1) Diarrhoea import- tance	(2) Breastfeeds	(3) Gave plain water	(4) Sanitation ladder
Panel A: Without gridcell FE				
WDP	-0.162** (0.072)	-0.254** (0.104)	-0.015 (0.158)	-0.194 (0.374)
Observations	10,230	21,432	14,054	16,471
Clusters	202	252	251	252
Panel B: With gridcell FE				
WDP	-0.020 (0.066)	0.021 (0.097)	0.261** (0.120)	0.013 (0.446)
Observations	10,230	21,432	14,053	16,469
Clusters	202	252	250	250

Note: Standard errors in parentheses clustered on DHS gridcell level.* p<0.01 ** p<0.05 *** p<0.1. WDP (%) is the average percent share of area covered in stagnant water the eight weeks prior to date of survey. 'Diarrhea importance' is an indicator for whether diarrhea is stated as the most important health problem in the community. 'Breastfeeds' is an indicator for whether the mother is currently breastfeeding. 'Gave plain water' is an indicator for whether a child received plain (untreated) water in the last 24 hours. 'Sanitation ladder' is a sanitation quality index from 0-5.

7.2 Alternative explanations

Malaria: Stagnant water may act as a breeding ground for mosquitos carrying the malaria-parasite as well as other vector-borne diseases, such as the West Nile virus and dengue fever. However, as [Table 2](#) reports, we do not find that exposure to stagnant water shocks causes fever, the most common sign of malaria infection. Notably, and somewhat reassuringly given the established link between stagnant water and mosquitoes, in *cross-sectional* regressions we do find a positive association between waterborne disease potential and fever. That is, we use the same specification as for our main results in [Table 2](#) but without the gridcell fixed effects, which means our treatment variable only captures variation across regions. [Table E.6](#) reports the results of this exercise. Indeed, we see that in the *cross-sectional* regression, stagnant water exposure strongly predicts fever prevalence, the main symptom of malaria, and this effect is similar in magnitude to the effect on diarrhea. Similar to the specification accounting for gridcell fixed effects, there are no significant effects on other health outcomes unrelated to either malaria or waterborne diseases.

Moreover, we also investigate both difference-in-difference estimates and cross-sectional estimates on indicators related to awareness and preventative behaviour around malaria. As [Table E.7](#) reports, we find that areas with more stagnant water exposure households also report that malaria is an important problem, are more likely to use bednets and to be aware of ways to avoid malaria. However, these effects disappear once gridcell fixed effects are included.³⁵ This suggests that areas more prone to stagnant water do indeed have a higher baseline risk of malaria, and that households are also informed of this risk, but, importantly for our interpretation, that malaria does not respond to short-term stagnant water shocks the same way that waterborne diseases do³⁶. Reassuringly, estimates of the cross-sectional effect on diseases that should not be associated with stagnant water in general such as anemia and cough remain small and insignificant. Finally, to further disentangle effects of malaria from waterborne diseases, we follow [Kuecken et al. \(2021\)](#) and use prevalence data from the Malaria Atlas Project for 2010 ([Sinka et al., 2012](#)), the last year before our education data starts. [Table F.7](#) reports the results from our baseline specification run separately for high and low malaria prevalence areas, split by median malaria prevalence. Although the treatment effect is more precisely estimated for the high malaria prevalence areas, we do not find any statistically or economically significant differences between the two coefficients, which suggests that the effect of stagnant water on children's test scores is unlikely to be explained by malaria.

Floods: Stagnant water shocks are certainly correlated with floods that may damage infrastructure and cause other disruptions that make children unable to get to school. We argue that these disruption effects are unlikely to bias our estimates for several reasons. First, our exposure measure only takes into account *stagnant* water. This means that any flowing water is excluded from the treatment. Moreover, areas with sloping terrain, which induces high flow velocities and enables erosion of roads and other critical infrastructure typically see little stagnant water and will be orthogonal to our treatment measure. Second, in our heterogeneity analysis we see that the effect is driven by wards with a higher temperature, indicating that the channel is through pathogen growth rather than damaged infrastructure. Third, we find significant effects at small doses of the treatment (around 5-10 %), while the effects seem to saturate at levels above 15-20%³⁷, suggesting that our results are not driven by large and disruptive floods.

³⁵ See the conclusion for a discussion on policy implications given the negative findings on diarrheal awareness reported in this table.

³⁶ By short-term, we mean periods lasting from two to eight weeks. That malaria does not necessarily respond to short-term shocks is likely due to two specific reasons. First, for mosquitoes to effectively spread malaria, they must first reproduce and their larvae undergo the full development phase before subsequently transmitting the disease to humans, which is estimated to take in the order of 8-12 weeks from the first rainfall to the first visible symptoms ([Teklehaimanot et al., 2004](#)). In contrast, waterborne diseases typically spread within 1-5 weeks, which is within the windows we use for the treatment definition. Secondly, there is typically a non-linear response between rainfall and malaria. Moderate amounts of rainfall tends to increase malaria risk, but too much rainfall may “wash out” mosquito larvae; empirically an inverted U-shaped curve between malaria risk and rainfall is usually found ([Paaijmans et al., 2007; Lowe et al., 2013](#)). Since the treatment effects are driven primarily by areas with a high amount of stagnant water, it is likely that rainfall in these areas was intense enough to crowd out any positive effects from stagnant water itself. This is also supported by the fact that there was no visible increase in bednet use or increase in malaria being reported as an important health problem in the specification with gridcell fixed effects.

³⁷ Gradually excluding outliers with stagnant water values >50% down to >20% only yield larger and more significant treatment effects. Results available on request.

Agricultural productivity and child labour: One of the reasons why we control for local rainfall is to disentangle the results from the confounding effects of agricultural labour demand and income. It may however be the case that the accumulation of stagnant water is a useful resource for e.g. crop irrigation. Using crop calendar data for Tanzania, we do not find that the results change significantly for months where water availability is critical for plant growth.³⁸ Moreover, we find no significant differences between farming and non-farming households. If anything the treatment effects are larger for urban than rural households, as [Table F.4](#) reports, suggesting that our results are not confounded by effects of local weather on child labour or income.

Anticipation and migration: We believe it is unlikely that households are able to anticipate stagnant water shocks, since there was no flood warning system in place during our period of study. Moreover, the short-term nature of the shock makes it unlikely that migration is biasing our estimates for health and learning. However, it is possible that long-term results on learning, as reported in the event study in [Figure F.6](#), are underestimated due to overall migration inducing classical measurement error, while selective migration may bias our findings in either direction, depending on whether households following a stagnant water shock positively or negatively select into migration.³⁹

8 Impacts of climate change

8.1 Framework for climate change multiplier

How will climate change affect the frequency and severity of waterborne diseases? From the literature, we know that both temperature and rainfall are important determinants of waterborne diseases ([Levy et al., 2016](#)), and in this paper we find that rainfall acts through the formation of local stagnant water. High temperatures speed up the growth of waterborne pathogens, while intense rainfall events causing more stagnant water shocks are expected to become more common with climate change. Climate change is thus likely to affect both the *severity* and *frequency* of waterborne disease outbreaks. There is, however, still a lack of understanding on how the combined effects of these two phenomenon will affect waterborne diseases ([Levy et al., 2018](#)). To estimate the combined multiplier effects of climate change of the way waterborne diseases may affect children's human capital, we use the latest climate projections for East Africa from [Ayugi et al. \(2021\)](#) in combination with our estimates on how waterborne disease potential and climate affects children's test scores. A more detailed explanation of the underlying assumptions and results of related to the climate change analysis is provided in [Appendix H](#).

Assuming that climate change will affect future temperatures, we can use our estimates for temperature heterogeneity reported in [Figure H.1](#). For diarrhea, which we use as our main measure of waterborne diseases, we find that one degree of warmer weather increases the effect size of WDP

³⁸Results are available upon request.

³⁹However, a preliminary analysis using data from the Tanzania population census of 2012 shows no effect of stagnant water exposure on migration. Results are available upon request.

by 0.11 units, compared to a baseline effect of 0.3. To get a measure of how the frequency of shocks may change with climate change, we use estimates from [Li et al. \(2021\)](#), who find that for every degree of warming, the frequency of intense rainfall events will increase by about 25%.^{[40](#)}

8.2 Results

We can thus provide a first estimate of the impact of climate change by multiplying the frequency and severity (magnitude) increases for each degree C of projected warming. [Figure 6](#) shows how much the combined effect size and frequency increases for each degree C of projected warming. The range of considered temperatures is taken from two established climate change projections: SSP 4.5 and SSP 8.5 (Shared Socioeconomic Pathways).^{[41](#)} SSP 4.5 indicates that the projected warming will be a “Middle of the Road” climate change scenario, whereas the SSP 8.5 scenario is likely an upper-bound, with dramatic temperature increases due to runaway increases in greenhouse gas emissions. This can be interpreted as the scenario where no improvements in climate change policy or emissions have been realised. As can be seen, there is an exponential increase in the multiplier with increased warming, and with as little as 2 degrees C of global warming, the combined effect size and frequency increase by a factor of 3. While these estimates are large, they are comparable to recent epidemiological research, which has found that El Niño events, which are associated with both higher temperatures and more rainfall across East Africa, leads to a three-fold increase in cholera ([Moore et al., 2017](#)). These increases stem approximately in equal parts from direct effects of temperature and increased rainfall variability respectively, as [Figure H.3](#) shows. For more details on our climate change impact analysis, see [Appendix H](#).

⁴⁰This refers to rainfall events with return periods of 10-50 years, lasting 1-5 days, which roughly correspond to the return period of the large stagnant water shocks that drive our treatment effects.

⁴¹See [International Panel of Climate Change \(2022\)](#) and [Appendix H](#) for more detailed descriptions. Note that SSP scenarios also take into account projections of technological advancement, population growth, energy policies and more.

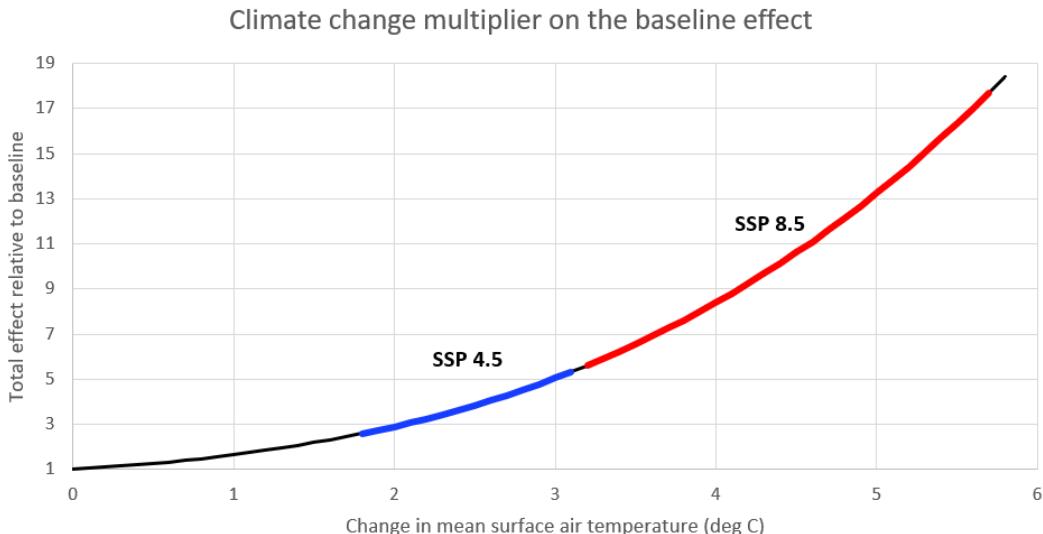


Figure 6

Impact of climate change on the combined effect size and frequency of WDP on test scores

Note: This figure shows the combined increase in our baseline effect on test scores from the effect of an increase in temperature multiplied by the concomitant increase in frequency of intense rainfall events that drive stagnant water shocks. SSP 4.5 and SSP 8.5 constitute two Shared Socioeconomic Pathways (SSP) scenarios commonly used in climate change impact analysis.

9 Conclusion and policy implications

In this paper we have estimated the effect of exposure to stagnant water, a geographically determined risk factor of waterborne diseases, on children's health and learning. We do this by constructing a hydrological model to simulate the local share of stagnant water, the WDP, in each location in the eight weeks prior to the date of collection of the health and learning data. We hypothesise that stagnant water pools encourage waterborne pathogens to proliferate: Contact with these pathogens causes a local increase in waterborne diseases which affects children's health and capacity to learn.

Applying a DiD specification to extract the causal effect of waterborne diseases through WDP we first validate our interpretation of WDP as affecting the main symptom of waterborne diseases – diarrhea – while having no effects on other disease symptoms or long-run outcomes. In our main specification, we find that a 10pp increase in stagnant water lowers test scores by 8.9% of a standard deviation. To compare against another type of environmental shock, [Hyland and Russ \(2019\)](#) estimate that early childhood droughts reduce the years of educational attainment by 0.44 years, or 0.1 standard deviations of years of schooling for children in Sub-Saharan Africa. In contrast to their long-run results, our main estimate measures the effect of a contemporaneous shock directly on student performance and learning: students' test scores. Our estimated short-term effect is smaller than they estimate on years of schooling but in line with their findings that environmental factors have significant effects on children's education. In addition, our event-study estimates suggest that these effects could persist in the long run, at least for younger children.⁴²

⁴²See [Figure F.6](#) and [Figure F.7](#).

We also analyse how behavioral practices interact with WDP and its effects. We find that the results on test scores are stronger in urban than rural wards, and that WASH quality, as measured by access to improved water and sanitation, has a larger mitigating effect against diarrhea in urban than rural locations. This is consistent with historical events and documented disease outbreaks, where sanitation has been found to be a crucial factor in combatting waterborne diseases, especially in locations of high population density.

We generate two key insights. First, that there is substantial heterogeneity in the effects of WDP, and that the effect on both diarrhea, test scores, and absence are driven primarily by warm and densely populated areas. Second, that it is in particular high-quality facilities that offset the increased risk of waterborne diseases, consistent with null results from RCT's that have adopted a piecemeal approach. Thus, WASH programs in Tanzania and other developing countries that battle reoccurring outbreaks of waterborne disease will likely have to invest in higher-quality (and more expensive) toilet- and sanitation facilities to reduce the incidence of waterborne diseases to that of the developed world. One way to make this more cost-efficient is to target areas where WDP is likelier to be higher or more volatile.

While large-scale investment in WASH infrastructure is likely necessary, albeit expensive, in order to improve the local disease environment, an intermediate and potentially cost-efficient step in reducing the worst consequences of an outbreak could be improved targeting of medical treatment to areas at higher risk of outbreaks. To reiterate, we find that one standard deviation increase in WDP reduces test scores by 0.03 standard deviations. This effect size is equivalent to the effect on test scores estimated by [Mbiti et al. \(2019\)](#) who over a two-year period incentivise teachers in Tanzania with 5,000 TZS (3 USD) per student's passing grade (although their estimate is not statistically significant). The cost of this program can be compared to diarrhea rehydration treatment which costs only 0.56 USD. Despite this high cost-effectiveness, only about one in six children receive the treatment in rural Tanzania. We thus leave it to future research to explore demand and supply-side policies to increase children's access to treatment.

We estimate that the effects of climate change on future disease outbreaks will be dramatic: as little as 2 degrees of global warming may lead to a threefold increase in the total effect of stagnant water shocks, and this increases exponentially with temperature increases. Yet, as [Table E.7](#) reports, we find little evidence that stagnant water shocks, even if they have occurred in the recent past, lead to an increase in diarrhea awareness, either in the short or long run. Rather, in these areas households seem more adapted to malaria risk, which is sensible since stagnant water is an established mechanism both for water- and vectorborne diseases. Perhaps it is the adaptation of households to malaria, but not waterborne disease risk, that makes diarrheal diseases such a dominating health mechanism in our analysis. This also motivates policy that informs households of these risks and mitigating strategies, such as breastfeeding, which we find is protective. If households are less aware, it is likely that decision-makers also underestimate this risk factor, which, together with our empirical approach, can motivate better targeting of water and sanitation, and health clinics that can provide oral rehydration salts.

The burden of waterborne disease on both children's health and learning which we document

in this paper implies that policies to combat waterborne diseases should also take climatic risk into account. Our results are especially concerning as access to water will be even more strained with the onset of more severe climate change, as water shortage may increase the propensity and need of communities to draw water from unsafe sources. The important interaction between waterborne diseases and sanitation provides hopeful evidence for how the costs of waterborne diseases can be combated with sanitation policies if targeted to vulnerable areas.

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A Geographic data and modelling stagnant water

A. I Data sources

Table A.1
Data sources used in the WDP algorithm

Input category	Dataset	Resolution and accuracy of input data
<i>Topography</i>	Digital Elevation Model from the Shuttle Radar Topography Mission	30 m horizontal resolution, aggregated into 90 m. 6 m vertical accuracy.
<i>Rainfall</i>	ERA5-reanalysis data for Precipitation	27 km horizontal resolution. 1 hour time resolution.
<i>Evaporation</i>	ERA5-reanalysis data for Potential Evapotranspiration	27 km horizontal resolution. 1 hour time resolution.
<i>Soil data</i>	ISRIC 2.0 global soil database	1 km horizontal resolution.

We use four different categories of data for our algorithm to compute WDP, as summarised in [Figure 1](#) and [Table A.1](#), which are: Topographical data, rainfall data, evaporation data and soil infiltration data. See [Farr et al. \(2007\)](#); [Hersbach et al. \(2018\)](#); [Hersbach et al. \(2018\)](#); [Poggio et al. \(2021\)](#).

We use topographical data from the Shuttle Radar Topography Mission ([Farr et al., 2007](#)). This is one of the most commonly used high-resolution global datasets on topography in scientific research, and is assembled by satellite data gathered in February 2000. Since we are not aiming to resolve detailed features in urban areas but rather large-scale runoff processes, we believe that this data is representative of the topography in Tanzania for the time period of our survey data. The resolution for each grid cell is approximately 30 m, with a vertical accuracy of around 6 m. Since we run our algorithm for the whole country, we aggregate this data to a 90 m resolution in order to make the computation feasible. This still leaves us with more than 120 million grid cells for the area of Tanzania and likely provides a detailed enough horizontal resolution for the large-scale analysis we run. Recent evaluations of large-scale 2D hydrodynamic simulations for several European rivers show that resulting flooded area and water level are insensitive to variations in spatial resolution once it is finer than 100 m ([Dazzi et al., 2021](#); [Falter et al., 2013, 2016](#)). The topography is used to determine the flow direction of each cell during the simulation, which may change dynamically as a function of the water depth over the cell, and to determine the water depth across cells at each point in time.

For rainfall input, we use data from ERA5, which provides a global gridded dataset with hourly estimates of a multitude of atmospheric variables, including precipitation ([Hersbach et al., 2018](#)). We use the reanalysis data, which is based on an ensemble of forecast models which take both satellite data and local weather station data into account and updates predicted atmospheric variables at a 1-hour resolution. The spatial resolution is 0.25 decimal degrees, which approximately

translates to 27 km at the equator. This gives us close to 1400 data points for Tanzania, which is vastly greater than the number of rainfall stations in the country. Gridded rainfall data derived from satellite observations is especially useful in developing countries, where there is often a relative scarcity and lower quality of data from rainfall gauges. Moreover, rainfall stations may vary systematically with local development, which means that measurement error may become systematically correlated with the local level of development. The advantage of using satellite-adjusted data is that measurement error is arguably orthogonal to local development. With regards to hydrological simulations, rainfall is especially important since it tends to be one of the main sources of uncertainty. Fortunately, a recent study investigating 22 global gridded rainfall datasets systematically found that ERA5 reanalysis data provided one of the best calibration scores and lowest inaccuracies when used operationally in hydrological models ([Beck et al., 2017](#)). The rainfall data provides all the input of water in the model, and thus drives the resulting surface runoff, depending on local infiltration rates, topography and evaporation.

For evaporation we also rely on the ERA5 reanalysis data, for the same reasons given above ([Hersbach et al., 2018](#)). This also lends consistency to the rainfall data, since these two datasets are produced jointly and dependent on each other. Specifically, we use the *potential* evapotranspiration rate which is applied to cells with a water depth greater than zero.

Lastly, for soil infiltration, we use soil data from the ISRIC 2.0 global soil database ([Poggio et al., 2021](#)). This is a state-of-the-art high resolution soil dataset which provides a resolution as fine as 1 km and provides a distribution of the content of clay, silt and sand at different soil depths. We depth-integrate this data and use the distribution of soils in each cell to classify each cell as a soil type according to the USDA classification system. From this classification we can then derive soil parameters such as saturated infiltration capacity and soil porosity. The current version of our algorithm uses a simplified infiltration measure by applying only the saturated conductivity of the soil to account for losses due to soil infiltration. Due to the high resolution of the topography we do not apply any slope-adjusted infiltration rates as some low-resolution hydrological models do. Instead a greater slope will translate into a faster runoff process, which will reduce the resulting infiltration. Future iterations of this algorithm could potentially be improved by applying a full soil infiltration mode commonly used in state-of-the-art hydrological models, such as the Green-Ampt method, and also by simulating the groundwater storage layer as a separate entity able to refeed the infiltrated water as groundwater seepage into rivers and streams, which as of current is not handled by the algorithm.

A. II Algorithm for WBD Potential

The purpose of the algorithm is to model the time evolution of stagnant water surfaces over time, which we wish to aggregate to a weekly-level treatment measure, using a combination of hydrological and hydraulic calculations. For this purpose, we run the model with 5-minute temporal resolution. Before the model is run, input data are processed to cover the same extent and transformed to the same coordinate system. Below is a schematic explaining how the algorithm, which

is implemented in Python, works.

1. Initialise and run the model starting > 3 months before the first interview date until the last interview date.
 - (a) Initialise a new day d
 - (b) For every 5-minute timestep $t \in \{1, \dots, 288\}$ in each day:
 - i. Accumulate the last 5 minutes of precipitation on all grid cells
 - ii. For each cell i, j identify the immediately neighboring cell with the lowest current water level, where water level is the sum of the current water depth and elevation of the cell.
 - iii. If this cell has a lower water level than cell i, j , then transfer the 5-minute accumulated volumetric flow V from cell i, j to the receiving cell, otherwise do nothing. The volumetric flow rate $V = f(\Delta h)$ is an increasing function in the difference in energy levels (Δh) between the emitting and receiving cells, based on the Manning formula for open channel flow.
 - iv. Finally, remove the 5-minute accumulated volumetric infiltration rate from cell i, j : $\min(d_{i,j}, f(K))$ where $d_{i,j}$ is the water depth of cell i, j and K_s is a soil infiltration parameter. The current version, $f(K) = K_s$ where K_s is the saturated hydraulic conductivity of soil s in cell i, j .
 - (c) At the last time step of each day, $t = 288$, subtract the volumetric actual evaporation rate E from all cells where $E = \min(d_{i,j,t}, E_d)$ where $E_{i,j,d}$ is the potential evaporation rate in cell i, j for day d .
2. Export water depth for all cells to daily georeferenced arrays, which are then aggregated into weekly-level treatment measures at the ward-level using ArcGIS and the ArcPy package for Python.

A. III Validation with satellite data

Optimally, one would want to have a measure of the actual surface water. One such way could be through satellite imagery. There now exists a global database of surface water down to a 30 m resolution, released by the Joint Research Centre of the European Commission and spanning the time period 1984-2021 ([Pekel et al., 2016](#)). One problem, however, is that data is only available at the monthly level, which is too aggregated for the short-run effects we analyze, which is at the weekly level. Moreover, with the temporally disaggregated data (at the month level) missing data due to e.g. incomplete satellite coverage and cloud cover is common, at least for Tanzania. Lastly, there is also the potential issue that observed surface water is endogenous to human behaviour in ways that correlate with our outcomes of interest. It could, for instance, be that areas where water is cleared away faster have better access to functioning infrastructure and are more developed.

By instead simulating surface water using static topography and using time variation in climate variables, as we do in our baseline algorithm, we arguably get an exogenous source of variation in surface water.

One way to get around the potential endogeneity issue and also deal with missing data is to use the long-term occurrence data, which reports the percentage of months a cell was covered by surface water (p_{sw}), and then use variation only in hydrological input (rainfall, infiltration, evaporation) to predict whether a specific cell is covered by water. This method would rely on only simulating variation in hydrology through the simulation of local runoff, and then to infer whether a given cell is covered by water or not in that scenario.

Table A.2
Validating WDB potential with observed surface water from satellite data

	All wards	Switchers (5%)	Switchers (10%)
Panel A: All surface water (0-100% occurrence)			
WDP	0.092*** (0.015)	0.419*** (0.114)	0.255** (0.125)
R ²	0.01	0.09	0.06
Observations	2,966	142	71
Panel B: Intermittent surface water (0-50% occurrence)			
WDP	0.043*** (0.004)	0.209*** (0.038)	0.278*** (0.054)
R ²	0.05	0.18	0.28
Observations	2,966	142	71
Panel C: Infrequent surface water (0-25% occurrence)			
WDP	0.017*** (0.002)	0.135*** (0.016)	0.124*** (0.019)
R ²	0.04	0.34	0.39
Observations	2,966	142	71

Note: * p<0.01 ** p<0.05 *** p<0.1. WDP is the eight-week average share of area of ward covered in stagnant water, ~(0,1) and the outcome is the average share of surface water in each ward over the period 1984-2021. Switchers (5%) and (10%) indicate the wards that switch from going to below 5% to above 5%, and below 10% to above 10% stagnant water respectively, within the period of our test score analysis. Panel A uses all observed surface water, including permanent water (100% occurrence), in the outcome variable. Panel B limits the outcome variable to only intermittent surface water (0-50% occurrence), while Panel C limits it to only infrequent surface water (0-25% occurrence).

A. IV Validation with DHS data

To complement the satellite validation and ensure that our measure captures the *dynamic* variation of surface water over time in each location, we utilise an indicator which is collected in DHS: The amount of time (in minutes) the household has to their main source of water. We estimate the effect of WBD Potential on the time it takes to the water source, and also divide the sample by the source of water of the household: Whether they preliminarily obtain water from a tap, well or from

a natural source of surface water. Intuitively, if WBD Potential accurately captures an increase in stagnant water, the amount of nearby surface water in the ward should increase, reducing the time it takes to get to the closest water source *only for households that collect water from nature*. **Table A.3** reports these results. We find that higher WBD potential leads to a lower reported time to nearest water source only for *natural* sources of water, such as lakes, ponds and rivers. Relating the coefficient to the mean of the dependent variable, we find that a 100% treated area, a fully flooded location, would lead to a close to zero distance to the nearest source of natural water. This suggests that an increase in WBD Potential leads to a salient increase in the local stagnant water share reported by water-collecting households.

Table A.3
Time to water by water source

	(1)	(2)	(3)	(4)
	Dependent: Time to water (minutes)			
	All	Tap	Well	Nature
WDP	-4.967 (23.80)	-37.56 (33.73)	13.25 (34.46)	-68.38** (28.98)
Mean DV	40	33	42	49
Obs.	13,546	3,479	4,617	2,514
Clusters	241	155	202	176

Note: Standard errors parentheses clustered on DHS grid-cell level. All estimations use calendar month, grid cell and wave fixed effects. * p<0.01 ** p<0.05 *** p<0.1. WBD Potential is the average per cent share of the area covered in stagnant water the eight weeks prior to the date of survey. Each column represents a DiD estimation with a different subsample. The first column includes the full sample. We then divide the sample by which source of water the household states: Piped or tap water (2), from any type of well (3), from nature i.e. a river, dam, lake, stream, canal, pond (4). Individual level controls include birth order, multiple birth, gender, age, mother's age, total fertility of mother, toilet type.' Sum of past eight weeks precipitation per village/cluster also included.

B Detailed background on Waterborne Diseases

B. I Mechanisms for contagion and disease symptoms

Waterborne diseases are adverse health conditions caused by pathogens that are transmitted by the intake of or contact with pathogen-polluted water, such as by the intake of harmful bacteria or worms. These pathogens include bacteria, viruses and worms, and common diseases are cholera, typhoid fever, and dysentery. **Table B.1** summarises some of the most commonly occurring diseases and symptoms, by type of pathogen. Symptoms vary depending on its cause, but the most common one by far is diarrhoea and other issues relating to the gastrointestinal system, such as abdominal pain ([Magana-Arachchi and Wanigatunge, 2020](#)).

For clarification, neither malaria nor chemically polluted water that causes health issues are typically considered waterborne diseases. First, malaria is not a waterborne disease. Malaria is a serious and sometimes fatal disease which infects humans via a parasite carried by mosquitoes

Table B.1
Common waterborne diseases and symptoms

Pathogen type	Example	Common symptoms
<i>Bacteria</i>	Cholera, Salmonella (Typhoid fever), Shigella (Dysentery), E.coli, Legionella	Diarrhoea, Fever, blood in stool
<i>Viruses</i>	Rotavirus, Adenovirus, Astrovirus, Hepatitis A and E	Diarrhoea, Gastroenteritis, Fever
<i>Protozoa parasites</i>	Cryptosporidia, E. histolytica	Diarrhoea, Gastrointestinal illness
<i>Parasitic worms (Helminths)</i>	Roundworms, Hookworms, Trematodes (flat worms), Schistosomiasis	Fever, Abdominal pain, Diarrhoea, Gastrointestinal illness, Malnutrition,

Note: Sources: [Magana-Arachchi and Wanigatunge \(2020\)](#), [Hedley and Wani \(2015\)](#), [WHO \(2019\)](#).

(and not through contact with contaminated water per se) ([WHO, 2019](#)). Malaria causes flu-like symptoms such as fever and vomiting, but is in contrast to waterborne diseases not associated with diarrhoea. Second, chemically polluted water is not a waterborne disease. While water polluted with e.g. pesticides ([Boedeker et al., 2199](#)) or arsenic ([Mandal et al., 1996](#)) lead to large adverse health effects, they are not waterborne diseases since this term typically is reserved for pathogen-induced disease.

B. II Spread, infection and recovery of waterborne diseases

Waterborne diseases spread via water, either by directly drinking water infected with waterborne disease pathogens or by swimming in contaminated water. For most waterborne diseases, the so-called faecal-oral channel where contaminated human feces spread to water or food which then is ingested by another human is the most common source of an outbreak ([Magana-Arachchi and Wanigatunge, 2020](#)). For example, a person could become infected by eating food prepared from agricultural output using human faeces as fertiliser. Thus, the spread of waterborne disease is inextricably associated with Water, Hygiene and Sanitation Practices (WASH) and improving these can break the faecal-oral transmission channel.

Waterborne pathogens occur naturally in rivers and lakes, but grow exponentially under conditions with stagnant water that become contaminated. Stagnant water enables pathogens to form biofilms and cluster close together which enables faster reproduction. Poor sanitation means an outbreak can spread across persons and households via the fecal-oral channel. In a lab-controlled environment, exponential growth of bacteria causing waterborne disease can occur within hours ([Farhat et al., 2018](#); [Zlatanović et al., 2017](#)). [Ling et al. \(2018\)](#) found that the whole water supply of a Chinese city became contaminated within six days under conditions where water stayed stagnant in the city's plumbing system.

For most contractions of waterborne diseases, the time from first contact to disease outbreak is a few days. For example, cholera takes between 2 hours to 5 days for a person to show symptoms after first ingesting contaminated food or water (Azman et al., 2199). Symptoms can last from days (e.g. a virus infection) to years (some types of worms), but most common is days or a few weeks (Percival et al., 2014). In severe cases waterborne disease can be fatal, in particular for young children, with severe diarrhoea leading to dehydration and death if left untreated.

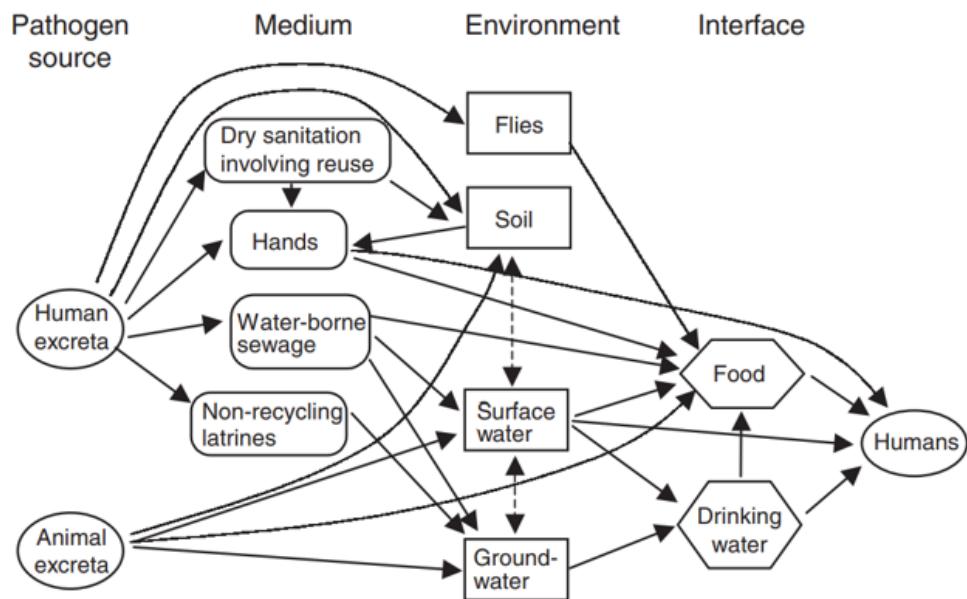


Figure B.1
Main transmission pathways of waterborne diseases

Note: This diagram shows the main transmission mechanisms for diarrhoeal diseases through the fecal-oral route, adapted from Prüss-Üstün et al. (2004).

B. III Waterborne disease burden in Sub-Saharan Africa

According to the UN, the Sustainable Development Goal to achieve safe drinking water for all will fall short of 1.6 billion people by 2030 (UN, 2022). The consequence of this shortfall is dire: The United Nations estimate that 829,000 people die from a lack of safely managed drinking water each year (UN, 2022). Strikingly, the burden of waterborne disease caused by unclean water fall almost exclusively on developing countries and in particular Sub-Saharan Africa (Anthonj et al., 2018). For instance, Black et al. (2010) estimate that each day 2,000 children under the age of five die in Africa due to diarrhoea – the second largest source of child mortality⁴³.

However, the mortality number masks an even higher incidence of disease: Troeger et al. (2018) found that average diarrhoea episodes per person in Sub-Saharan Africa were 1.05 per year. Figure B.2 shows that despite improvements in child mortality due to the introduction of treatments such as ORS, incidence remains high and has barely changed in the last 30 years. Thus, waterborne

⁴³ 18% of total mortality, the largest source of mortality being neonatal conditions in the first 28 days of life for infants (Black et al., 2010)

disease does not only pose a fatal risk, but also when less severe is likely to affect the vast majority of people in the region several points in their lives.

In terms of age heterogeneity, the most vulnerable are typically young children and old individuals. [Figure B.3](#) shows diarrhea prevalence in Tanzania by age group. There is typically a large reduction in prevalence from the age of below 1 years to 5 years, but after this it tends to flatten out. In the DHS data, we do find larger baseline diarrhea prevalence for 1-year olds which then reduces by age. However, as [Figure E.2](#) based on our empirical analysis shows, the *increase* in diarrhea risk following a stagnant water shock does not follow the same age pattern as baseline diarrhoea, but instead seems to peak at age 1-2 years, around the age where most children are no longer breastfed, and flattens out after this, with children up to 5 still seeing a large increase in diarrhoea prevalence from exposure to stagnant water.

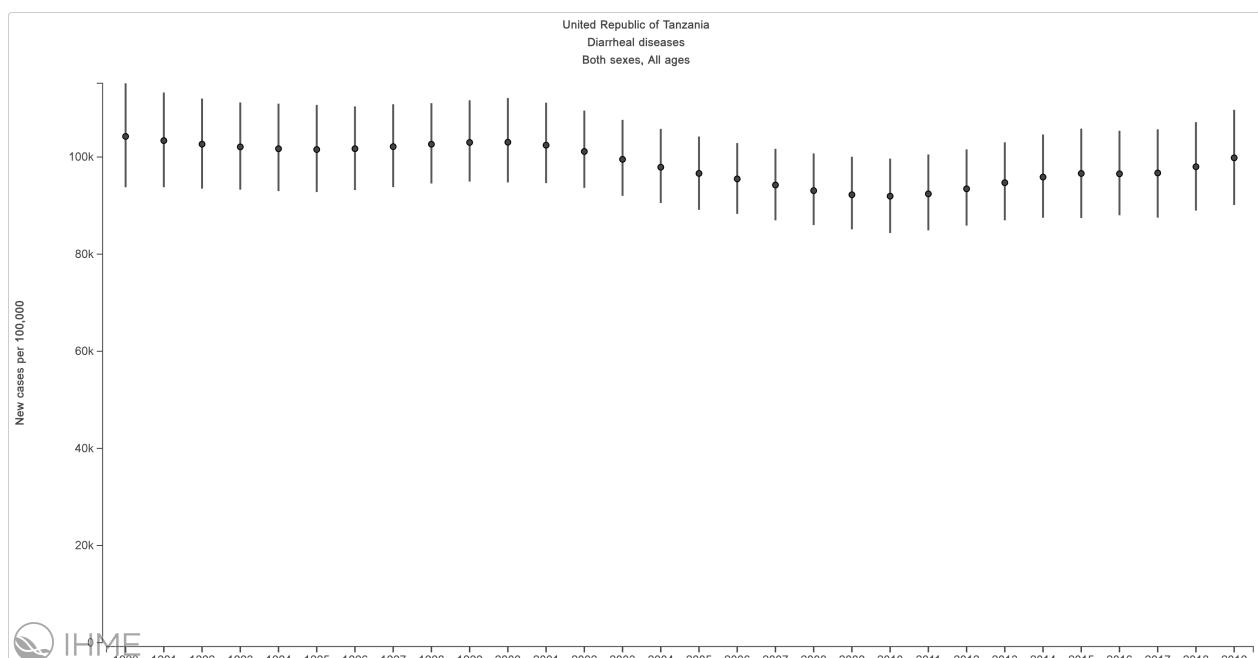


Figure B.2
Diarrhoea incidence by year in Tanzania

Note: This figure shows yearly diarrhoea incidence rates for Tanzania, based on data from the Global Health Data Exchange ([Global Burden of Disease Collaborative Network, 2021](#))

B. IV Geographic distribution of diarrhoeal disease in Tanzania

C Institutional background

C. I Prevalence and causes of waterborne disease in Tanzania

Waterborne diseases are illnesses caused by pathogens that are transmitted through contaminated water sources⁴⁴. Tanzania lies in the ‘belt’ of the world’s highest waterborne disease incidence, which stretches through central and eastern Sub-Saharan Africa, a region characterised both by a

⁴⁴See [Appendix B](#) for more details on the definition and characteristics of waterborne diseases

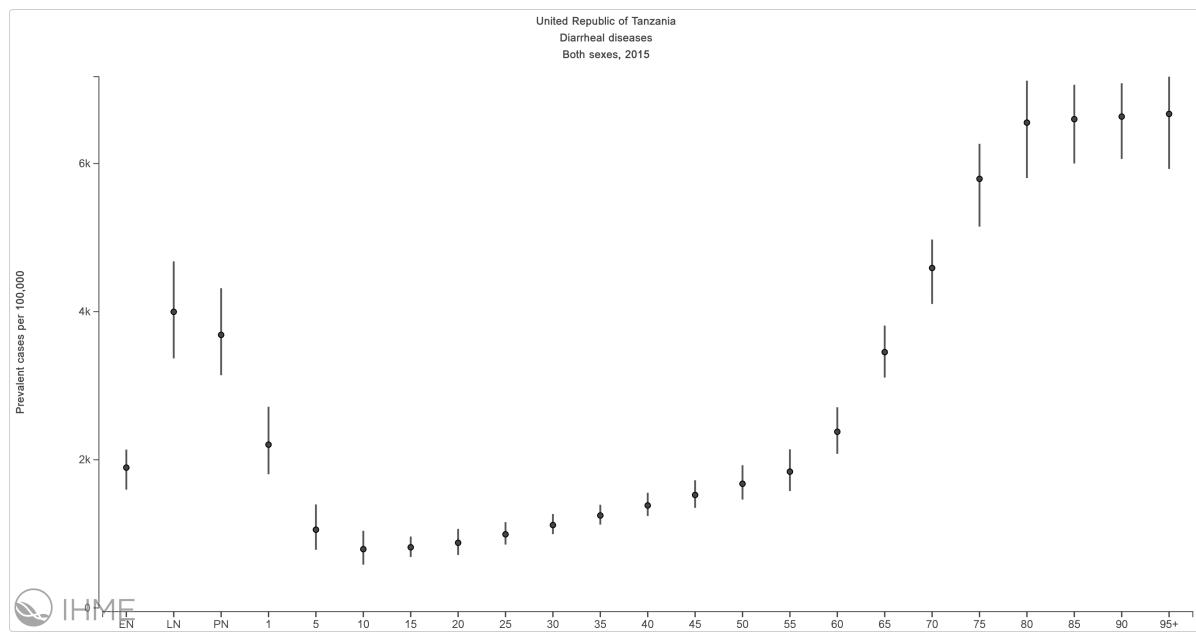


Figure B.3
Diarrhoea prevalence by age in Tanzania

Note: This figure shows diarrhoea prevalence rates for Tanzania, based on data from the Global Health Data Exchange ([Global Burden of Disease Collaborative Network, 2021](#))

favourable climate for waterborne pathogens as well as lack of access to safe water and sanitation. The number of diarrheal episodes per person per year is 1.1 episodes in Eastern Sub-Saharan where Tanzania is located, which is only second in the world to Central Sub-Saharan Africa (1.21) ([Troeger et al., 2018](#)). Tanzania has had several outbreaks of waterborne diseases such as cholera in the past few decades. A recent example of a cholera outbreak occurred around Tanzania's capital, Dar es Salaam, in 2015, with 16,521 reported cholera cases in total ([Chae et al., 2022](#)), but other waterborne disease outbreaks have been documented as recently as 2022 ([Masunga et al., 2022](#); [WHO, 2022](#)). Waterborne diseases have historically been classified as 'neglected' diseases by Tanzanian authorities and have received much less attention than officially prioritised diseases such as HIV and malaria ([Tanzania Ministry of Health and Social Welfare, 2008](#)), despite the fact that waterborne diseases carry a higher disease burden than HIV and Malaria for children in Tanzania ([Vos et al., 2020](#)).

Environmental factors play a critical role in enabling the survival, growth and transmission of waterborne pathogens⁴⁵. El Niño events, which occur every 3-5 years, and lead to an increase in rainfall and flood events across East Africa are associated with a three-fold increase in cholera incidence ([Moore et al., 2017](#)). In a review of the epidemiological literature, [Levy et al. \(2016\)](#) found that the most important predictors of diarrheal disease outbreaks were floods, followed by heavy rainfall and high temperature. Heavy rainfall is thought to affect diarrhea incidence primarily through its effect on floods and surface water contamination ([Levy et al., 2016](#)). Detailed mapping in a district in Tanzania showed that low-lying areas with high water-tables are more susceptible to cholera as a result of rainfall, as this leads to the accumulation of surface water which contaminates

⁴⁵ See [Appendix B. II](#) for more details on transmission mechanisms

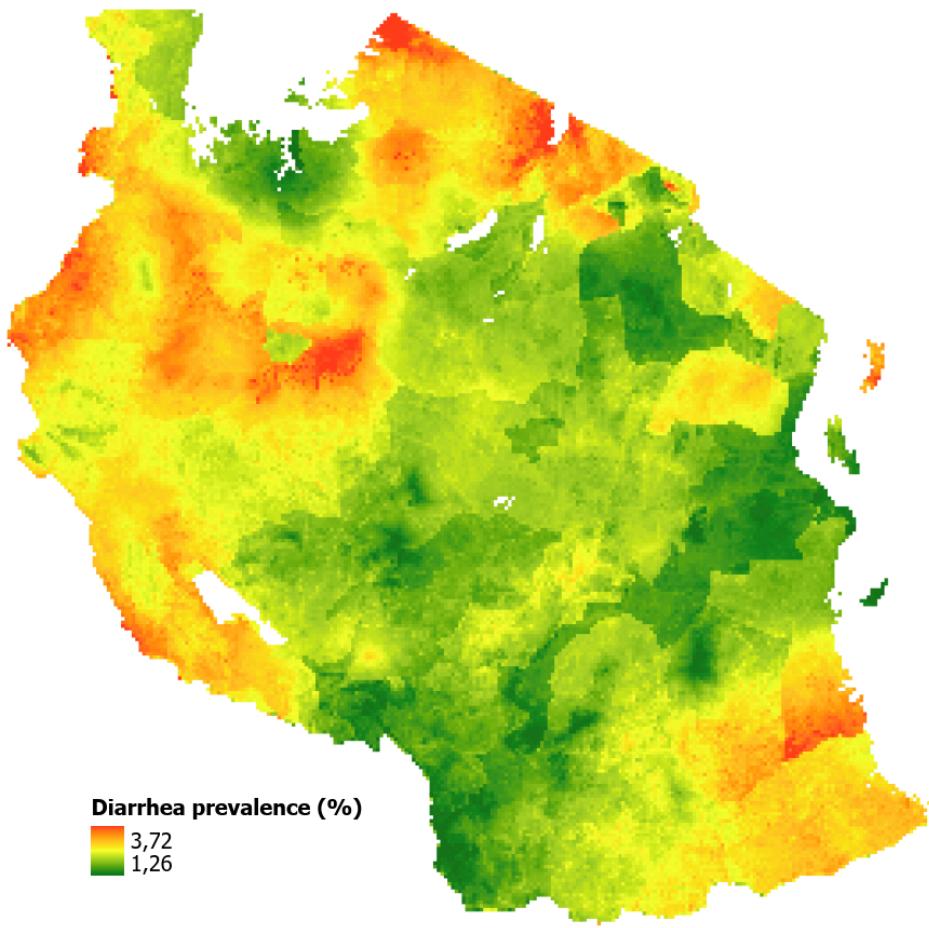


Figure B.4
Modelled geographic distribution of diarrhoeal disease in Tanzania

Note: This map shows the modelled geographic distribution of diarrhoeal prevalence for Tanzania, based on a Bayesian geostatistical model for diarrhea prevalence, from [Reiner et al. \(2020\)](#).

pit latrines and shallow drinking water wells ([Mayala et al., 2003](#)), which lends support to our empirical strategy.

C. II Prevention and treatment of waterborne diseases in Tanzania

Tanzania has made great strides in reducing childhood diarrhea mortality, with the mortality rate declining by 89% from 1980 to 2015 ([Masanja et al., 2019](#)), but incidence remains largely unchanged. Most of the lives saved are attributed to treatment with oral rehydration salts (ORS), which replenishes fluids lost by diarrhea. This makes children more resilient to the worst consequences of diarrhea, but does not affect the probability of contracting the disease in the first place. These are relatively affordable on the global market, costing close to 0.56 USD per treatment ([UNICEF, 2022](#)). In contrast, less than 3% of the reduction in diarrhea-related mortality is attributed to improvements in water and sanitation, which is evident by the fact that higher-quality sanitation coverage increased only from 8 to 15% over the same period ([Masanja et al., 2019](#)). Hence, the focus has been on treatment rather than prevention, which has left room for large sub-national variation in disease incidence ([Reiner et al., 2020](#)). In high-income countries, large-scale

historical investments in water and sanitation have been crucial in reducing childhood mortality and diarrheal incidence ([Alsan and Goldin, 2019](#)). However, this infrastructure is costly, and lower-cost piecemeal approaches in Tanzania have been unable to curb diarrhea-related morbidity ([Briceño et al., 2017](#)).

Treatment with ORS or antibiotics is usually administered at health clinics, which implies that access to high-quality healthcare is necessary to relieve consequences of waterborne disease infection. In 1984 Tanzania set up the National Control of diarrheal Disease to combat childhood mortality, which focused on creating local clinics that could administer ORS ([Masanja et al., 2019](#)). This may explain why care-seeking for diarrhea in children in Tanzania is among the highest in Sub-Saharan Africa, with more than half of children sick with diarrhea taken to a health clinic ([Schellenberg et al., 2003](#)), and more than 90% live closer than 5 km from a primary health facility ([Tanzania Ministry of Health and Social Welfare, 2008](#)). However, this masks large inequalities in the quality of received healthcare. A study in rural Tanzania found that only 1 of 6 children with diarrhea received ORS, citing low diagnostic capabilities and a lack of medical supplies ([Schellenberg et al., 2003](#)), while other parts of Tanzania see ORS administration rates over 50% ([Masanja et al., 2019](#)). It is not uncommon for rural families to bypass the local health clinic, especially if living close to a hospital ([Kahabuka et al., 2011](#)), which suggests that while there is high access to health clinics, quality is often poor and unevenly distributed. Thus, we expect waterborne diseases to spread more easily in urban, densely populated areas, where contamination of water sources is more common, but that health consequences conditional on having contracted a waterborne disease may be worse in rural areas with lower access to high-quality healthcare.

C. III Education and learning in Tanzania

In Tanzania, schooling starts with pre-primary schooling at ages 5-6, although it is common that children also attend pre-school for 2-3 years, which by global standards is relatively formal schooling ([Bietenbeck et al., 2019](#)). This is followed by seven years of primary schooling at ages 7-13, four years of ordinary secondary school (ages 14-17), and two years of advanced level secondary school (ages 18-19)⁴⁶. The adult literacy rate was 77.5% for men and 62.2% for women in 2012, but literacy among current pupils is higher, with the literacy rate being 86% for 15-24 year-olds. In 2012, 83% of the population reported having attained primary schooling, and 12.9% secondary schooling. Schooling expansion has been swift in Tanzania, and for example ordinary secondary school enrolment has increased from 6% in 2002 to 34% in 2013.

The effect of waterborne diseases on learning and education as a whole is understudied, but there are key links between health and the state of learning in Tanzania. For one, many students drop out and do not finish primary schooling; 65.1% (72.8%) of boys (girls) finish primary schooling. Most dropouts are due to unknown reasons (truancy), but health reasons are common. For one, early-life stunting, from e.g. repeated exposure to diarrheal diseases, is an important cause of both absence and lower cognitive ability. Second, current health issues also lead to greater dropouts. For

⁴⁶This information and subsequent on Tanzania school system from [UNESCO \(2014\)](#) and [World Bank \(2016\)](#)

example, in 2014, a survey conducted in three Tanzanian regions stated that health problems caused 6.2% of dropouts, and in a 2013 school census, 3% of survey respondents reported dropping out due to illness or having to take care of someone ill ([UNESCO, 2014](#)). Even school absence as short as 10 days has been shown to have long-term consequences ([Cattan et al., 2023](#)) indicating that even if health is only temporarily affected, losses from learning may be permanent. Lastly, even when attending school, learning can be limited: In a survey performed by the World Bank, only 40% of students in year 4 (mainly ten-year-olds) could perform a year 3 mathematics task such as $6 \div 3$ or 7×8 . An important cause of limited learning is low quality of educational resources ([Mgema, 2022](#); [Ilomo and Mlavi, 2016](#)), however fatigue or frequent absences due to waterborne diseases are likely to also negatively affect the capacity for learning.

D Descriptive statistics about treatment

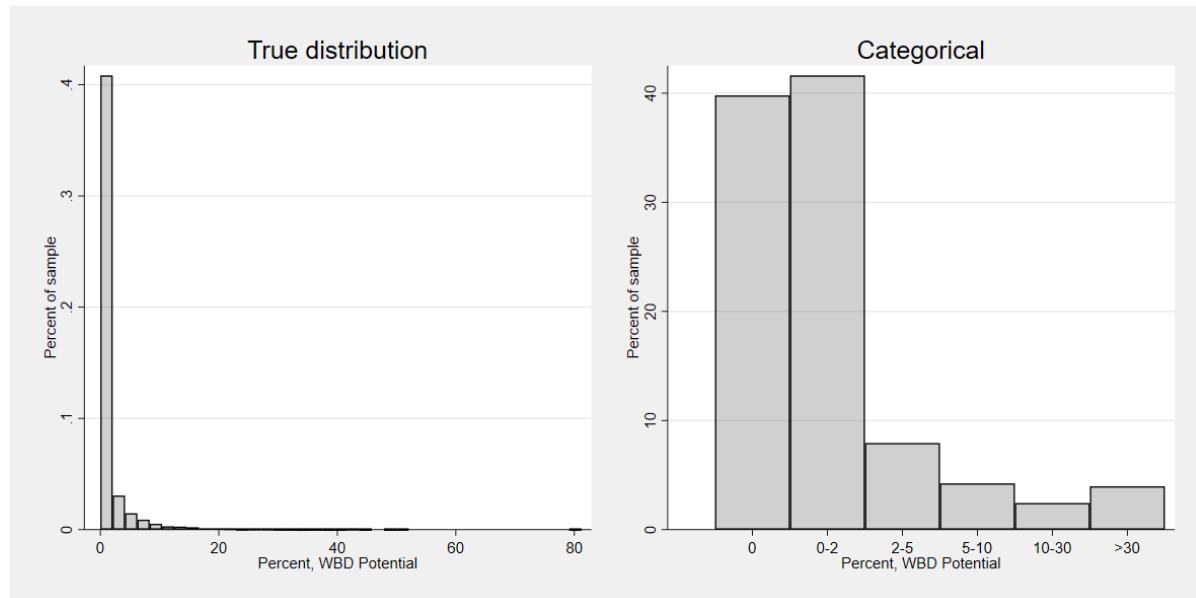


Figure D.1
Distribution of WBD Potential

Note: This figure depicts the distribution of waterborne disease potential (WBD Potential), where a ward w during wave and survey year y has one simulated value between 0 and 1, which is the share of the ward area covered by stagnant water. The left subplot depicts the distribution of WBD Potential, and to provide additional clarity the right subplot displays the distribution across five categories of shares of stagnant water.

E Additional results with DHS

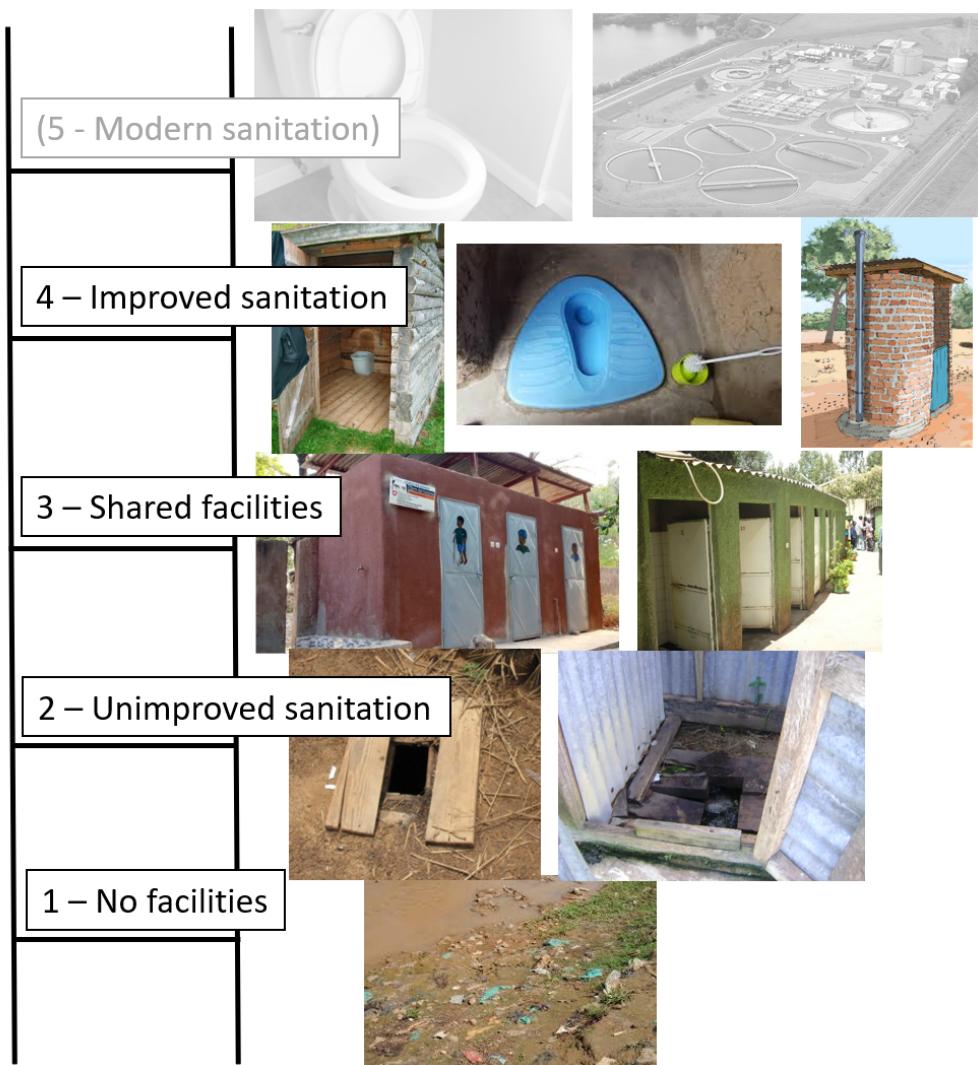


Figure E.1
UN Sanitation ladder: Examples

Note: In this image we provide examples of how the different steps in the UN sanitation may look like. In reality, there are five steps of the ladder, the highest rung being modern sanitation. However, this category is not represented in our sample so for our case we consider a four-step ladder of sanitation. In 1, there are no facilities which includes open defecation. In 2, there are unimproved sanitation facilities that are not integrated to a well-functioning sanitation system. In 3, we include shared facilities. In 4, we have improved sanitation that are well-maintained and of higher standard and technology.

Table E.1
Effect of WBD Potential on diarrhoea: Sensitivity to control variables

	(1)	(2)	(3)	(4)	(5)	(6)
<i>Dependent: Child has had diarrhoea</i>						
WDP 1	0.303*** (0.0895)	0.302*** (0.115)	0.305*** (0.116)	0.295*** (0.0901)	0.289*** (0.0939)	0.297*** (0.0910)
Mean DV	0.13	0.13	0.13	0.13	0.13	0.13
Obs.	19,399	16,740	16,740	19,399	19,399	19,399
Clusters	252	242	242	252	252	252
Full sample:	✓			✓	✓	✓
Cov sample:		✓	✓			
Covariates:		✓		✓	✓	✓
Local precip:					✓	✓
Sampling weights:					✓	

Note: Standard errors in parentheses clustered on DHS gridcell level. All estimations use calendar month, gridcell and wave fixed effects. * p<0.01 ** p<0.05 *** p<0.1. WBD Potential is the average percent share of area covered in stagnant water the eight weeks prior to date of survey. Individual covariates include: Birth order, twin child, child sex, child age, mother's age, mother's total fertility, household toilet type, wealth index. Full sample includes all children's where the outcome, diarrhoea, is available. Covariate sample is the sample with all covariates – only toilet type and wealth index are ever missing. Column 1 is the specification with the full set of fixed effects but no control variables. In column 2 all individual covariates are included without accounting for the share of missing, and in column 3 we still limit the sample to children without controls but excluding covariates. In column 4 we account for missing variables by imputing an arbitrary number (-999) for missing variables and including a corresponding dummy variable = 1 for when the variable takes that value. Finally, in column 5 we include local precipitation as a control. Column 5 applies sampling weights and column 6 is our preferred specification weightout weights.

Table E.2
Shared toilets and the effect of waterborne disease on diarrhoea

	(1)	(2)	(3)
<i>Dependent: Child has had diarrhoea</i>			
	All	Urban	Rural
WDP	0.12 (0.13)	0.16 (0.21)	0.059 (0.24)
Shared toilet	0.028*** (0.0090)	0.015 (0.018)	0.026** (0.010)
WDP*Shared toilet	0.13 (0.18)	0.38** (0.15)	-0.11 (0.21)
Obs.	13,131	3,219	9,912
Clusters	237	98	228

Note: Standard errors in parentheses clustered on DHS gridcell level. All estimations use calendar month, gridcell and wave fixed effects.* p<0.01 ** p<0.05 *** p<0.1. WBD Potential (%) is the average percent share of area covered in stagnant water the eight weeks prior to date of survey. 'Shared toilet'=1 for households who use shared toilet facilities.

Table E.3
Effect of WBD potential on diarrhoea: Urban-rural heterogeneity

	Urban	Rural
	(1)	(2)
WDP	0.356*** (0.105)	0.251** (0.124)
Observations	4,209	15,190
Clusters	109	244

Note: Standard errors in parentheses clustered on DHS gridcell level. All estimations use calendar month, gridcell and wave fixed effects and include individual covariates.* p<0.01 ** p<0.05 *** p<0.1. WBD Potential (%) is the average percent share of area covered in stagnant water the eight weeks prior to date of survey.

Table E.4
Effect of WBD potential on diarrhoea: Heterogeneity by annual precipitation

	Dry	Rainy
	(1)	(2)
WDP	0.378*** (0.088)	0.158 (0.145)
Observations	9,416	9,647
Clusters	138	112

Note: Standard errors in parentheses clustered on DHS gridcell level. All estimations use calendar month, gridcell and wave fixed effects and include individual covariates.* p<0.01 ** p<0.05 *** p<0.1. WBD Potential (%) is the average percent share of area covered in stagnant water the eight weeks prior to date of survey. FIX TABLENOTES

Table E.5
Effect of WBD potential on diarrhoea: Heterogeneity by gender

	Girls	Boys
	(1)	(2)
WDP	0.407*** (0.143)	0.164 (0.099)
Observations	9,705	9,691
Clusters	250	251

Note: Standard errors in parentheses clustered on DHS gridcell level. All estimations use calendar month, gridcell and wave fixed effects and include individual covariates.* p<0.01 ** p<0.05 *** p<0.1. WBD Potential (%) is the average percent share of area covered in stagnant water the eight weeks prior to date of survey.

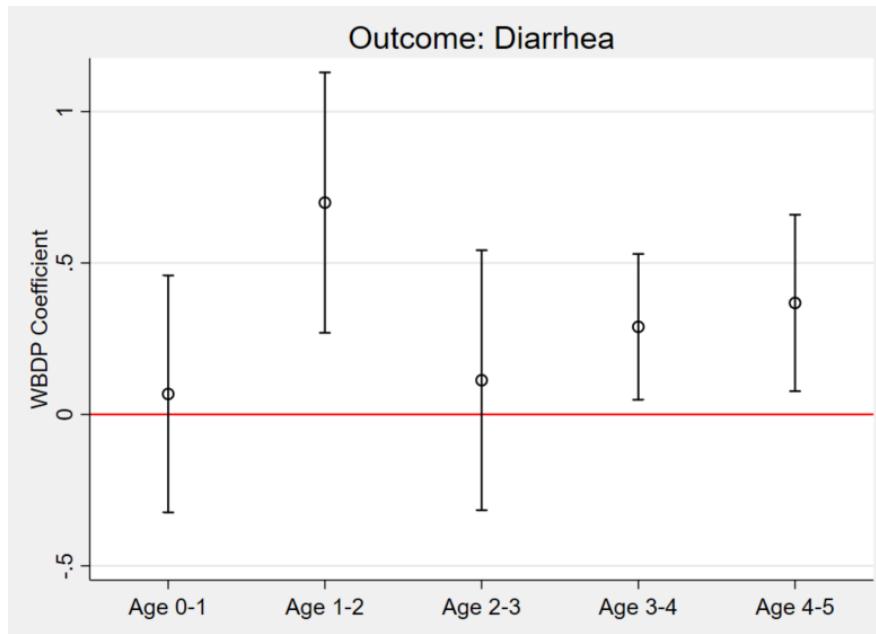


Figure E.2
WDP on diarrhea by age

Note: This figure summarises DiD estimate of WBD Potential on the share of children with recent diarrhoea. Coefficient and 95% confidence intervals are displayed. We run each regression separately for each age group. In all estimations we include calendar month, wave and ward fixed effects as well as individual and household covariates. Standard errors are clustered on ward-level.

Table E.6
Effects of WBD potential on health outcomes without gridcell FE

	(1)	(2)	(3)	(4)	(5)	(6)
	WBD		Placebo			
	Diarrhoea	W.Age	Fever	Cough	Anemia	Height
WDP	0.298*** (0.068)	1.476 (3.547)	0.362*** (0.092)	0.143* (0.078)	0.030 (0.101)	-6.536 (14.790)
Observations	19,399	18,152	19,468	19,469	21,108	18,783
Clusters	252	252	252	252	252	252

Note: Standard errors in parentheses clustered on DHS gridcell level. All estimations use calendar month and wave fixed effects and include individual covariates.* p<0.01 ** p<0.05 *** p<0.1. WBD Potential (%) is the average percent share of area covered in stagnant water the eight weeks prior to date of survey.

Table E.7
Effects of WBD potential on malaria awareness and preparedness

	(1) Malaria important	(2) Avoid malaria	(3) Can protect	(4) Use bednet
	(1)	(2)	(3)	(4)
Panel A: Without gridcell FE				
WDP	1.242*** (0.352)	0.353*** (0.097)	0.084*** (0.031)	0.761*** (0.152)
Observations	10,230	10,233	21,432	16,395
Clusters	202	202	252	242
Panel B: With gridcell FE				
WDP	0.331 (0.370)	-0.019 (0.122)	0.031 (0.030)	0.129 (0.156)
Observations	10,230	10,233	21,432	16,395
Clusters	202	202	252	242

Note: Standard errors in parentheses clustered on DHS gridcell level.* p<0.01 ** p<0.05 *** p<0.1. WBD Potential (%) is the average percent share of area covered in stagnant water the eight weeks prior to date of survey. 'Malaria important' is an indicator for whether malaria is stated as the most important health problem in the community. 'Avoid malaria' and 'Can protect' are indicators for whether an individual knows how to avoid contracting malaria and can protect themselves against malaria, respectively. 'Use bednet' is an indicator for whether an individual is currently using bednets against malaria.

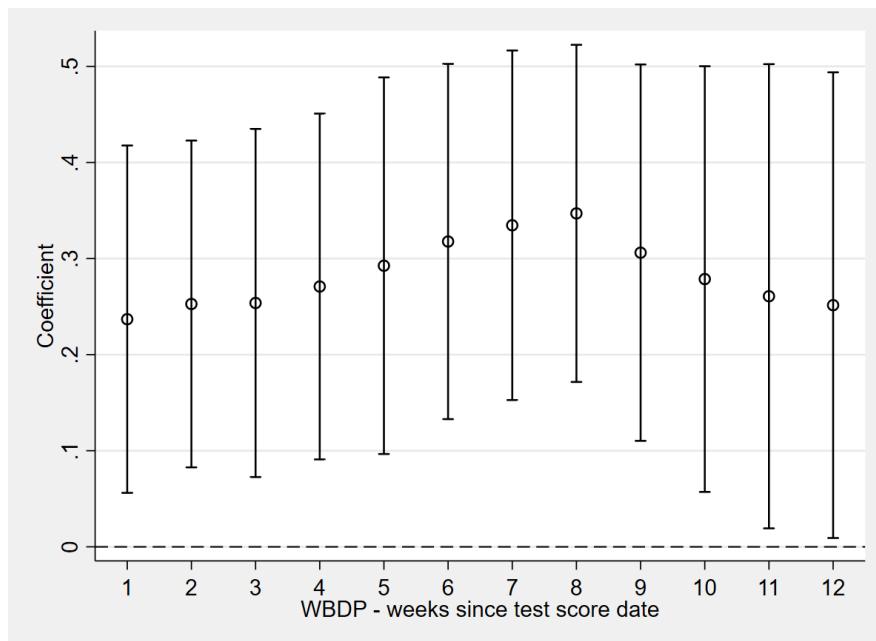


Figure E.3
WDP by week: Effect on diarrhoea

Note: This figure summarises DiD estimate of WBD Potential on the share of children with recent diarrhoea. Coefficient and 95% confidence intervals are displayed. Here, for each estimation we redefine which week we include as the measure of WBD Potential. In the first estimation, we define WBD Potential as the share of stagnant water in the one week preceding the date of survey when the child is tested. In the second estimate, we instead take the share of stagnant water in the week which starts two weeks before the date of the survey and ends the week before the survey. Similarly, in the estimation labelled "3" we define WBD Potential as the share of stagnant water in (only) the week three weeks prior the date of survey. In all estimations we include calendar month, wave and ward fixed effects as well as individual and household covariates. Standard errors are clustered on ward-level.

F Additional results with test scores

F. I Results with test scores: Specification and heterogeneity

[Table F.1](#) shows additional specifications for the effect of WBD Potential on test scores, and we find that the main coefficient of interest does not change in an economically meaningful way across specifications, although we lose some precision when adding wave*month fixed effects if we do not also include calendar month fixed effects (non-interacted).

Table F.1
Effect of WBD Potential on test scores: Additional specifications

	(1)	(2)	(3)	(4)	(5)	(6)
<i>Dependent: Test score (std)</i>						
WDP	-0.716** (0.316)	-0.656** (0.314)	-0.590* (0.318)	-0.683** (0.314)	-0.590* (0.318)	-0.683** (0.314)
Local precipitation	0.00318*** (0.00118)			0.00315*** (0.00122)		0.00315*** (0.00122)
Obs.	368,444	368,493	368,493	368,444	368,493	368,444
Clusters	3,842	3,842	3,842	3,842	3,842	3,842
Covs	✓	✓		✓		
Ward FE	✓	✓	✓	✓	✓	✓
Wave FE	✓	✓				
Month FE	✓	✓			✓	✓
Wave*Month FE			✓	✓	✓	✓

Note: Results is the effect of WBD Potential on test scores. Standard errors in parentheses clustered on ward. * p<0.01 ** p<0.05 *** p<0.1. WBD Potential is the average share of ward covered in stagnant water in the eight weeks preceding the date of the survey. Covariates include child's gender and age, and mother's age and secondary education, a wealth index, local past eight weeks of ward precipitation. Ward is the treatment unit of observation. Wave is the year of the survey and waves included are 2011, 2013, 2014, 2015 and 2017. Month is the calendar month of treatment.

Table F.2
Correlation between local precipitation and WBD Potential in the Uwezo sample

	(1)	(2)	(3)	(4)
	WDP	Test score (std)	Test score (std)	Test score (std)
WDP		-0.750** (0.349)		-0.745** (0.350)
Local rainfall	-0.000 (0.000)		-0.000 (0.001)	-0.000 (0.001)
Observations	496,353	368,202	368,153	368,153
Clusters	3,841	3,840	3,840	3,840

Note: Standard errors parentheses clustered on ward. Wave, calendar month and ward fixed effects included in all estimations. * p<0.01 ** p<0.05 *** p<0.1. WBD Potential is the eight-week average share of area of ward covered by stagnant water, $\sim(0,1)$. 'Local rainfall' is the sum of rainfall in mm over the past eight weeks, by ward and wave.

Table F.3
Correlation between local precipitation and WBD Potential in the DHS sample

	(1)	(2)	(3)	(4)
	WDP	Diarrhea	Diarrhea	Diarrhea
WDP		0.303*** (0.090)		0.304*** (0.090)
Local rainfall	-0.145 (0.106)		0.000 (0.161)	0.042 (0.163)
Observations	21,432	19,399	19,399	19,399
Clusters	252	252	252	252

Note: Standard errors parentheses clustered on gridcell. Wave, calendar month and gridcell fixed effects included in all estimations. * p<0.01 ** p<0.05 *** p<0.1. WBD Potential is the eight-week average share of area within a 10 km radius of the village covered by stagnant water, $\sim(0,1)$. 'Local rainfall' is the sum of rainfall in mm over the past eight weeks, by DHS village and wave.

Table F.4
Effect of WBD Potential on test scores: Heterogeneity by urban/rural location

	Urban	Rural
	(1)	(2)
WDP	-1.626*	-0.809**
	(0.924)	(0.384)
Observations	60,284	307,918
Clusters	488	3,352

Note: Results is the effect of WBD Potential on test scores. Standard errors in parentheses clustered on ward. * p<0.01 ** p<0.05 *** p<0.1. WBD Potential is the average share of ward covered in stagnant water in the eight weeks preceding the date of the survey. Covariates include child's gender and age, and mother's age and secondary education, a wealth index, local past eight weeks of ward precipitation. Ward is the treatment unit of observation. Wave is the year of the survey and waves included are 2011, 2013, 2014, 2015 and 2017. Month is the calendar month of treatment.

Table F.5
Effect of WBD Potential on test scores: By annual precipitation

	Dry	Rainy
	(1)	(2)
WDP	-0.799**	-0.205
	(0.403)	(0.774)
Observations	163,195	162,771
Clusters	1,507	1,600

Note: Results is the effect of WBD Potential on test scores. Standard errors in parentheses clustered on ward. * p<0.01 ** p<0.05 *** p<0.1. WBD Potential is the average share of ward covered in stagnant water in the eight weeks preceding the date of the survey. Covariates include child's gender and age, and mother's age and secondary education, a wealth index, local past eight weeks of ward precipitation. Ward is the treatment unit of observation. Wave is the year of the survey and waves included are 2011, 2013, 2014, 2015 and 2017. Month is the calendar month of treatment. FIX TABLE NOTES

Table F.6
Effect of WBD Potential on test scores: By gender

	Girls	Boys
	(1)	(2)
WDP	-0.659	-0.931**
	(0.439)	(0.362)
Observations	170,639	197,549
Clusters	3,822	3,836

Note: Results is the effect of WBD Potential on test scores. Standard errors in parentheses clustered on ward. * p<0.01 ** p<0.05 *** p<0.1. WBD Potential is the average share of ward covered in stagnant water in the eight weeks preceding the date of the survey. Covariates include child's gender and age, and mother's age and secondary education, a wealth index, local past eight weeks of ward precipitation. Ward is the treatment unit of observation. Wave is the year of the survey and waves included are 2011, 2013, 2014, 2015 and 2017. Month is the calendar month of treatment. FIX TABLE NOTES



Figure F.1
WBD Potential on test scores, by age

Note: This figure summarises DiD estimate of WBD Potential on test scores. Coefficient and 95% confidence intervals are displayed. We separately present results by the age of the child at survey. In all estimations we include calendar month, Wave and ward fixed effects as well as individual and household covariates. Standard errors are clustered on ward-level.

Table F.7
Effect of WBD Potential on test scores: By malaria prevalence in children

	High malaria	Low malaria
WDP	-0.840*	-0.997
	(0.431)	(0.624)
Observations	165,195	162,664
Clusters	1,612	1,519
Wave FE	✓	✓
Ward FE	✓	✓
Month FE	✓	✓

Note: Results is the effect of WBD Potential on test scores. Wards are split by median Malaria mean prevalence in children aged 2-10 years in 2010. Standard errors in parentheses clustered on ward. * p<0.01 ** p<0.05 *** p<0.1. WBD Potential is the average share of ward covered in stagnant water in the eight weeks preceding the date of the survey. Ward is the treatment unit of observation. Wave is the year of the survey and waves included are 2011, 2013, 2014, 2015 and 2017. Month is the calendar month of treatment.

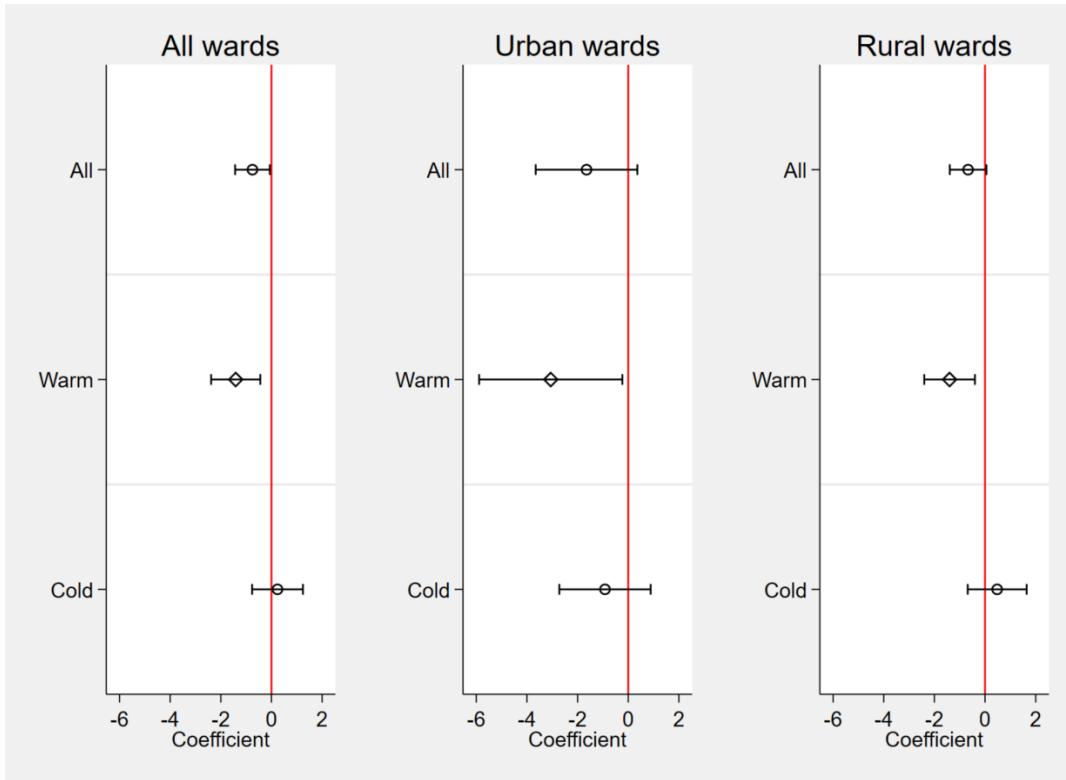


Figure F.2
WBD Potential, by urban-rural wards

Note: This figure summarises DiD estimate of WBD Potential on test scores, splitting each subplot by whether the ward is urban or rural. Coefficient and 95% confidence intervals are displayed. The solid red line represents a zero coefficient of WBD Potential on test scores, and the dashed black line the estimated coefficient size for the full sample. Within each subplot we further split wards by median temperature in the past 8 weeks: *Warm* if the temperature in the past 8 weeks $> 23.23^{\circ}\text{C}$, and *Cold* otherwise.

F. II Results with test scores: Robustness

Table F.8
WBD Potential on test scores, binary treatment definition

Dependent: Test score (std)			
	All	Dry wards	Rainy wards
WDP $\geq 5\%$	-0.101** (0.0406)	-0.102** (0.0457)	-0.0932 (0.0890)
Obs.	368,444	178,449	189,995
Clusters	3,842	1,669	2,173
Wave*District FE			
Ward FE	✓	✓	✓
Month FE	✓	✓	✓

Note: Standard errors in parentheses clustered on ward. * $p < 0.01$ ** $p < 0.05$ *** $p < 0.1$. WBD Potential $> 5\%$ is an indicator for whether the two-week average share of area of ward covered in stagnant water exceeded 5%. Dry ward if mean precipitation < 1000 mm precipitation. Rainy ward if ≥ 1000 mm precipitation. Wave, Calendar month, Ward fixed effects, and ward-level 2-week sum of precipitation, squared WDP included in all estimations. Household covariates included are child's gender and age, and mother's age and whether secondary education or above.

The purpose of [Figure F.6](#) compared to [Figure F.5](#) is that here we can estimate to what extent we have dynamic effects. If children are treated once are more or less affected in later waves by additional treatments (either from additive effects, or from reduced effects due to already being incapacitated), we could expect a bias in either direction. We thus use the even more restricted and only evaluate the *first* time a ward switches into treatment. This yields a coefficient of, but it is less precisely estimated, and again not significantly different from the previous estimates. Since standard errors are bootstrapped for the DHDC estimator, we also provide a comparison to the TWFE with bootstrapped instead of clustered standard errors in our main specification, but this has only a small impact on the standard error.

In [Figure F.6](#) we report event study coefficients using the estimator by [de Chaisemartin and D'Haultfoeuille \(2022\)](#), which allows for heterogeneous and dynamic treatment effects. Due to the restricted sample size and the unbalanced nature of our panel data these coefficients are relatively imprecise, yet we find a significant negative effect on test scores in the year when children are exposed to a stagnant water shock. Interestingly the effect size is as large one year later, but insignificant. Two years later, there is no sign of a negative effect. However, note that this sample is limited to only those locations which were treated once. Locations with multiple treatments over the period are excluded, and hence we are unable to estimate the long-term effects for children who have been subject to multiple shocks.

Given that the results show high autocorrelation of WBD Potential over time, one concern is that our model captures a phenomenon in the future, which we then estimate effects for due to the correlation with past events. Such would be the case if past WBD Potential captures that children expect more favourable environmental conditions or events in the future in a way which affects test scores. If this is the case, *future* WBD Potential (in terms of the date children are surveyed)

Table F.9
Exploring non-linearities: Including squared WDP

Dependent: Test score (std)			
	All	Dry wards	Rainy wards
WDP	-1.133** (0.557)	-1.423** (0.645)	-0.346 (1.785)
WDP Squared	1.152 (0.974)	1.560 (1.072)	3.272 (8.976)
Obs.	368,444	178,449	189,995
Clusters	3,842	1,669	2,173

Note: Standard errors in parentheses clustered on ward. WBD Potential is two-week average share of area of ward covered in stagnant water, $\sim(0,1)$. Dry ward if mean precipitation < 1000 mm precipitation. Rainy ward if ≥ 1000 mm precipitation. Wave, Calendar month, Ward fixed effects, and ward-level 2-week sum of precipitation. Household covariates included are child's gender and age, and mother's age and whether secondary education or above.

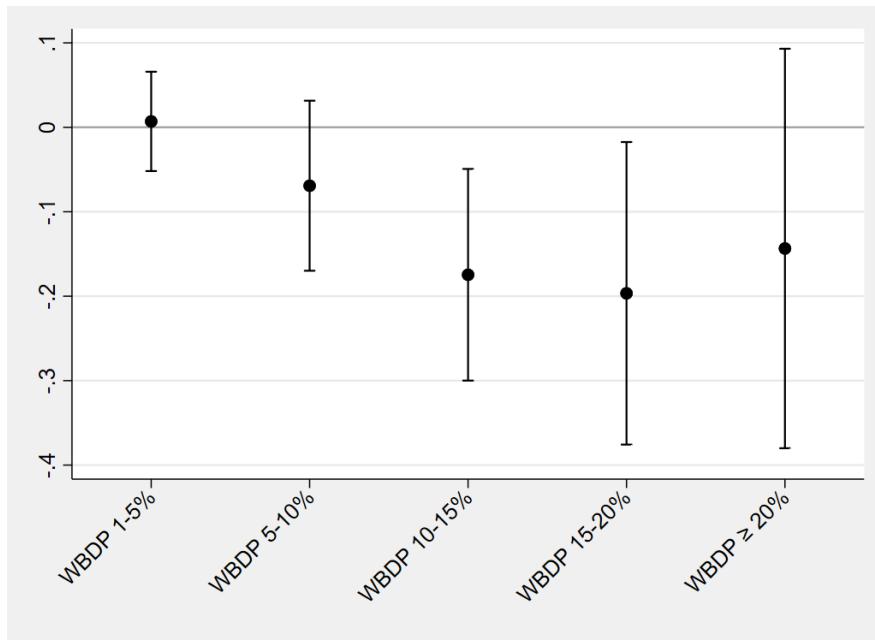


Figure F.3
WDP as indicator with different thresholds

Note: This figure summarises DiD estimate of WBD Potential on test scores. Coefficient and 95% confidence intervals are displayed. Here, we provide a series of dummies for different thresholds of WBD Potential. At baseline, WBD Potential is less than 1%. The first dummy variable thus captures the effect on test scores from WBD Potential between 1 and 5%. Calendar month, wave and ward fixed effects are included in the estimation as well as individual and household covariates. Standard errors are clustered on ward-level.

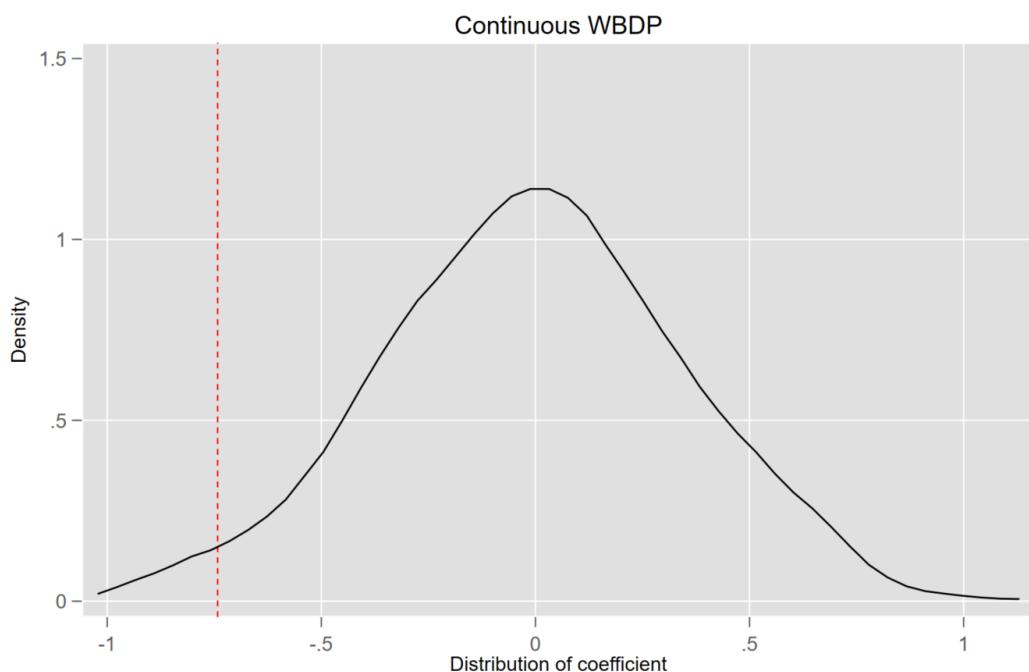


Figure F.4
Randomisation test: Effect of WBD Potential on test scores

Note: This figure depicts the coefficient main DiD estimate capturing the effect of WBD Potential on test scores for 500 randomisation inference tests where WBD Potential has been permuted and the coefficients of WBD Potential on test scores summarised in a kernel density plot. Wave, Calendar month and Ward fixed effects and individual covariates are included in all estimations as well as individual covariates and standard errors are clustered by ward. 3% of the simulated coefficients are greater than our estimate (indicated by the red line).

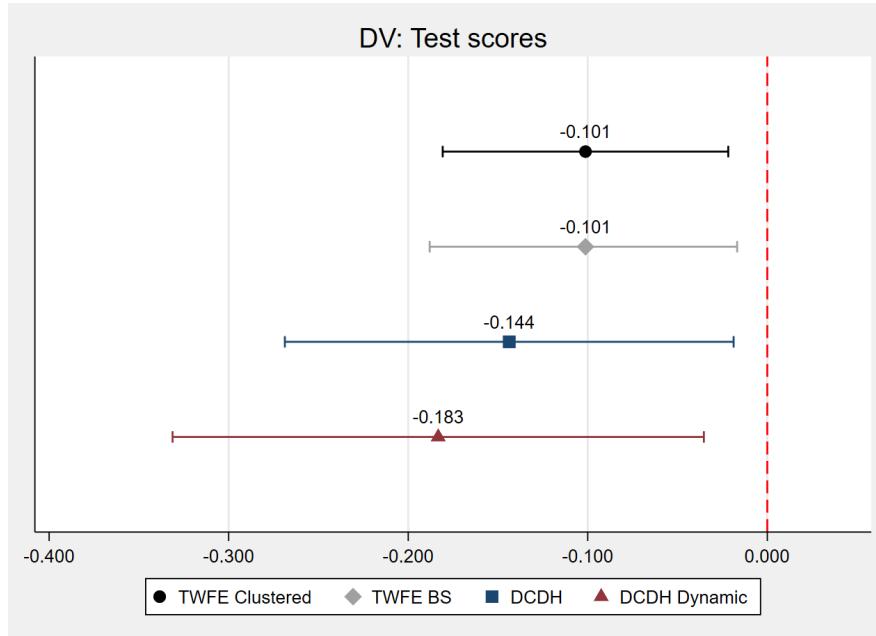


Figure F.5

Comparison of TWFE and alternative estimator robust to heterogeneous treatment effects

Note: This figure reports the DiD estimate and 95% confidence intervals for two different estimators when running the effect of WBD Potential on test scores: Two-way fixed effects (denoted TWFE) and the group-time DiD estimator robust to heterogeneous treatment effects as discussed in and developed by [de Chaisemartin and D'Haultfoeuille \(2018\)](#) (denoted DHDC, where DCDH allows for dynamic effects). For this comparison, we re-frame our treatment as a binary indicator as in [Table F.8](#), such that a ward is treated during wave in year y if the share of stagnant water exceeds 5%. Here, we redefine WBD Potential into a dummy which is equal to 1 for wards in waves where the simulated stagnant water share exceeds 5%. Since the standard errors are bootstrapped for the DHDC estimator, we also provide a comparison to the TWFE with bootstrapped standard errors instead of clustered as in our main specification. Bootstrapped standard errors produced through 999 replications.

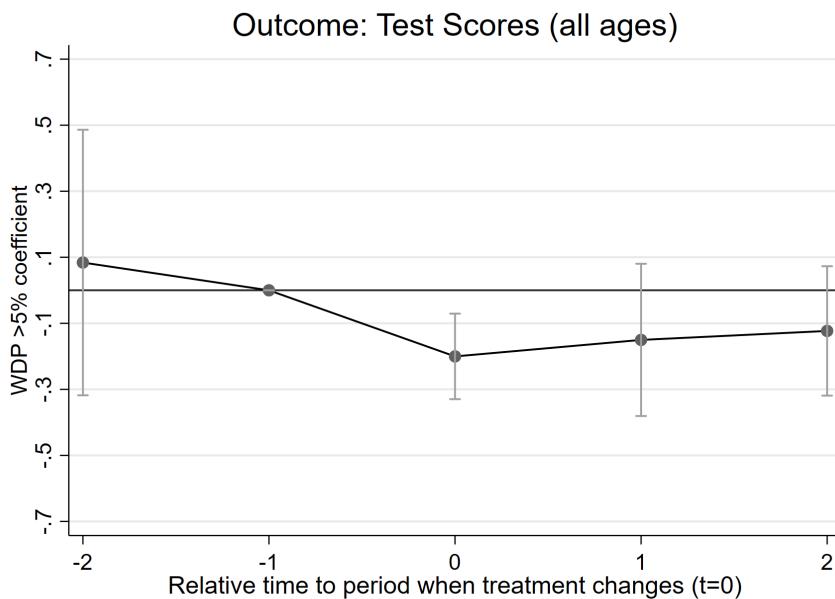


Figure F.6

Event study allowing for heterogeneous treatment effects

Note: This figure reports event study coefficients using the estimator by [de Chaisemartin and D'Haultfoeuille \(2022\)](#), where $t = 0$ indicates the first period when treatment changes. Here, we redefine WDP into a dummy equal to 1 for wards in waves where the simulated stagnant water share exceeds 5%.

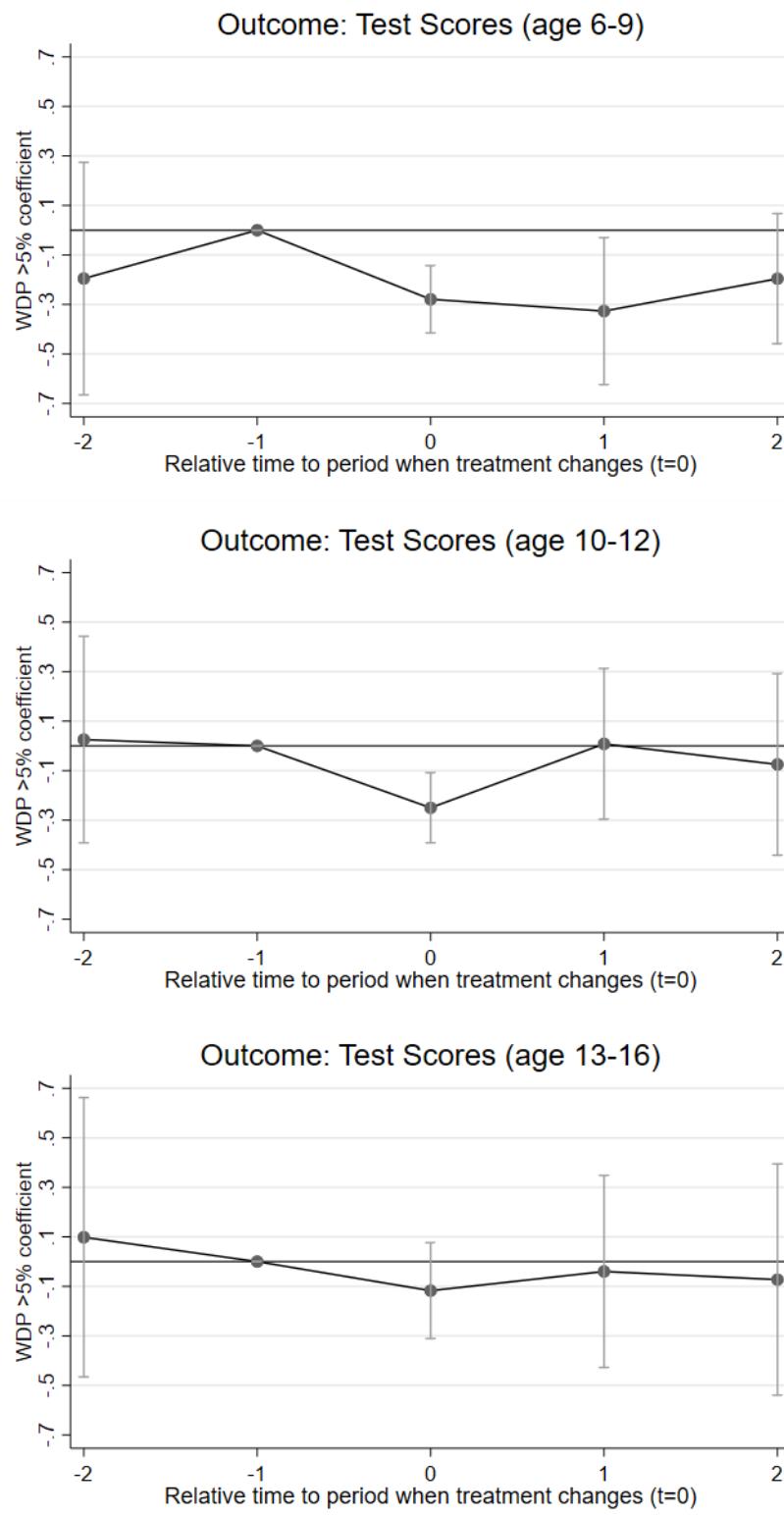


Figure F.7
Event study allowing for heterogeneous treatment effects by age group

Note: This figure reports event study coefficients split by age group using the estimator by [de Chaisemartin and D'Haultfoeuille \(2022\)](#), where $t = 0$ indicates the first period when treatment changes. Here, we redefine WDP into a dummy equal to 1 for wards in waves where the simulated stagnant water share exceeds 5%.

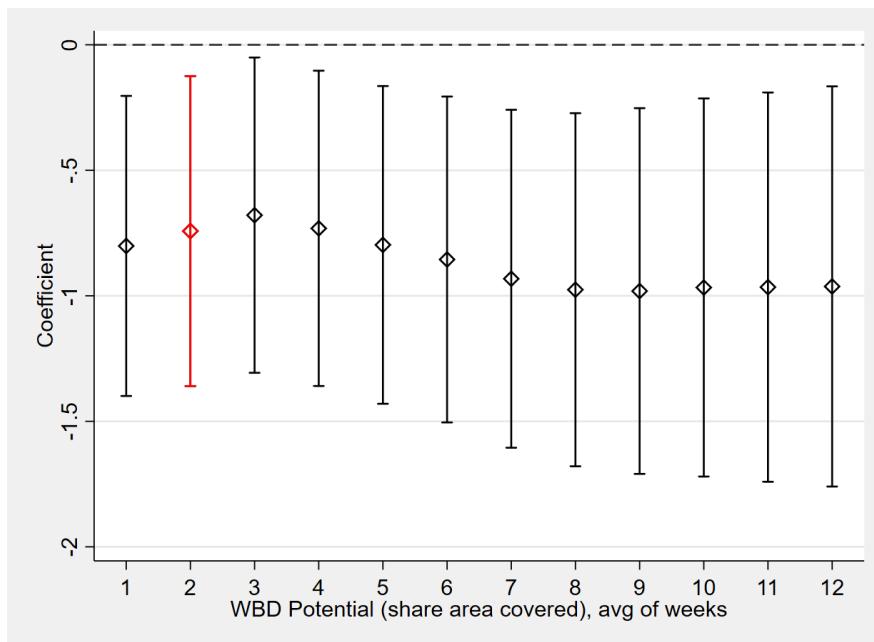


Figure F.8
Varying weeks in average measure of WDP

Note: This figure summarises DiD estimate of WBD Potential on test scores. Coefficient and 95% confidence intervals are displayed. Here, for each estimation we redefine the aggregation average of WBD Potential. In the first estimation, we define WBD Potential as the share of stagnant water in the one week preceding the date of survey when the child is tested. In the second estimate, we instead take the average share of stagnant water in the two weeks preceding the date of survey, and so on. The highlighted red estimate and confidence intervals reflect the definition we use in our main results, where we take the average of the eight weeks. In all estimations we include calendar month, wave and ward fixed effects as well as individual and household covariates. Standard errors are clustered on ward-level.

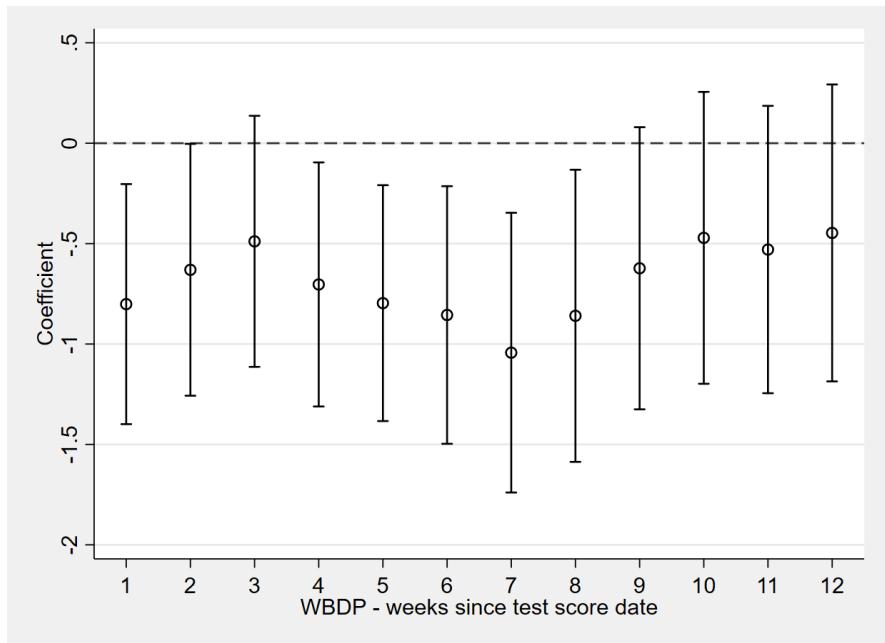


Figure F.9
WDP, by week discretely

Note: This figure summarises DiD estimate of WBD Potential on test scores. Coefficient and 95% confidence intervals are displayed. Here, for each estimation we redefine which week we include as the measure of WBD Potential. In the first estimation, we define WBD Potential as the share of stagnant water in the one week preceding the date of survey when the child is tested. In the second estimate, we instead take the share of stagnant water in the week which starts two weeks before the date of the survey and ends the week before the survey. Similarly, in the estimation labelled "3" we define WBD Potential as the share of stagnant water in (only) the week three weeks prior the date of survey. In all estimations we include calendar month, wave and ward fixed effects as well as individual and household covariates. Standard errors are clustered on ward-level.

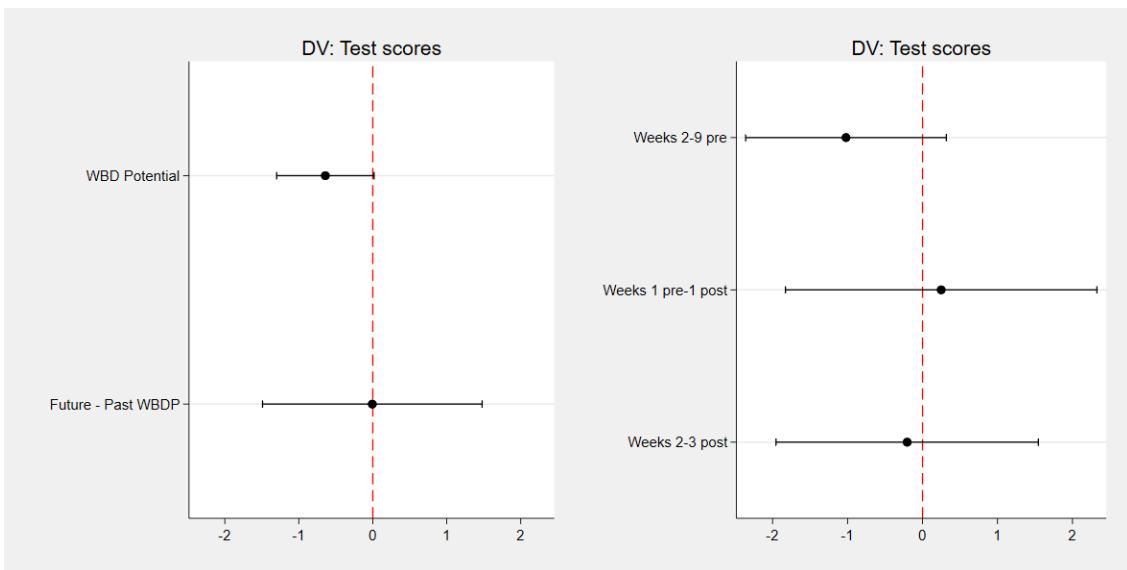


Figure F.10
Placebo: Effect of future WBD Potential

Note: This figure summarises DiD estimates of WBD Potential on test scores. Coefficient and 95% confidence intervals are displayed. Here, the two subplot represent one estimation each. On the left, we estimate with the main specification WBD Potential, and include the effect of the change in WBD Potential between the average stagnant water share *pre* survey, and the average stagnant water share *post* survey. On the right, we instead estimate longer-run means: Weeks 2-9 is the WBD Potential as the average 2-9 weeks prior to the survey. Weeks 1 pre- 1 post is the average WBD Potential the two weeks around the date of the survey, and the last estimate similarly is the average WBD Potential in the two weeks after the survey.

Table F.10
Effect of WBD Potential on test scores: Test of pretrends

	All periods, no early-treated	Waves 2011 and 2013, no early-treated
WDP $\geq 5\%$	-0.253*** (0.084)	
Later-treated \times Wave		0.005 (0.048)
Observations	342,801	180,900
Clusters	3,634	2,957

Note: The left column reports the results of the binary indicator for WDP using a cutoff of 5% estimated by excluding wards treated in 2011 or 2013. The right column reports the results of a test of pretrends for the same sample, but run only for the period 2011 to 2013, where the coefficient on the interaction term indicates the yearly change in test scores for the later-treated group relative to the never-treated group over the period 2011 to 2013. Standard errors in parentheses clustered on ward. * p<0.01 ** p<0.05 *** p<0.1. WBD Potential is the average share of ward covered in stagnant water in the eight weeks preceding the date of the survey. Ward is the treatment unit of observation. Wave is the year of the survey and waves included are 2011, 2013, 2014, 2015 and 2017. Month is the calendar month of treatment.

Table F.11
Effect of WBD Potential on test scores: Robustness to other contemporaneous shocks

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Dependent: Test score (std)								
WDP	-1.390*** (0.280)	-1.254*** (0.221)	-0.934*** (0.334)	-0.660** (0.324)	-0.647** (0.319)	-0.742** (0.315)	-0.791** (0.345)	-0.667** (0.335)	-1.040*** (0.351)
Obs.	368,446	368,446	368,444	368,444	368,444	368,444	368,444	368,444	368,444
Clusters	3,844	3,844	3,842	3,842	3,842	3,842	3,842	3,842	3,842
Covs	✓					✓	✓	✓	✓
Ward FE		✓		✓		✓	✓	✓	✓
Wave FE			✓		✓		✓	✓	✓
Month FE				✓	✓		✓	✓	✓
District \times Wave FE							✓		
High WBDP \times Wave FE								✓	
Ward-specific linear trends									✓

Note: Results is the effect of WBD Potential on test scores. Standard errors in parentheses clustered on ward. * p<0.01 ** p<0.05 *** p<0.1. WBD Potential is the average share of ward covered in stagnant water in the eight weeks preceding the date of the survey. Covariates include child's gender and age, and mother's age and secondary education, a wealth index, local past eight weeks of ward precipitation. Ward is the treatment unit of observation. Wave is the year of the survey and waves included are 2011, 2013, 2014, 2015 and 2017. Month is the calendar month of treatment. High WDP is an indicator for wards that at any time was exposed to more than 5% stagnant water.

should better predict the change in test scores than past events. However, due to the high correlation (95%) between weeks, models including weekly WBD Potential in the same estimation are likely to induce problems associated with multicollinearity. [Figure F.10](#) displays two attempts to disentangle the potential role of future WBD Potential while reducing the influence of multicollinearity. First, in the left subplot, we estimate a model with two variables: Our main WBD Potential measure (average eight weeks pre survey), and the difference between WBD Potential with that measure and the WBD Potential as measured two weeks after the test was taken. Thus, the coefficient would measure the marginal effect on test scores from the change in WBD Potential eight weeks prior to the survey and two weeks past. We find that the main measure has a negative effect on test

scores consistent with our main result, while the "Future minus Past WBD Potential" is measured with a coefficient close to zero and more imprecision. The right subplot Divides up WBD Potential into three time periods across a longer time span: WBD Potential 2-9 weeks prior to the survey, WBD Potential one week on either side of the week of the survey, and WBD Potential measured 2-3 weeks after the survey. As expected from our hypothesis on the mechanism and the results in [Figure F.9](#), we find that the WBD Potential 2-9 weeks prior to the survey is negative with a similar magnitude to that of our main results with the other two indicators centred closer to zero. However all three coefficients are statistically insignificant from zero.

G Mechanisms

G. I Absence

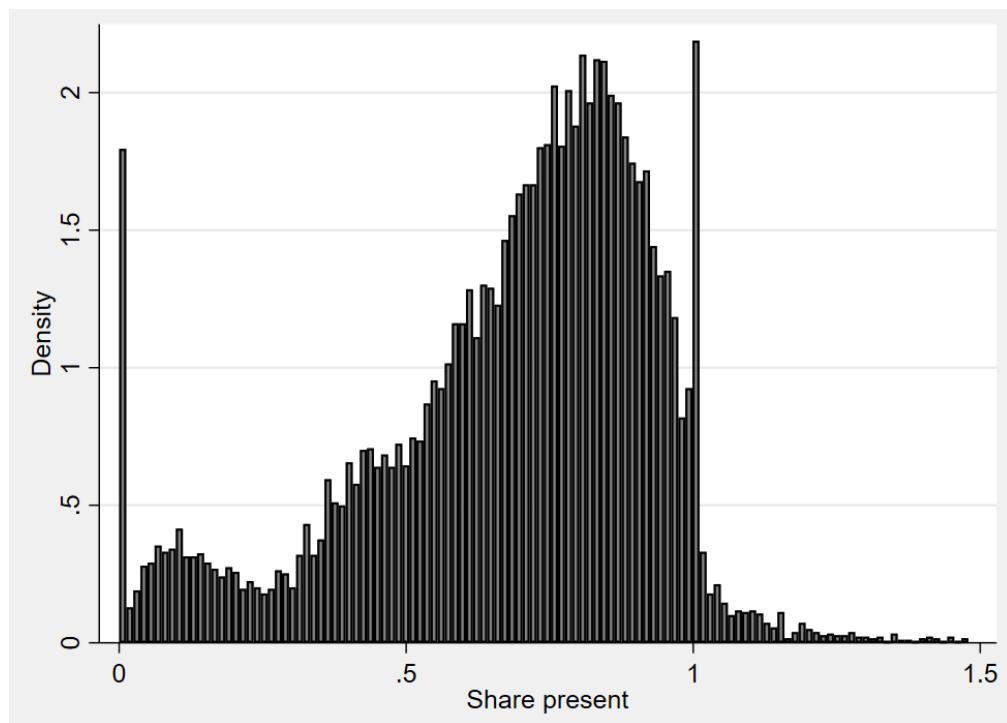


Figure G.1
Histogram on school presence

Note: This figure shows the histogram of school presence, which is defined as the ratio of children present in school to the number of children enrolled in school, for each school. Values of 0 or 1 and greater are likely due to measurement error.

G. II Mediation analysis

Table G.1
Mediation analysis of the effect of WDP on test scores

	Non-weighted			Weighted		
	(1)	(2)	(3)	(4)	(5)	(6)
WDP	-0.571 (0.445)	-0.564 (0.426)	-0.040 (0.446)	-1.003*** (0.375)	-0.992*** (0.361)	-0.639 (0.433)
Share absent		-0.127*** (0.028)	-0.110*** (0.029)		-0.137*** (0.030)	-0.126*** (0.031)
WDP * Share absent			-1.465** (0.716)			-0.939 (0.721)
Observations	12,108	12,108	12,108	12,108	12,108	12,108

Note: Standard errors in parentheses clustered on ward. * p<0.01 ** p<0.05 *** p<0.1. Wave, Calendar month, and Ward fixed effects included in all estimations.

H Impacts of climate change

H. I Climate and waterborne diseases

Climate change is expected to lead both to greater variation in rainfall and increased temperatures, which may lead to large increases in the incidence and distribution of waterborne diseases due to multiplicative effects ([Levy et al., 2018](#)). A study by [Traerup et al. \(2011\)](#) found that a one degree Celsius temperature increase in Tanzania alone leads to an increase in cholera risk alone by 15 to 29%, and this does take into account potential effects from a simultaneous increase in rainfall variability. Moreover, there is currently a lack of understanding how the combination of multiple weather events and the interaction effects between rainfall and temperature affect waterborne disease incidence ([Levy et al., 2018](#)). [Moore et al. \(2017\)](#), for instance, found that El Niño events, which lead to a simultaneous increase in rainfall and warm weather across East Africa, leads to a threefold increase in Cholera outbreaks.

H. II Climate change scenarios

Climate change impact analyses are typically based on so called climate change scenarios, which take into account projected future CO₂ concentrations and socioeconomic development, among other factors. In the IPCC Sixth Assessment Report by the UN Intergovernmental Panel on Climate Change (IPCC), climate change scenarios are represented by Shared Socio-economic Pathways (SSPs) ([International Panel of Climate Change, 2022](#)). The two scenarios most commonly used in impact analyses are the SSP2-4.5: “Middle of the Road” and SSP5-8.5: “Fossil-fueled Development”.

Each scenario assumes a trajectory taken in terms of sustainability measures. SSP2-4.5 assumes that greenhouse gas emissions remain at current levels as today until 2050, and then gradually fall towards 2100. This can be considered a relatively optimistic future scenario, though it may still lead to warming of up to about 2-3 degrees C relative to pre-industrial levels. The pessimistic scenario, which is commonly considered as an upper bound, is SSP5-8.5, which assumes very high greenhouse gas emissions, that triple by year 2075. This scenario may lead to rampant warming of up to almost 6 degrees C.

While our analysis is based on a continuous range of temperature increases instead of a fixed interval, we highlight in our results in [Figure 6](#) which temperature increase interval is associated with each scenario.

H. III The direct effects from temperature increases

Temperature is an important independent risk factor of waterborne diseases ([Levy et al., 2016](#)), and an essential condition for pathogen growth potential. To estimate the impact of a temperature increase on the effects of stagnant water, we use the results from a heterogeneity analysis where we split the data over +/- 1 degree C temperature windows. This captures any non-linearity in the

Table H.1
Climate change scenarios used as references for the impact analysis

Scenario	Description	Avg. Temp. (C) Δ Pre-industrial	Freq 50-yr rainfall events Δ Pre-industrial
SSP2-4.5	<i>Middle of the road</i>	1.8-3.3	1.5-2.5
SSP5-8.5	<i>Fossil-Fuelled Development</i>	3.3-5.8	2.5-5.3

Note: SSPs includes projections of population growth, economic output and income available for adaptation.

effect of temperature. We run this for the outcomes of diarrhea, test scores and school absence. The results of this analysis are reported in [Figure H.1](#).

While this analysis loses some power, we find suggestive evidence that effects increase with temperature consistent with the public health literature, and this trend is consistent across our three outcomes. Since diarrhea is the most salient outcome of the effects of waterborne disease, we use the heterogeneity results from these regressions as input to our impact analysis. Assuming a linear relationship, a regression run with the point estimates of the WBD coefficients for diarrhea gives a temperature slope coefficient of 0.18 with a p-value of 0.026. However, this does not take into account the uncertainty of each point estimate and may overstate the significance of this trend. To account for this, we instead run the trend regression weighted by the variance of each point estimate that takes into account the uncertainty of our temperature coefficients. This reduces the temperature slope coefficient to 0.11, with a p-value of 0.087. We consider this a more reliable and arguably more conservative estimate of the effects from temperature. In other words, this implies that a one degree C increase in temperature increases the WBD coefficient in the diarrhea regression by 0.11 units, which, relative to our baseline coefficient of 0.3, indicates about a 30% increase in the effect on diarrhea per degree of warming, depending on assumed total warming.⁴⁷ This compares favorably to [Traerup et al. \(2011\)](#) who found an increase in Cholera incidence of up to 29% per each degree C of temperature increase.

H. IV The indirect effects from increases in rainfall variability

Climate change is expected to lead to a greater increase in rainfall variability, including an increase in intense rainfall events. This is because warm air enables more vapor and a greater evapotranspiration rates, which are the fundamental drivers of high-intensity convective rainfall ([Pendergrass and Hartmann, 2014](#)). This is the primary type of rainfall that drives our variation in stagnant water: when rainfall intensity exceeds soil infiltration capacity, this leads to surface runoff, which accumulates as stagnant water in low-lying areas. Hence, one way to estimate the impact of an increase in rainfall on stagnant water is to analyze how the frequency of intense rainfall events are expected to change with a warmer climate.

⁴⁷To elaborate, one degree C of warming would increase the effect per degree C by 37%, while three degrees C of warming would increase the effect per degree C by 28%.

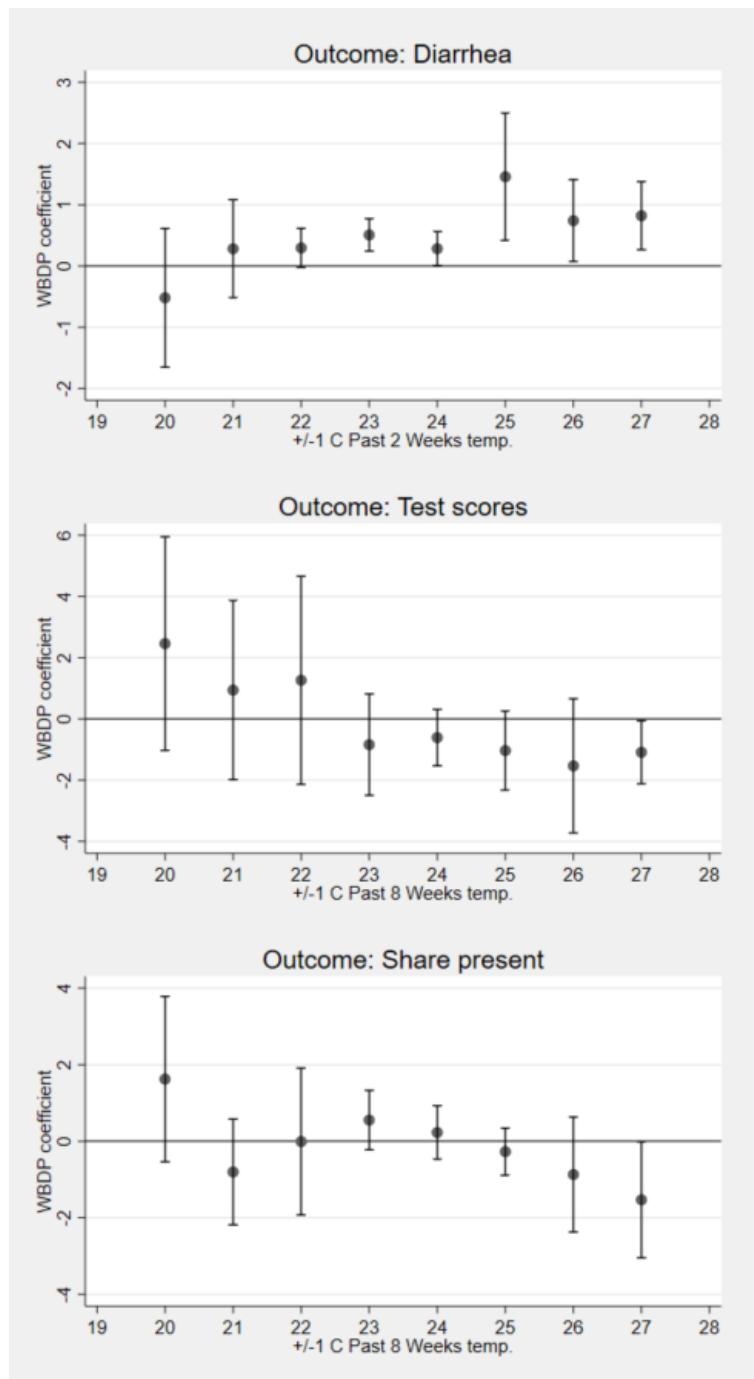


Figure H.1
Temperature heterogeneity

Note: This figure shows the effects of WDP on diarrhea, test scores and school presence, split by temperature levels in the past two weeks. Each point represents a regression run over a temperature window of +/- 1 degree Celsius. Coefficients and 95% confidence intervals are displayed.

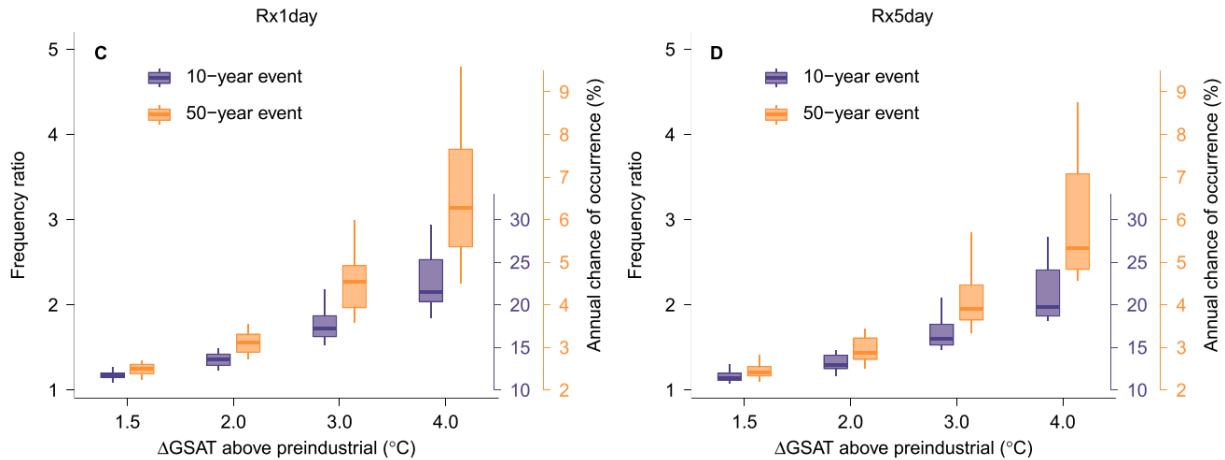


Figure H.2
Effects of global warming on the frequency of intense rainfall events

Note: From [Li et al. \(2021\)](#).

We use the latest projections from [Li et al. \(2021\)](#) from the latest CMIP6 Models⁴⁸ on how the frequency of rainfall events lasting 1 to 5 days with a current return period of 10 and 50 years⁴⁹ is expected to change with future temperature increases. [Figure H.2](#) shows these relationships for East Africa specifically, where Tanzania is located.⁵⁰ They find that the frequency increases more or less exponentially with warming, and more so for the more intense 50-year rainfall events, for durations over both 1 and 5 days. Given the similarity in these increases for both return periods and durations, and given that these types of rainfall events are what are driving large increases in stagnant water, we use the estimated relationship in [Figure H.2](#) to derive the effect of increases in rainfall variability on the baseline coefficient.

H. V Total effect

We then combine the direct effects from a temperature increase with the indirect effect temperature has on rainfall variability. The total burden of stagnant water can be considered as a function of the frequency with which exposure to stagnant water happens $F(T)$, which is a function of temperature T , and the risk of an outbreak happening conditional on exposure to stagnant water, i.e. the magnitude of our baseline coefficient $M(T)$, also a function of T . Hence, a back-of-the-envelope estimation of the combination of these effects is simply: $F(T) \times M(T)$, which captures the multiplicative effects of a simultaneous increase in frequency and magnitude.

[Figure H.3](#) shows the effect from each respective channel together with the total effect. The effect of temperature increases in a slightly more linear way than the effect of rainfall, but they are similar in magnitude over the temperature increases associated with the analyzed SSPs.

⁴⁸This is an ensemble of simulations of future climate from the latest climate models that build on the SSP scenarios.

⁴⁹The return period is defined as the inverse of the annual probability of the event, i.e. a 10-year event has an annual probability of 10%.

⁵⁰There heterogeneity between regions is relatively small, and we would thus expect to find a similar increase in other locations subject to stagnant water and waterborne diseases.

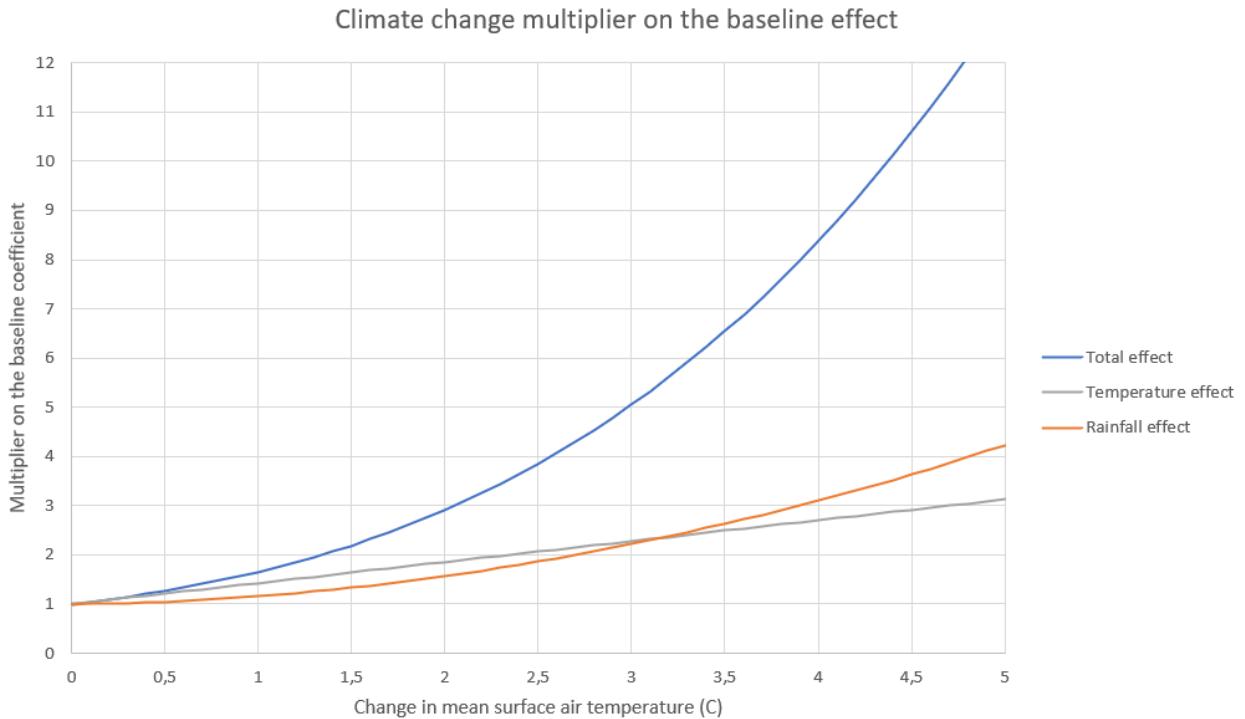


Figure H.3
Effects of global warming on the frequency of intense rainfall events

Note: This figure shows how the baseline effect of WDP on diarrhea increases with global warming through the separate effects on temperature and rainfall variability, together with the combined total effect. A value of 1 indicates that the baseline coefficient is unchanged.

We find that two degrees of warming leads to a threefold increase in the effect of stagnant water on waterborne disease outbreaks. This is identical in magnitude to the effect of El Niño events on Cholera in Tanzania, which are associated with simultaneous increases in rainfall and warm weather across East Africa ([Moore et al., 2017](#)).

H. VI Uncertainty and crucial assumptions

A complicating factor of climate change impact analysis is that society is expected to change across many dimensions other than just climate. By the end of this century, it is likely that developing countries such as Tanzania have seen an increase in economic growth and investments in public health and sanitation, factors that are likely to make the population more resilient to waterborne diseases. However, these developments are fundamentally uncertain, and we have thus abstracted away from them in the above analysis to arrive at the specific effects from climate change alone.

To get a better sense of how expected future conditions and ongoing trends may shape the waterborne diseases burden in the future, we summarize some of the relevant conditions in Table H.2, categorized by whether uncertainty is low, medium or high and whether it has a decreasing or increasing effect on the multiplier size. For example, economic growth is likely associated with urbanisation and population growth, and while economic growth itself may make households more resilient to disease through e.g. improved nutrition, population density itself, whether this is due to a greater overall population, or urbanisation, will likely make it easier for waterborne diseases

to spread. This is especially the case if urbanisation increasingly happens on marginal land that is at a higher risk of flooding and less connected to sewer networks that enable modern sanitation facilities.

The level of uncertainty is itself also of interest. For example, both climate scenarios and climate models are notoriously uncertain, and estimates for future temperatures differ widely depending on assumptions of future greenhouse gas emissions, and how the climate responds to these emissions. High uncertainty allows for small or insignificant changes in future climate, but in terms of social welfare, it likely only exacerbates social welfare. This is because, as [Figure H.3](#) shows, the effects of stagnant water on diarrhea is a *convex* function of temperature increases. The wider the temperature interval is, the greater is the expected cost from a temperature increase in terms of waterborne disease burden, which follows directly from Jensen's inequality. For example, while a scenario in which we know for certain that a future climate will be 2 degrees C warmer is expected to see a threefold increase in burden, a scenario in which we are equally likely to see warming over the span of 1 to 3 degrees C, the expected increase in the burden is about 3.4, more than 10% higher, and this only increases with our level of uncertainty.

In addition to uncertainty in future conditions that will affect how stagnant water affects waterborne diseases, an implicit assumption of our analysis is that there is no behavioral or physiological adaptation to an increase in waterborne disease risk. We believe this is still reasonable for two reasons. First, households today seem unaware of the risks associated with stagnant water, as discussed in [Section 7.1](#), and hence we do not expect an increase in future risks to affect future awareness, *ceteris paribus*. Secondly, while immunity is typically developed after having survived exposure to bacteria and viruses, waterborne pathogens also include protozoa and parasitic worms, which are harder for the immune system to resist. Moreover, immunity against common viruses that cause stomach illness only tends to last a few months, and the sheer number and variation of different waterborne diseases occurring means that it is unlikely that any single individual develops immunity to the majority of these diseases. Lastly, the results of our analysis are consistent with the effects seen from El Niño events which recur with regular intervals. If there were behavioral and immune responses to these events in areas prone to stagnant water, we would not be likely to see the large increase in waterborne diseases that these events typically lead to.

Table H.2
Factors affecting the future health burden of stagnant water, categorized by the direction of their impact and their estimated uncertainty.

		EXPECTED IMPACT ON MULTIPLIER SIZE		
		Decreasing	Neutral	Increasing
UNCERTAINTY	High	<ul style="list-style-type: none"> • Medical innovation 	<ul style="list-style-type: none"> • Behavioral adaptation 	<ul style="list-style-type: none"> • Temperature
	Medium	<ul style="list-style-type: none"> • Improved water and sanitation 		
		<ul style="list-style-type: none"> • Public health investments 		
	Low	<ul style="list-style-type: none"> • Economic growth 	<ul style="list-style-type: none"> • Immunity 	<ul style="list-style-type: none"> • Population growth
				<ul style="list-style-type: none"> • Urbanisation

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