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Terrorism and Child Mortality: Evidence from Africa

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Abstract

How does terrorism affect child mortality? We use geo-coded data on terrorism and highly spatially disaggregated data on child mortality to study the relationship between both variables for 52 African countries between 2000 and 2017 at the 0.5x0.5 degree grid-cell level. A two-way fixed-effects approach indicates that higher levels of terrorist activity correlate with higher levels of child mortality risk. Our estimates suggest that moderate increases in the terrorism index are linked to several thousand additional deaths of children under the age of five per year. Employing instrumental-variable and panel event-study approaches, we also provide causal evidence that terrorism increases the risk of death for children under the age of five. Effect sizes associated with these causal estimates are several times larger than those from the more conservative two-way fixed-effects approach. Finally, interrogating our data, we show that the direct effects of terrorism (e.g., in terms of its lethality and destruction of public health infrastructure) tend to be very small. This, in turn, suggests that increases in child mortality primarily emerge through the behavioral response of economic agents (e.g., parents, doctors, medical staff, aid workers and policymakers) to terrorism. Indeed, we provide evidence that higher levels of terrorist activity unfavorably correlate with several proximate causes of child mortality.

JEL Classification: D74, I10, I12

Keywords: terrorism; child mortality; Africa; instrumental-variable approach; panel event-study

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1. Introduction

The consequences of armed conflict on children's health are dire. Between 1995 and 2015, more than 10 million deaths of children younger than 5 years can be attributed to adverse effects of violent conflict (Bendavid et al. 2021). Beside its direct effects, child mortality may increase as a consequence of large-scale conflict because of the spread of infectious diseases (e.g., Iqbal and Zorn 2010; Charchuk et al. 2016), in utero exposure to conflict (Akbulut-Yuksel 2017; Dagnelie, Luca, and Maystadt 2018), the destruction of health infrastructure and the flight of health workers from conflict-ridden areas (e.g., McKay 1998; Sharara and Kanj 2014; Price and Bohara 2013; Chi et al. 2015; Chukwuma and Ekhator-Mobayode 2019), the destruction of sanitation, waste and water treatment (Kirschner and Finaret 2021), the underfunding of healthcare institutions by the government in times of conflict (Iqbal 2006; Gates et al. 2012) and reduced food supply (e.g., Lin 2020), which, in turn, is expected to correlate with weight loss and stunting (Bundervoet, Verwimp, and Akresh 2009; Kirschner and Finaret 2021; Dunn 2018; Wagner et al. 2018; 2019; Bendavid et al. 2021).[1]

In this paper, we focus on the effect of *terrorism* on child mortality.[2] Compared to large-scale armed conflicts (e.g., civil wars), the level of violence associated with terrorism is very low (Gaibulloev and Sandler 2019).[3] Terrorism is also distinct from large-scale violence in other ways (e.g., Sambanis 2008). For instance, terrorist groups may more strongly focus on civilian targets and do not control territory, meaning that terrorism may create much more diffused and punctuated threats rather than the objectively high threat levels affecting large areas that are common to armed civil conflicts and wars. This, in turn, may mean that the role of terrorism in child health and mortality is different from larger-scale armed conflicts that have been investigated – as shown above – in the past. For instance, compared to full-out civil war, the

[1] For a review of the consequences of armed conflict for child health and development, see Kadir et al. (2019).

[2] In this contribution, we follow the definition of Gaibulloev and Sandler (2019, 278) who define terrorism as "the premeditated use or threat to use violence against noncombatants by individuals or subnational groups to obtain a political objective through the intimidation of a large audience beyond that of the immediate victims".

[3] For instance, a common definition of armed civil conflict involves the use of force that results in at least 25 battle-related deaths per year in a specific country, while civil wars involve at least 1,000 battle-related deaths per country-year pair (Blattman and Miguel 2010). The magnitude of violence associated with terrorism is commonly smaller than this threshold (Gaibulloev and Sandler 2019).

direct destruction of health infrastructure stemming from terrorist attacks usually ought to be limited, given that the overall level of violence is much smaller. Instead, indirect effects stemming from behavioral responses to terrorism may be more relevant.

There is some evidence concerning the adverse effect of terrorism on child health. For example, several empirical studies find that in utero exposure of fetuses to terrorist attacks is associated with lower infant weight, an effect likely driven by maternal stress and poor nutrition (e.g., Lauderdale 2006; Camacho 2008; Mansour and Rees 2012; Quintana-Domeque and Ródenas-Serrano 2017). However, and compared to large-scale armed conflicts, there is little evidence concerning the role of terrorism in child mortality. For instance, in their systematic of the effects of armed conflict on child health and development, Kadir et al. (2019, 4) explicitly do not consider terrorism because "terrorist incidents are not universally associated with armed conflict". In other words, the potentially adverse effect of terrorism on child mortality has so far been largely neglected because terrorism is distinct from other forms of conflict (see also Sambanis 2008; Gaibulloev and Sandler 2019).

At the same time, however, terrorism has become more common and deadly after 2001 (Gaibulloev and Sandler 2019), while large-scale conflicts have become less common since the 1990s (Blattman and Miguel 2010). That is, terrorism has become a more common form of violence, suggesting that its relevancy for the patterns of child mortality may have similarly grown.

This is especially true for Africa, a continent both plagued by comparatively high levels of child mortality and – at least since the 2000s – terrorist activity (Gaibulloev and Sandler 2019). We study the role of terrorism in child mortality for a sample of 52 African countries between 2000 and 2017. We combine geo-coded data on terrorism with highly spatially disaggregated data on child mortality and morbidity at the 0.5x0.5 degree grid-cell level. This allows us to track child mortality and its likely causes on a sub-national level. Our study complements a small number of case-studies on the consequences of terrorism for child health in Africa. For Burkina Faso and Nigeria, this evidence indicates that terrorism impedes access to perinatal healthcare (Chukwuma and Ekhator-Mobayode 2019; Druetz et al. 2020). In Cameroon, the Boko Haram insurgency is associated with lower height-to-weight ratios in children under five, likely caused by infectious diseases and underutilization of health services (Kaila, Nawo, and Son 2021; 2021).

We contribute to the literature in several ways. First, by using high-resolution data for a much larger geographical area – much of Sub-Saharan Africa and part of Northern Africa – and a longer time span (2000–2017), we provide systematic evidence on the terrorism-child mortality nexus in this part of the world. Second, unlike studies that either focus on wartime violence or lump together several different types of violence, we specifically focus on terrorism. As discussed above, this focus is worthwhile given the potentially substantial differences between terrorism and other forms of (larger scale) armed conflict. Third, our rich and fine-grained data makes it possible to apply a variety of econometric methods to derive convincingly identified causal estimates. Fourth, we study several potential pathways from terrorism to child mortality. Finally, by highlighting the – previously mostly neglected – role of terrorism in child health and mortality, we also add to the larger empirical literature on the consequences of terrorism for socio-economic variables such as economic growth, poverty and life satisfaction (e.g. Tavares 2004; Frey, Luechinger, and Stutzer 2007; Gaibulloev and Sandler 2011; Meierrieks and Gries 2013; for an overview see Gaibulloev and Sandler 2019).

Employing a two-way fixed-effects approach, we show that higher levels of terrorist activity – operationalized as a terrorism index that accounts for both the frequency and intensity of terrorism – is associated with higher levels of child mortality risk. Using instrumental-variable and panel event-study approaches, we come to the same conclusion. The latter two approaches allow us to interpret this finding in a causal way. Estimated effects are also economically substantive. Even our most conservative estimates suggest that moderate increases in the terrorism index are linked to several thousand additional deaths of children under the age of five per year. Moreover, empirical approaches that account for endogeneity point to economic effects that are several times larger. Interrogating our data, we show that the direct effects of terrorism (e.g., in terms of its lethality and destruction of public health infrastructure) tend to be very small. This, in turn, suggests that increases in child mortality primarily emerge through the behavioral response of economic agents (e.g., parents, doctors, medical staff, aid workers and policymakers) to terrorism. Indeed, we provide evidence that higher levels of terrorist activity unfavorably correlate with several proximate causes of child mortality (the incidence of malaria and diarrhea, vaccination rates and malnourishment).

The rest of this paper is organized as follows. Section 2 discusses our theoretical framework and develops a testable hypothesis. We introduce and discuss the data on child mortality and terrorism in Section 3. In Sections 4 to 6, we discuss several empirical approaches to the terrorism-child mortality nexus and present our related empirical results. In Section 7, we explore potential transmission channels from terrorism to child health. Finally, Section 8 concludes.

2. Theoretical Framework

In this section, we discuss the theoretical linkages between child mortality and terrorism, considering the direct and indirect consequences of terrorism for children's health. We conclude by introducing a testable hypothesis.

2.1 Direct Effects

Most obviously, terrorism can adversely affect child mortality when children are killed in a terrorist attack. What is more, children may be wounded in a terrorist attack in ways that are eventually lethal. Similarly, terrorism may kill or incapacitate the children's parents, doctors and other medical personnel or foreign aid workers. This, in turn, may also contribute to child mortality by denying children parental or medical care. Finally, terrorism may destroy public health infrastructure (e.g., by destroying hospitals), which would likewise have direct adverse consequences for children's health.

Still, while the direct effects of terrorism through the destruction of human life and the health infrastructure are eminently plausible, we do not expect them to affect child mortality in noticeable ways. This is because terrorism in general does not produce many victims, especially in comparison to many other sources of death. For instance, Arce (2019) estimates terrorism to lie in the bottom nine percent of the global burden of disease. That is, its burden is similar to that of Dengue fever, Vitamin A deficiency and Hodgkin's disease (Arce 2019, 390). Similarly, it is much more likely to be a victim of homicide than terrorism. For instance, for the early 2010s, Kamprad and Liem (2021) report that there roughly half a million deaths per year from homicide (implying a homicide rate of 6.2 per 100,000 individuals), while terrorism accounted for approximately 38,000 deaths per year (meaning a terrorism casualty rate of approximately 0.5 per 100,000 individuals).

2.2 Indirect Effects

It is more probable that the adverse consequences of terrorism for child mortality are due to terrorism's indirect effects. These indirect effects emerge from the behavioral response of a variety of economic agents (parents, especially mothers; doctors and other healthcare workers; the government) to terrorism. This response, in turn, affects both the demand for and supply of children's healthcare in ways that increase the risk of child mortality.

The parental perspective concerns the demand for children's healthcare. Here, we expect parents to be intimidated by terrorism. Indeed, the production of fear and intimidation for political leverage is a major goal of terrorist organizations (e.g., Gaibulloev and Sandler 2019). What is more, Sunstein (2003) stresses that role of *probability neglect*, where individuals focus on a bad outcome (in our case, being harmed by a terrorist attack) but do not consider that this outcome is very unlikely to occur (as terrorism is very rare). Sunstein (2003) also argues that this the probability of harm is especially likely to be neglected when people's emotions (e.g., when their children's lives are threatened) are activated. The interplay between fear and probability neglect is consequently expected to lead to a behavioral response to terrorism on the part of affected parents that is potentially excessive. For instance, parents may forego preventive care (e.g., vaccinations or regular check-ups) out of fear that their children will be victimized by terrorism – a behavior that is expected to eventually contribute to higher levels of child mortality (cp. Druetz et al. 2020). That is, the interaction between between fear of terrorism and probability neglect may lead parents to weigh the risk of their child being harmed by terrorism more strongly than the child's risk of being affected by infectious diseases or other preventable causes of harm – even though this is not warranted given the actual probabilities of suffering harm involved.

An excessive behavioral response due to fear of terrorism may also affect the supply of children's healthcare. Concretely, doctors, medical staff and international aid workers may stay at home rather than go to work, which could contribute to adverse consequences for children's health. At the same time, however, one could argue that the role of fear as a driving force of behavioral change in response to terrorism is less relevant for medical and aid workers. For instance, their professional experience may make them less vulnerable to emotional shocks due to a confrontation with fear and death. Also, they may feel less strong about children than parents, meaning that strong emotions are less likely to cloud their risk perception.

However, we can still hypothesize about behavioral responses to terrorism by medical and aid workers by considering a rational-choice perspective. Indeed, a number of theoretical contributions apply this perspective to provide an economic analysis of terrorism (e.g., Eckstein and Tsiddon 2004; Sandler and Enders 2004; Naor 2006; Becker and Rubinstein 2011; Schneider et al. 2015). The rational-choice approach posits that individuals are utility-maximizers, and that terrorism affects the individual utility-maximization process by influencing the costs and benefits associated with certain activities, potentially effecting behavioral changes when new utility-maximizing choices emerge. For one, terrorism is expected to reduce the benefits associated with certain choices of action of doctors, medical staff and international aid workers. For instance, it may reduce the benefits of work when parents do not come to see doctor and thus do not pay for the doctor's services. There are also the obvious disbenefits of stress and dissatisfaction that emerge from the prospect of an untimely death due to terrorism (e.g., Naor 2006; Becker and Rubinstein 2011). For another, terrorism may also impose additional costs on medical and aid workers, e.g., by necessitating additional investment into personal security. Ceteris paribus, the advent of terrorism is thus expected to make activities that may involve encountering terrorism (e.g., providing aid to or working and making patient visits in terror-ridden areas) less attractive. Instead, doctors, medical staff and international aid workers are expected to opt for those activities that avoid terrorism as those activities are now more likely to maximize utility. For instance, this utility-maximization process may involve aid workers withdrawing from certain areas, medical workers taking up employment in alternative fields or medical personnel migrating away from terror-affected areas (cp. Price and Bohara 2013; Chi et al. 2015; Chukwuma and Ekhator-Mobayode 2019).[4] Such behavioral adjustments that follow from utility-maximization considerations will eventually also result in poorer supply of child healthcare and higher levels of child mortality.

Finally, terrorism may adversely affect the supply of healthcare by influencing public spending decisions. Here, the threat of terrorism is expected to make it more likely that more public resources are spent on security (e.g., Gupta et al. 2004; Iqbal 2006; Gates et al. 2012; Cevik and Ricco 2020). We can explain this shift in spending by political considerations, where policymakers offer voters (who are intimated by terrorism) security spending as solution to the terrorist threat; by satisfying public demand for security in this manner, policymakers hope to maximize public and voter support. However, this shift in spending may come at the expense of public health expenditure, especially when resources – as it is often the case in Africa – are scarce in the first place. For example, there may be fewer resources available for clinics, doctors, prevention and vaccination programs or health education as a consequence of terrorism-induced cuts in health spending. This lack of funding, in turn, is expected to adversely affect child health.

2.3 Main Hypothesis

In line with our discussion, below we test the following main hypothesis:

Higher levels of terrorist activity result in a higher risk of child mortality.

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[4] For an empirical analysis of the migratory response to terrorism among the highly skilled, see, e.g., Dreher et al. (2011).

In detail, terrorism may adversely affect children's health through the destruction of human life and the health infrastructure (*direct effects*) and the response of economic agents to terrorism that is disproportionate to the terrorist threat and undermines the adequate demand for and supply of children's healthcare (*indirect effects*). The behavioral response to terrorism, in turn, may be rooted in psychological (fear and probability neglect), rational-choice (utility maximization) and political (public support and vote maximization) mechanisms. Given that terrorism's destructiveness tends to be comparatively low, we expect the various indirect consequences of terrorism to be the main reason for the hypothesized detrimental effects of terrorism on children's health.

3. Data

To test our main hypothesis, we use sub-national data aggregated at the 0.5x0.5° (around 55km^2 at the equator) grid-year level using the *PRIO-GRID* (Tollefsen et al. 2012) for a maximum of 52 African countries and territories for the 2000–2017 period.[5] The summary statistics for all variables employed in our analysis are reported in Supplementary Table 1. Below, we discuss in more detail our main variables of interest.

3.1 Measuring Child Health Outcomes

Our main outcome of interest is *child mortality*, measured as the probability for a given child to die before reaching the age of five. The data comes from Burstein et al. (2019) who provide high-resolution (5km^2) estimates for low- and middle-income countries covering the whole of mainland Africa and Madagascar for the 2000-2017 period. Their geospatial estimates are derived from the collection of available *Demographic and Health Surveys*, *UNICEF Multiple Indicator Cluster Surveys* and other country-specific surveys. We aggregate their data to the PRIO-Grid level. As part of our robustness checks, we also use two alternative child mortality measures, *neo-natal mortality* (i.e., the risk of death for a new-born in the first 28 days after

[5] These countries and territories are Algeria, Angola, Benin, Botswana, Burkina Faso, Burundi, Cameroon, Central African Republic, Chad, Comoros, Congo, Côte d'Ivoire, Democratic Republic of the Congo, Djibouti, Egypt, Equatorial Guinea, Eritrea, eSwatini, Ethiopia, Gabon, Gambia, Ghana, Guinea, Guinea-Bissau, Kenya, Lesotho, Liberia, Libya, Madagascar, Malawi, Mali, Mauritania, Morocco, Mozambique, Namibia, Niger, Nigeria, Rwanda, South Sudan, Senegal, Sierra Leone, Somalia, Somaliland, South Africa, Sudan, Tanzania, Togo, Tunisia, Uganda, Western Sahara, Zambia and Zimbabwe.

birth) and *infant mortality* (i.e., the mortality risk under the age of one), also from Burstein et al. (2019).

As shown in Figure 1, regardless of which indicator we choose, mortality rates generally saw a noticeable decline over our period of observation. For instance, the risk of mortality for children under the age of five was on average 12.8 percent in the year 2000 but fell to approximately seven percent in the year 2017. For our subsequent analysis, this implies that estimating the effect of terrorism on mortality outcomes primarily means assessing whether terrorism produced conspicuous setbacks from the general downward trend in mortality.

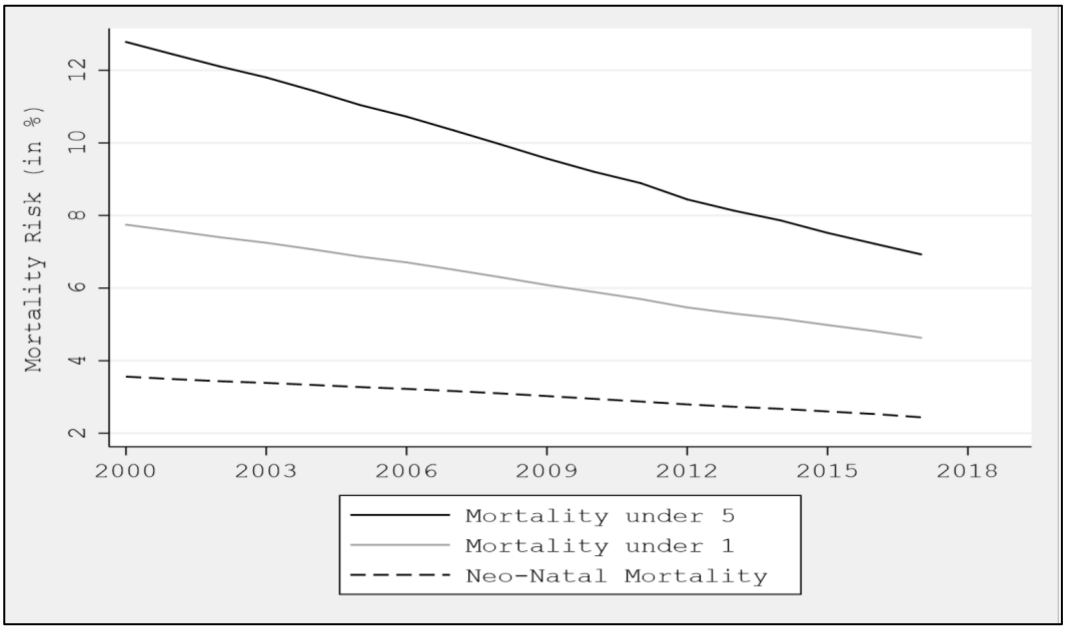

Figure 1: In-Sample Mean-Mortality Rates, 2000-2017

3.2 Measuring Terrorist Activity

Our main independent variable is an *index of terrorist activity*. It is defined as the sum of the *per capita number of terrorist attacks* and the *per capita number of terrorism casualties* per grid–year observation. The term "terrorism casualties" refers to the number of individuals that are killed in a terrorist attack. The index reflects both the frequency (number of terrorist attacks) and ferocity (number of casualties) of terrorism, and we follow Eckstein and Tsiddon (2004) and others in constructing it. In order to reduce the influence of outliers, we apply the inverse hyperbolic sine transformation to our terrorism index.[6] In weighing our terrorism variable by population size, we follow, e.g., Jetter and Stadelmann (2019). They suggest that per capita

[6] In contrast to the log trans-formation, the inverse hyperbolic sine transformation is also defined for grid-year observations with no terrorist activity (e.g., Burbidge, Magee, and Robb 1988).

measures of terrorism are more reflective of the (individual) risk associated with terrorism. As stressed above, we argue that is it is this very terrorism risk that explains how terrorism may (indirectly) affect child mortality. Similar population-adjusted terrorism indicators are used in, for instance, Tavares (2004), Gaibulloev and Sandler (2011) and Meierrieks and Gries (2013).

The data on grid-level population size (used to calculate per capita rates) come from the *LandScan* high-resolution global population data set (Bright et al. 2018). The terrorism data are drawn from the *Global Terrorism Database* (*GTD*). The GTD was first described in LaFree and Dugan (2007). It collects information on terrorist activity from reputable media outlets.[7] For a terrorist event to be recorded in the GTD, it must be documented by at least one high-quality media source (e.g., a renowned international newspaper such as the *New York Times*). To be considered a terrorist event, it must also (1) be intentional, (2) entail some level of violence or threat of violence and (3) be committed by non-state actors, meaning that violence by state actors is excluded (LaFree and Dugan 2007). Furthermore, it must meet at least two of the following three criteria: (1) the incident must be carried out to achieve a political, economic, religious or social goal, (2) there must be evidence of an intention to coerce, intimidate or convey some other message to a larger audience than the immediate victims and/or (3) the incident must be outside the context of conventional warfare (LaFree and Dugan 2007). The GTD provides geolocational information (latitude and longitude) on 99 percent of all attacks reported. This allows us to easily combine the terrorism index with our public health data.

To add to the robustness of our findings, below we also use the constituents of our terrorism index as explanatory variables, i.e., (1) the (inverse hyperbolic sine transformed) per capita number of terrorist incidents and (2) the (inverse hyperbolic sine transformed) per capita number of terrorism casualties per grid-year observation. This is to assess whether any effect of terrorism on child mortality is due to the frequency or ferocity of terrorism. Note, however, that the casualty variable is likely subject to under-counting as terrorism victim figures are unknown for many observations in the GTD.

Figure 2 visualizes the temporal trends in terrorism (indicated by annual mean of the terrorism index) in Africa over our period of observation. There is a clear uptick in terrorist activity after 2011. To illustrate, while there were, on average, approximately 310 terrorist attacks in all African countries between 2000 and 2011, the annual average was almost 2,350 attacks from 2012 onwards. This increase can partly be attributed to stronger Islamist terrorist activity in the

[7] The GTD can be accessed at https://www.start.umd.edu/gtd/.

2010s, e.g., to attacks by *Al-Qaida in the Islamic Maghreb* in Algeria and Mali as well as *Boko Haram* in Nigeria, Niger, and Chad. Furthermore, terrorism in Africa is linked to violent separatism such as in Ethiopia (e.g., by the *Oromo Liberation Front*) and Angola (e.g., by the *Front for the Liberation of the Enclave of Cabinda*).

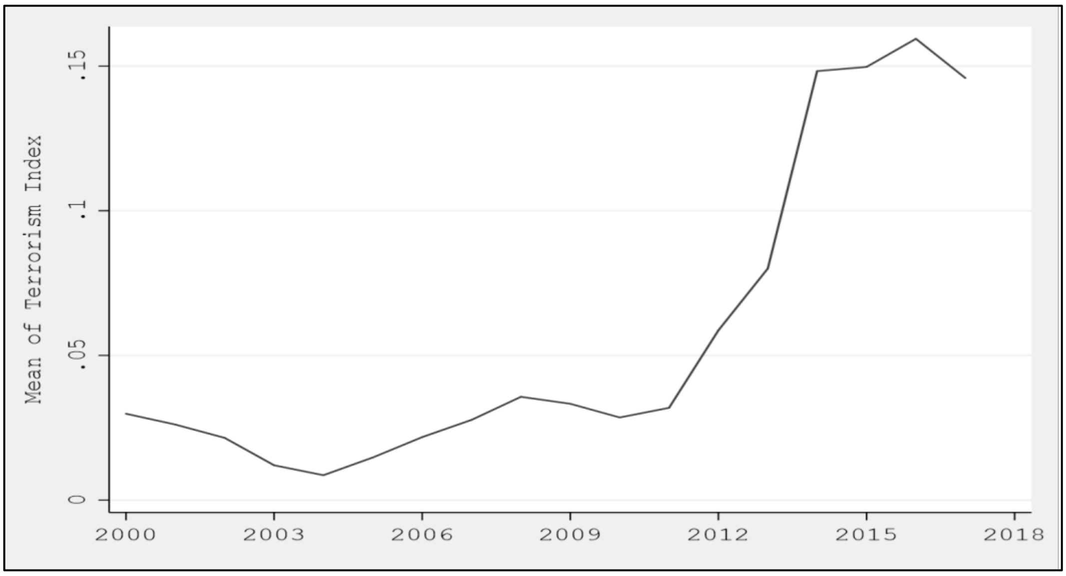

Figure 2: Terrorism in Africa, 2000-2017

3.3 Geography of Child Mortality and Terrorism

We illustrate the geographical distribution of both child mortality and terrorism in Figure 3. Concerning the patterns of child mortality, the strength of the red shading indicates the severity of child mortality, measured as the probability of a child to die before reaching the age of five averaged over the 2000-2017 period and aggregated at the 0.5x0.5 degree PRIO-grid-cell level. Figure 3 shows that child mortality rates are much higher in the Sahel and Central Africa compared to Southern and Northern Africa. Concerning terrorism, black markers indicate the location of individual terrorist attacks between 2000 and 2017. Some countries (e.g., the Comoros, Lesotho and Morocco) were almost completely unaffected by terrorist attacks, while other countries (e.g., Algeria, Egypt and Nigeria) saw substantial terrorist activity. Finally, Figure 3 also points to a strong intra-country heterogeneity both with respect to child mortality and terrorism. For instance, consider Nigeria, where the northern and south-eastern parts of the country saw both markedly higher child mortality and more terrorist attacks than the rest of the country.

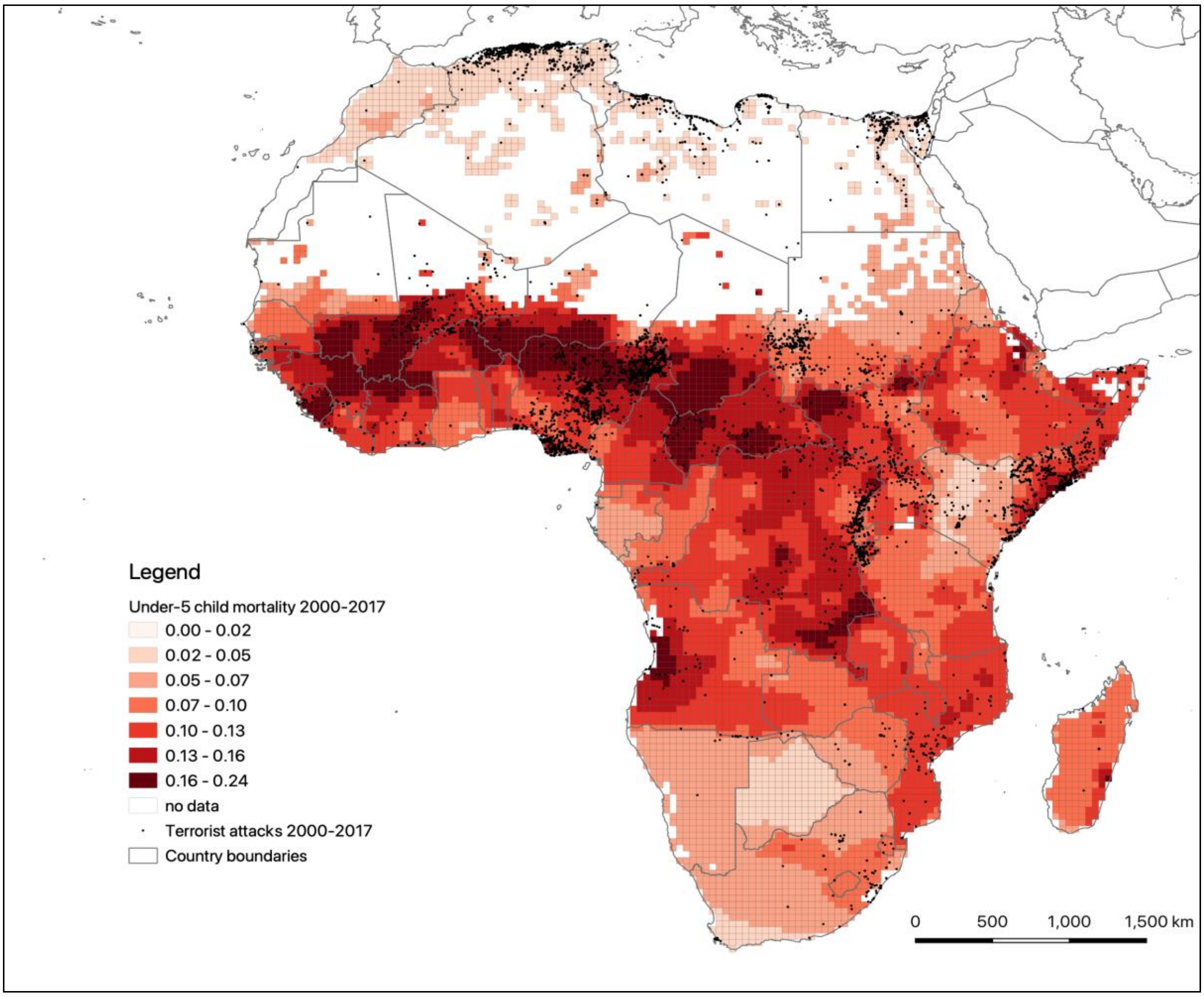

Figure 3: Geography of Terrorism and Child Mortality

4. Two-Way Fixed-Effects Approach

4.1 Empirical Model

To study the relationship between children's health and terrorist activity, we first consider the following two-way fixed-effects model, which we estimate using the OLS-estimator:

$$health_{k,it} = \beta_1 * terror_{j,it} + \beta_2 * X_{it} + \alpha_i + \lambda_t + v_{it} \quad (1)$$

Here, *health* refers to our *k*-th measure of children's heath in grid *i* and year *t*. Usually, this is the risk of child mortality for children under the age of five, but it may also indicate the neo-natal or infant mortality risk. The variable *terror* refers to our *j*-th measure of terrorist activity. Commonly, we employ our terrorism index but as a robustness check we also use two alternative terrorism indicators (the per capita number of terrorist attacks and the per capita number of terrorism casualties).

Fixed-Effects. We also include grid-specific fixed-effects (α) to control for the role of time-invariant factors that may confound the relationship between terrorism and children's health. For instance, local geographical conditions (e.g., proximity to mosquito habitats) may be conducive to the spread of malaria, which, in turn, is expected to adversely affect children's health. At the same time, certain geographical conditions (e.g., proximity to the jungle or rugged terrain) may provide potential militants with safe havens and thus facilitate terrorist activity. Similarly, year-fixed effects (λ) control for the influence of global trends and events that may have affected terrorism and public health. For instance, the introduction or diffusion of medical technology during our period of observation is expected to have reduced child mortality. At the same time, global trends in terrorism (e.g., the rise of *Al-Qaida* and the *Islamic State*) also ought to have influenced terrorism in Africa.

Controls. Finally, we account for a set of confounders (X) that may affect both public health and terrorist activity and thus also obfuscate – due to omitted variable bias – the relationship between terrorism and child mortality. Because data on the controls is not available for all grids and years, our sample size is reduced when including them. That is, when considering only the role of terrorism (plus the fixed effects) in child mortality in a parsimonious model, our sample covers 7,954 grids for the 2000-2017 period. A model that also considers the role of the various confounders allows us to consider 6,751 grids for the 2000-2015 period.

In detail, the confounders are nightlights, travel time to the nearest city, urbanization, female education, and climate conditions. First, we consider the influence of *nightlights*. This variable captures light emissions during nighttime. The data, originally recorded by the *U.S. Air Force Defense Meteorological Satellite Program*, was made available for research by Elvidge et al. (1997; 2021) and extended by Ghosh et al. (2021). Nightlights are used as a measure of economic development. We expect richer grids to see lower levels of child mortality (e.g., by virtue of better access to medical technology); at the same time, economic development may also affect terrorism, e.g., by influencing its opportunity costs (e.g., Freytag et al. 2011). Second, we control for *travel time to the nearest large city*. The data are from Müller-Crepon (2021). This variable indicates the quality of local infrastructure and is, consequently, expected to negatively affect children's health outcomes (e.g., as it negatively correlates with access to the public health infrastructure), while also mattering to terrorism (e.g., by affecting the speed with which counter-terrorism measures can be undertaken). Third, we control for *urban population*, using data from Meiyappan and Jain (2012). For one, urbanization is expected to correlate with health outcomes, e.g., by affecting the spread of communicable diseases and determining access to food, drinking water and medical treatment (e.g., Eckert and Kohler 2014). At the same time, urbanization may also affect terrorism, e.g., by facilitating terrorist access to the media and providing terrorists with more and closer targets (e.g., Ross 1993). Fourth, we control for *female education*, measured as the years of education of 20-24 y old women. The data are from Graetz et al. (2018). It is well-known that especially the education of mothers predicts child health, where better female education ought to correlate with better outcomes (e.g., Subbarao and Raney 1995; Hahn, Nuzhat, and Yang 2018). There are also arguments that education may affect terrorism. For instance, higher levels of education will usually result in higher wages, which will make it less attractive for individuals to engage in terrorism (Brockhoff, Krieger, and Meierrieks 2015). Finally, we consider the potentially confounding influence of climate variables: *temperature* (in °C) and *precipitation* (measured by the standardized precipitation evapotranspiration index), where the data are from Fan and van den Dool (2008) and Peng et al. (2019). For one, climatic conditions may affect health outcomes, e.g., by facilitating the spread of diseases vectors (e.g., mosquitos) or determining access to food and water (e.g., Sheffield and Landrigan 2011; Meierrieks 2021). For another, climate conditions may also affect terrorism; for example, higher temperatures tend to be associated with aggression and violence in humans (e.g., Craig, Overbeek, and Niedbala 2021).

4.2 Empirical Results

We report our findings in Table 1. We find that more terrorist activity is associated with higher levels of child mortality. Considering economic substantiveness, our baseline estimates (Model (3), Table 1) suggest that an increase in terrorism by ten percent is associated with an expected increase in child mortality of approximately 0.01 percentage points. To give a comparison, an increase in economic development (nightlights) by ten percent will yield a decrease in child mortality of approximately 0.04 percentage points.

Our main finding of an unfavorable association between terrorism and child mortality is robust to the inclusion of the baseline controls. The coefficients for the controls are as expected. For example, we find that higher levels of economic development (as indicated by nightlights) and female education to correlate with better health outcomes.

Our main finding is also robust to different measurements of mortality and when we focus on the frequency or ferocity of terrorism only. Concerning the latter analysis, the association between the frequency of terrorism and child mortality appears to be somewhat stronger than the association between the lethality of terrorism and the child mortality risk. For instance, this may be due to the fact that the ferocity of terrorism is more strongly clouded by uncertainty and underreporting, thus making behavioral responses to it by affected economic agents less straightforward.

Finally, allowing for a more complex lag structure with respect to the correlation between terrorism and the risk of child mortality suggests that this association becomes somewhat stronger over time.[8] This may correspond to behavioral changes in response to terrorism that take some time to materialize. For example, this may pertain to a migratory response to terrorism on the part of medical workers, which, in turn, will only impact child mortality in subsequent years.

—Table 1 here—

4.3 Robustness Checks

We probe whether our finding of a positive correlation between terrorism and child mortality is robust in a number of ways. These checks concern (1) different operationalizations of the terrorism variable, (2) changes to the set of confounders (e.g., with respect to their measurement), (3) the inverse hyperbolic sine transformation of our main dependent variable and (4) the use of data that is averaged over four years. More information on these robustness checks is provided in Supplementary Table 2, which also reports our regression estimates. All checks confirm the unfavorable association between terrorism and child mortality risk.

5. Instrumental-Variable Approach

5.1 Empirical Model

The two-way fixed-effects estimates are potentially subject to endogeneity. Endogeneity may have three sources. First, *omitted variable bias* may play a role. However, this seems unlikely, given that we include an extensive set of control variable and include grid-level fixed effects. Consequently, our two-way fixed-effects regressions yield very high coefficients of determination, leaving little variation left to be explained by additional confounders. Second, endogeneity may be due to *measurement error in terrorism*. Indeed, as the GTD employs data from public sources, reporting biases are possible. For example, terrorist activity in certain regions or countries may be systematically underreported by the (international) press when such

[8] Because allowing for a more complex lag structure reduces our sample size, these results cannot be directly compared to our other estimates. Allowing for further lags, however, yields similar findings. Allowing for a maximum of ten lags, the terrorism index will now longer share a statistically significant association with child mortality after the sixth lag.

news is not interesting to a broader audience or when, alternatively, press activity is curtailed by government repression (e.g., Drakos and Gofas 2006). Measurement error in the terrorism variable, in turn, is expected to give rise to attenuation bias, biasing the regression coefficients associated with terrorism towards zero. Third, endogeneity may be due to *feedback/reverse causation*. That is, terrorism may not only adversely affect children's health, but children's health may also determine terrorism. For instance, high levels of child mortality may encourage terrorism to voice dissent over and change this unfavorable status quo. Under such circumstances, OLS-estimates of the effect of terrorism on child mortality would be biased upwards. Alternatively, high levels of child mortality may mean that there are fewer resources available to engage in terrorism. For instance, high levels of child mortality may tie up money and time to child healthcare rather than terrorism. In this situation, OLS-estimates of the effect of terrorism on child mortality would be biased downwards.

To accommodate endogeneity concerns, we consider the following two-step instrumental-variable model:

$$terror_{j,it} = \beta_{11} * protest_{it} + \beta_{21} * X_{it} + \propto_i + \lambda_t + v_{it} \quad (2a)$$

$$health_{it} = \beta_{21} * \widehat{terror}_{j,it} + \beta_{22} * X_{it} + \propto_i + \lambda_t + \mu_{it} \quad (2b)$$

In the first stage (2a), we regress our terrorism variable on the baseline controls, country fixed- and year fixed-effects and our *instrumental variable* (*IV*), *protest*. The instrumental variable, *protest*, is a count variable measuring the number of protest events as recorded by the *Social Conflict in Africa* (*SCAD*) database (Salehyan et al. 2012); as with the other explanatory variables, we apply the inverse hyperbolic sine transformation to this variable to correct for skewness. The SCAD database contains information on protests, riots, strikes and other social disturbances. For our instrument, *protest*, we exclude all kinds of organized violence (e.g., organized violent riots) and only consider events that were intended to be non-violent (e.g., peaceful protests, demonstrations, and strikes).[9] In the second stage (2b), we use the fitted values of our terrorism variable from the first-stage regression ($\widehat{terror}$) to estimate its effect on child mortality. For the IV-estimates to be sound, the instrumental variable ought to (1) sufficiently correlate with the instrumented variable (instrument relevance) and (2) only affect

[9] There are still some cases where individual protesters were killed during intrinsically non-violent protests. We include these protest events as long as they fall below the threshold of organized violence according to the SCAD database.

the outcome (in our case, child mortality) via its effect on the instrumented variable (instrument exogeneity).

Instrument Relevance. We expect social protest to positively predict terrorist activity. Non-violent protest may lead to terrorism in various ways. First, non-violent protests (e.g., anti-government demonstrations) can delegitimize the government and encourage terrorists to use more violent methods. Second, if, despite the non-violent protests, grievances are not accommodated, this can lead to radicalization. Finally, protests may encourage outbidding between dissident groups over the same, mobilized sympathizers, which again will foster radicalization (Sprinzak 1991; Bakker et al. 2016). Indeed, Bakker et al. (2016) show that non-violent dissent is a positive predictor of terrorist activity.

To assess instrument relevance, we report the Kleibergen-Paap first-stage F-statistic. As a rule of thumb, if this statistic exceeds the critical value of F=10, the instrument is considered sufficiently strong. However, this rule of thumb has received some criticism for being anti-conservative, meaning that instruments may be weak even if F>10 (Lee et al. 2020). Thus, we also report results for the Anderson-Rubin test (Anderson and Rubin 1949) that is robust to arbitrarily weak instruments (Lee et al. 2020). Here, a rejection of the Anderson-Rubin test null hypothesis indicates that the coefficient of the endogenous regressor in the structural equation is equal to zero. Rejecting the null hypothesis thus suggests that our instrument is sufficiently strong. Finally, we report Anderson-Rubin confidence intervals due to Anderson and Rubin (1949), which are robust to arbitrarily weak instruments, and compare them to the confidence intervals constructed from the IV-regression. Finding that both confidence intervals do not include the null would further strengthen confidence in the strength of our instrument.

Instrument Exogeneity. There are two major threats to our identification strategy. First, it is possible that there are other changes over time that are spuriously correlated with both the instrument and child mortality. We account for this possibility by including year fixed-effects in all specifications. Second, there may be economic, demographic or other factors that might simultaneously affect terrorism and children's health at the grid-level. As already argued above when introducing the control variables, for our analysis we therefore choose an appropriate set of confounding variables that are expected to influence both child mortality and terrorism to mitigate the effect of confounders. Ultimately, while the exclusion restriction cannot be tested directly, we also rely on the plausibly exogenous framework of Conley et al. (2012) and van Kippersluis and Rietveld (2018) to strengthen confidence in the soundness of our instrumental-variable strategy. As described below in more detail, this method allows us to directly examine how plausible violations of the exclusion restriction – determined by the data – matter to causal inference. The idea is that allowing for violations of the exclusion restriction and still finding that terrorism matters to child mortality would raise confidence in our instrumental-variable approach.

5.2 Empirical Results

We report the instrumental-variable estimates in Table 2. Consistent with the two-way fixed-effects estimates reported above, we find that higher levels of terrorist activity result in higher levels of child mortality. This finding holds regardless of how we operationalize terrorist activity. The results for the various controls mirror those reported in Table 1.

—Table 2 here—

Importantly, the various IV-diagnostics indicate that the IV-estimates are trustworthy. First, as hypothesized, stronger social protest is correlated with more terrorism, confirming that social protests are a source of anti-system mobilization and violent radicalization. Second, the effect of social protest on terrorism is likely sufficiently strong, as indicated by the first-stage F-statistic being larger than ten. Third, the Anderson-Rubin test results, which are robust to potentially weak instruments, yield the same conclusion concerning instrument relevance. Finally, the Anderson-Rubin confidence intervals, again robust to weak instruments, are very similar to the confidence intervals constructed from the IV-regression, further raising our confidence in the relevance of the instrument.

While the IV-estimates are – as expected – less precisely estimated, substantively they point to a larger effect of terrorism on child mortality compared to the two-way fixed-effects OLS estimates. Our IV-estimates imply that a ten percent increase in the terrorism index results in an approximately 0.12 percentage point increase in the child mortality risk [95% *CI*: 0.02; 0.21]. The same increase is only associated with an 0.01 percentage point increase [95% *CI*: 0.007; 0.012] in the two-way fixed-effects OLS-setting. The downward bias associated with the OLS-estimates may have two sources. First, it may be due to reverse causality, where higher levels of child mortality disincentivize terrorism by reducing the amount of time and resources available to engage in terrorism. Second, the bias may be due to measurement error in terrorism, leading to attenuation bias.

5.3 Plausibly Exogenous Framework

While we have established the relevance of our instrumental variable through various statistical tests, the validity of our IV-approach also depends on the assumption of instrument exogeneity. Even though we cannot directly test for instrument exogeneity, we can probe the plausibility of this assumption – that non-violent protest only affects children's mortality via terrorism – by relying on the plausibly exogenous framework introduced by Conley et al. (2012) and further advanced by van Kippersluis and Rietveld (2018).

The main idea of the plausibly exogenous method is to relax the assumption of perfect instrument exogeneity. It involves the following variant of our previously introduced two-step IV-model:

$$terror_{it} = \beta_{11} * protest_{it} + \beta_{21} * X_{it} + \propto_i + \lambda_t + v_{it} \quad (3a)$$

$$health_{it} = \beta_{21} * \widehat{terror}_{it} + \gamma * protest_{it} + \beta_{22} * X_{it} + \propto_i + \lambda_t + \mu_{it} \quad (3b)$$

Here, we now allow our instrument, *protest*, to enter the second-stage regression (3b) with a coefficient γ. This implies that non-violent social protest can have a direct effect on children's health. For instance, this kind of protest may burden public and social infrastructure and thus make it more difficult to adequately provide children's healthcare. Allowing for a direct effect of social protest on children's health means, of course, that the exclusion restriction is violated.[10] By considering various values of γ, we can investigate how violations of the exclusion restriction matter to our IV-estimates (Conley, Hansen, and Rossi 2012; van Kippersluis and Rietveld 2018).

To find plausible values of γ, we consider the so-called *zero first-stage* following van Kippersluis and Rietveld (2018). That is, we consider a sample of grids that did not experience terrorism during our period of observation. Considering these grids serves two purposes. First, if protest only affects health via terrorism, then there should be no statistically significant association between protest and child mortality for grids that do not experience terrorism. Second, when we estimate the effect of protest on child mortality for the zero first-stage, the regression coefficient associated with protest can be used as a plausible value for γ.[11]

[10] By contrast, in case of perfect instrument exogeneity, γ would be equal to zero and the exclusion restriction would hold.

[11] To see this, consider that the reduced-form relationship between social protest and children's health captures both γ and β_{21}, i.e., the effect of social protest on children's mortality via local terrorism from Equation (3b). However, when in the zero first-stage case a grid does not see

We present our findings in Table 3. In Panel A, we report the reduced-form estimates of the effect of social protest on child mortality. We find that social protest is not a statistically significant predictor of children's mortality for the zero first-stage group; in contrast, higher levels of protest are associated with higher mortality risk for those grids that experience terrorism. This strongly indicates that social protest only affects child mortality via terrorist activity, in line with the instrument exogeneity argument we outlined above.

—Table 3 here—

We also use the regression coefficients associated with social protest from the zero first-stage group to augment our IV-estimations, as in Equation (3b). As shown in Panel B, allowing for respective (plausible) violations of the exclusion restriction as they follow from the zero first-stage group, we still find that higher levels of terrorist activity result in more negative child mortality outcomes. That is, even when allowing for plausible amounts of instrument endogeneity, our main empirical conclusion that terrorism adversely affects children's health still stands.

6. Panel Event-Study Approach

6.1 Empirical Model

To further strengthen our claim that the effect of terrorism on child mortality risk is indeed causal, we resort to the panel event-study approach as an alternative empirical method. This method is related to older difference-in-differences or two-way fixed effect models. For further discussion of this approach, we refer to, e.g., Clarke and Schythe (2021), Sun and Abraham (2021) and Freyaldenhoven et al. (2021). For the panel event-study approach, we consider the following model:

$$health_{it} = \sum_{m=-G}^{M} \beta_m td_{i,t-m} + \beta_2 * X_{it} + \alpha_i + \lambda_t + v_{it} \quad (4)$$

The dependent variable, fixed effects and controls are defined and operationalized as above. In contrast to previous models, Equation (4) includes the term *td*, a dummy variable that allows us to estimate the dynamic effect of a terrorism treatment on the outcome. Here, the outcome at time *t* can only be directly affected by the value of the terrorism treatment variable, *td*, at most $M \geq 0$ periods before *t* and at most $G \leq 0$ periods after *t*.

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terrorism over our period of observation, then the reduced-form estimate associated with social protest only captures γ.

The dummy variable *td* changes once a specific grid switches from the "no terrorism" to the "sustained terrorism" state. That is, the variable *td* is equal to zero in periods before this switch and equal to unity in the period of the switch and thereafter. Here, "sustained terrorism" means that (1) the mean-level of terrorist activity (either in terms of our terrorism index or the per capita number of terrorist incidents or casualties, respectively) is in the top 10% of all grids considered and (2) there are at least two years of terrorist activity. By using this definition of the terrorism event, we exclude grids that are barely affected by terrorism; indeed, for these grids a behavioral response to terrorism is unlikely.[12] Grid-cases that do not meet the aforementioned two criteria are the counterfactuals where *td* is always equal to zero. Given the definition of the treatment variable *td*, in our analysis we thus consider an absorbing treatment such that the treatment status is a non-decreasing series of zeros and ones (Sun and Abraham 2021).

By examining the variation in child mortality risk around the beginning of sustained terrorist activity (as defined above) compared with a baseline reference period, we can estimate both events lags ($M \geq 0$ periods before the treatment) and event leads $G \leq 0$ periods after the treatment (cp. Clarke and Tapia-Schythe 2021). For our analysis, we choose an effect window of *G*=*M*=*4*. The baseline reference period for our analysis is always $\beta_{-1} = 0$, meaning the period before the switch from "no terrorism" to "sustained terrorism" occurs. This normalization is necessary to avoid collinearity issues. Below, we summarize the leads and lags estimates in graphical form as an event-study plot, following Freyaldenhoven et al. (2021). Here, the normalization at $\beta_{-1} = 0$ means that the plotted coefficients can be interpreted as estimated effects relative to the period before the switch to "sustained terrorism".

An important assumption associated with Equation (4) is that in the absence of the treatment (i.e., without the emergence of sustained terrorism), trends in child mortality risk would be parallel between treated and untreated grids. We can use the event lead estimates to inspect for parallel trends in the pre-treatment period. Finding that event leads predict the outcome would imply anticipatory behavior or the presence of a confound. For instance, doctors and aid workers may expect terrorist violence to break out in the near future and emigrate from such potentially threatened areas. This, in turn, would adversely affects child health even before sustained terrorist violence erupts. Indeed, finding that child mortality trends in treated and

[12] As a robustness check, however, we also use several alternative definitions of the *td* variable (see Supplementary Table 4).

untreated grids are not parallel pre-event suggests that the identifying assumption (parallel post-event trends in the absence of the treatment) of the model is unlikely to be met (cp. Clarke and Tapia-Schythe 2021). Consequently, following Freyaldenhoven et al. (2021), we always report the pre-trend test results as part of our event-study plots.

6.2 Empirical Results

We present our estimates of Equation (4) in Supplementary Table 3. Below, we report the event-study plots in Figures 4 to 6, using the terrorism index, terrorism incidents or terrorism casualties, respectively, to define the terrorism treatment. Figures 4 to 6 report both the conventional 95% confidence intervals as they follow from the two-way fixed-effects estimates (these intervals are shown as the inner bars) as well as sup-*t* confidence bands described by Montiel Olea and Plagborg-Møller (2019). Here, the sup-*t* confidence bands are especially appropriate when we are interested in the entire event-time path, i.e., in implicitly testing multiple hypotheses at once (Freyaldenhoven et al. 2021). Finally, Figures 4 to 6 also report the sample-mean of the dependent variable (under-five child mortality risk, figure in parentheses on the y-axis) one period in advance of the treatment occurring in order to make it easier to evaluate economic significance (Freyaldenhoven et al. 2021).

Inspecting Figures 4 to 6 and the results reported in Supplementary Table 3, our results can be summarized as follows. First, regardless of which terrorism variable we use, switching from the "no terrorism" to the "sustained terrorism" state always results in an increase in child mortality risk. These results are also economically substantive. For instance, depending on which terrorism variable we use, in the period after the treatment sets in (i.e., at t=1), the child mortality risk is estimated to increase by 0.22 percentage points [95% *CI*: 0.10; 0.36] (terrorism index), 0.34 percentage points [95% *CI*: 0.16; 0.53] (terrorism incidents) and 0.22 percentage points [95% *CI*: 0.09; 0.34] (terrorism casualties), respectively.[13] Second, the strength of this effect tends to accumulate over time but also tends to vanish in later periods.[14] This may point to the possibility that certain behavioral adjustments to terrorism (e.g., migration) take some time, consequently also leading to a lagged effect on child mortality. Third, there is no evidence that event leads affect the dependent variable. This latter conclusion is also supported by the test of the parallel-trend assumption, where we do not find that this assumption is violated. This

[13] These confidence intervals follow from with the conventional two-way fixed-effects estimates.
[14] While effects appear to eventually level off with time, there is still the possibility that richer dynamic effects are at play than we consider in Equation (4). This may be an interesting avenue of future research. See also Freyaldenhoven et al. (2021) for a further discussion.

strengthens our argument that the identifying assumption (parallel post-event trends in the absence of the treatment) of the model is likely to be met. Finally, concerning statistical inference, all results reported above hold regardless of whether we rely on the conventional confidence intervals constructed from the two-way fixed-effects estimates or on sup-*t* confidence bands following Montiel Olea and Plagborg-Møller (2019).

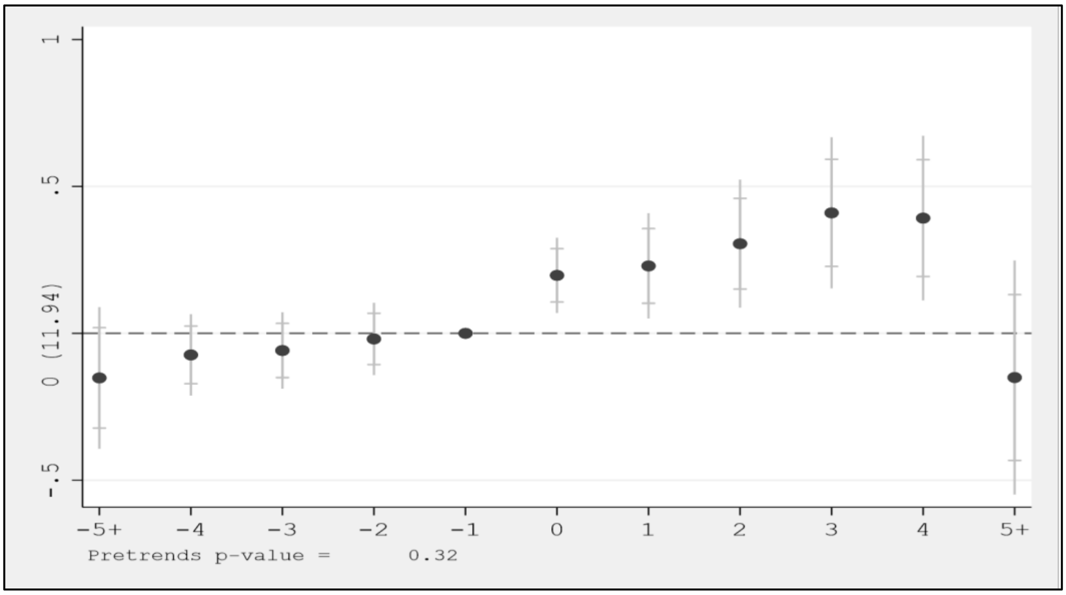

Figure 4: Event-Study Plot (Terrorism Index)

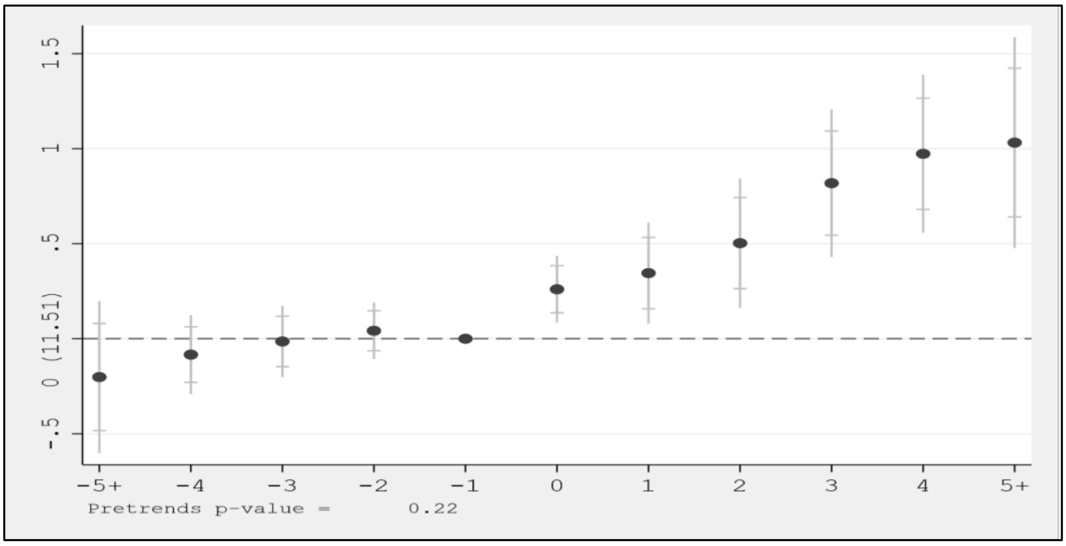

Figure 5: Event-Study Plot (Per Capita Terrorist Incidents)

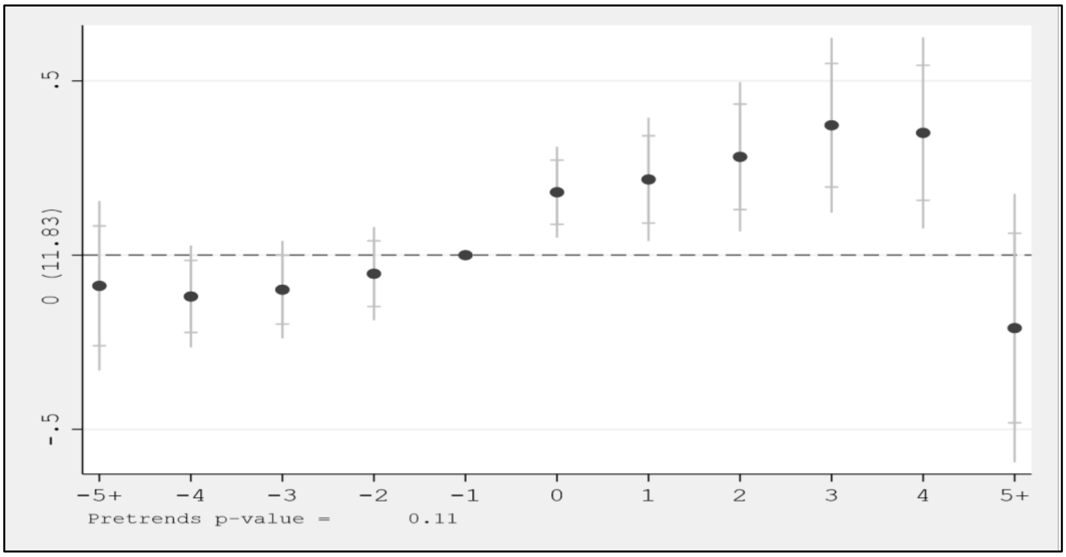

Figure 6: Event-Study Plot (Per Capita Terrorist Casualties)

6.3 Robustness Checks

Above, we used specific criteria to differentiate between treated and untreated grids. In Supplementary Table 4, we assess how robust our empirical results are to different operationalizations of the treatment. Specifically, we consider whether using different cut-offs with respect to the level of terrorism or the duration of terrorist activity to define the beginning of a sustained terrorist threat affecting a specific grid matter to our panel event-study results. As reported in Supplementary Table 4, changing the definition of the treatment variable does not affect our main empirical conclusion that grids treated by sustained terrorist activity see higher levels of child mortality risk compared to the untreated grids. What is more, the parallel-trend assumption is never violated, supporting the causal interpretation of our panel event-study estimates.

7. Exploration of Mechanisms

Through our previously presented empirical efforts, we have demonstrated that higher levels of terrorist activity are linked to higher levels of child mortality. Following the confirmation of our main hypothesis, in this sub-section we explore through which mechanisms this effect may materialize.

7.1 Potential Direct Effects through Destruction

When discussing our theoretical framework, we argued that terrorism may directly contribute to child mortality by victimizing children and their caretakers as well as by destroying the public health infrastructure. With respect to terrorism casualties, the GTD data does not provide concrete information on the number of African children (or parents) killed in terrorism.

However, we have information on the total number of casualties produced by terrorism. Here, the GTD tells us that terrorism killed approximately 78,000 individuals between 2000 and 2017 (or approximately 4,300 victims per year) for the grids and countries considered in our analysis. As shown in Figure 7, this amounts to less than one individual being killed by terrorism on average per grid-year pair.

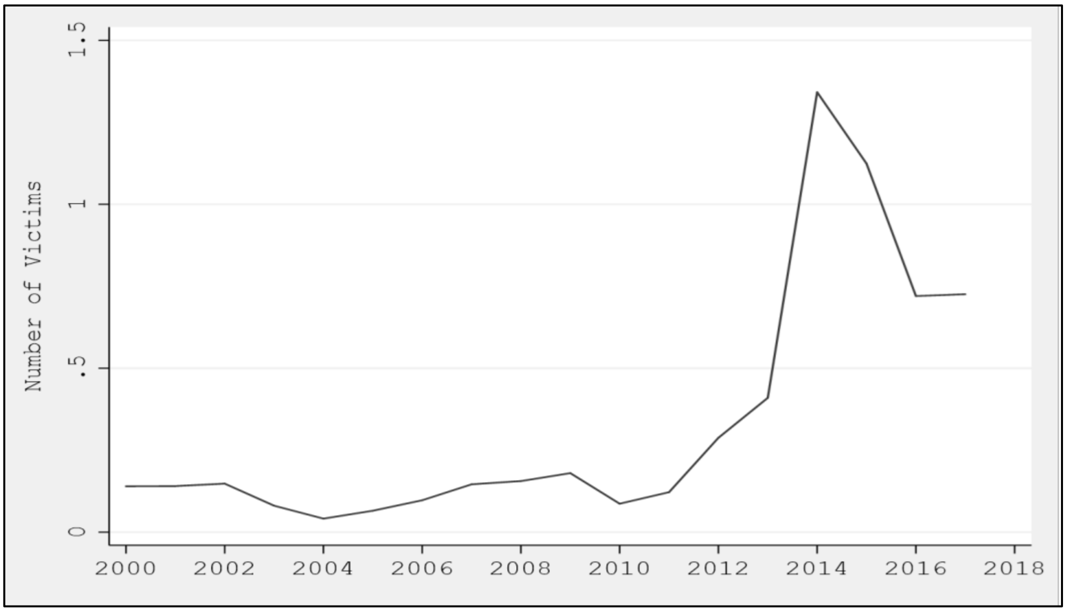

Figure 7: Mean-Number of Terrorism Casualties per Grid, 2000-2017

At the same time, according to the *United Nations Department of Economic and Social Affairs*, there were on average approximately 36 million births per year in Africa.[15] Assuming a risk of under-five child mortality of approximately 11 percent (see Supplementary Table 1), this implies that almost four million out of these 36 million children would die before the children would reach the age of five.[16] Accordingly, even if a quarter of all annual terrorism casualties were children under the age of five (which is a very strong assumption), this would mean doubling this casualty number (from approximately 1,100 to 2,200 children per year) would only have a miniscule effect on the overall child mortality risk, increasing this risk by approximately 0.003 percentage points. In sum, these back-of-the-envelope calculations point to a very benign *direct* effect of terrorism on child mortality (i.e., children being killed in terrorist incidents).

[15] The data can be found at https://population.un.org/wpp/.

[16] Note that both figures are averages over our observation period. Due to population growth, total births are substantially higher in 2017 compared to 2000, while the child mortality risk (e.g., due to medical advances) is considerably lower in 2017 compared to 2000.

Interrogating the GTD data with respect to the direct effect of terrorism on the public health infrastructure paints a similar picture. The GTD provides information on the targets of terrorist attacks. Reporting issues notwithstanding, out of the approximately 22,500 terrorist incidents recorded in our African sample between 2000 and 2017, only approximately 120 attacks were directed against public health infrastructure targets such as clinics, ambulances, and pharmacies. These figures, therefore, again point to benign direct effects of terrorism on child mortality through the destruction of public health institutions.

7.2 Correlations between Terrorism and Proximate Causes of Child Mortality

Given that its direct effects tend to be rather innocuous, terrorism's indirect effects seem to play a more important role. This is also in line with our theoretical argument outlined above.

Terrorism, we argued, can increase child mortality by producing a behavioral response in economic agents (parents, doctors, government officials etc.) that disbenefits child health. Ideally, we would want to directly measure such behavioral reactions, e.g., changes in parental decision-making in response to terrorism. Unfortunately, our data does not permit to do this. However, we do have data on various proximate causes of child mortality, which allows us to make some tentative conclusions regarding the most likely mechanisms. Thus, we study how terrorism correlates with these proximate mortality causes by considering the following two-way fixed-effects model:

$$cause_{j,it} = \beta_1 * terror_{it} + \beta_2 * X_{it} + \alpha_i + \lambda_t + v_{it} \quad (5)$$

Here, the main explanatory variable, *terror*, and the remaining controls and fixed-effects are as described above. The dependent variable, *cause*, refers to the j-th proximate cause of child mortality. We consider the following four variables aggregated at the grid-level: (1) the incidence of *malaria* as measured by the annual number of children under four who become infected by Plasmodium falciparum, (2) the under-five annual incidence rate of serious *diarrhea*, (3) the *diphtheria-pertussis-tetanus vaccine coverage* measured as the share of one- and two-year-old who are fully vaccinated (i.e., who have received three shots or more) and (4) *malnourishment*, measured as the share of under-five year old children that are underweight. The malaria and diarrhea incidence rates are from Weiss et al. (2019) and Reiner et al. (2018), respectively, while the vaccination and malnourishment data are from Mosser et al. (2019) and Osgood-Zimmerman et al. (2018), respectively.[17]

We argue that these four variables measure proximate causes of child mortality in the form of disease, lack of medical care and malnourishment. We argue that these constitute plausible indirect effects of terrorism, which are driven by the psychological and behavioral response to terrorism. For example, terrorism may produce fear among parents, making it less likely that they receive a sound health education (e.g., concerning malaria prevention) or seek preventive checkups for their children (which could reduce vaccination rates). To give a second example, terrorism may also trigger the departure of foreign aid workers and may induce changes in local public spending on healthcare, thereby causing the deterioration of medical and food supplies and a higher prevalence of infectious diseases. We hence expect terrorism to correlate with these proximate causes of child mortality.

7.3 Empirical Results

We report our empirical findings in Table 4. As shown in Panel A, terrorism positively correlates with the incidence of malaria and diarrhea, respectively. It is also positively associated with the risk of malnutrition. Finally, terrorism negatively correlates with vaccination rates. What is more, in Panel B of Table 4, we show that the various proximate causes of child mortality, in turn, unfavorably correlate with child mortality. For instance, lower vaccination rates are associated with a higher risk of child mortality.

—Table 4 here—

In sum, these findings are consistent with our expectations. Terrorism is associated with (1) the spread of potentially deadly diseases, (2) poorer vaccination efforts that would protect against potentially lethal infections and (3) higher levels of malnourishment risk which, in turn, is anticipated to contribute to child mortality, e.g., by curtailing the body's defenses. In other words, the findings reported in Table 4 speak to our theoretical argument that terrorism especially harms children's health by inducing various behavioral responses to terrorism that come at the expense of children's health.

8. Conclusion

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[17] All outcome measures are made available by the *Institute for Health Metrics and Evaluation* on their website https://ghdx.healthdata.org/local-and-small-area-estimation.

We study the relationship between terrorism and child mortality using high-resolution sub-national data (at the 0.5x0.5° grid-year level) for 52 African countries and territories between 2000 and 2015/2017. We rely on a two-way fixed effect approach as well as instrumental-variable and panel event-study approaches to examine whether terrorism adversely affects child mortality risk. The latter two approaches allow us to interpret our findings as causal.

Each econometric method we employ in our study has potential disadvantages. For instance, one may challenge the choice of our instrumental variable and whether it meets the exclusion restriction. Still, all empirical approaches point in the same direction: terrorism adversely influences child mortality in the African countries and territories we consider. Using our preferred baseline specification, we estimate that an increase of our terrorism index by 50 percent raises the child mortality risk by 0.04 percentage points [95% *CI*: 0.02; 0.06] (two-way fixed-effects OLS) and 0.63 percentage points [95% *CI*: 0.12; 1.13] (IV-estimates) in the same year, respectively.[18] Similarly, the panel event-study estimates suggest that the beginning of sustained terrorist activity leads to a contemporaneous rise in child mortality risk by 0.10 percentage points [95% *CI*: 0.06; 0.14]. Our analyses also suggest that adverse effects of terrorism become more pronounced over time. Effects seem to accumulate over several years, likely because the behavioral responses to terrorism (and thus their impact on child mortality) take some time to fully materialize. An interesting avenue of future research could be to study heterogeneity in the terrorism-child mortality nexus with respect to specific types of terrorism being especially impactful (e.g., terrorism against civilian vis-à-vis military targets). Such heterogeneity could also be influential on econometric grounds (e.g., Sun and Abraham 2021).

To put the estimated effect sizes into perspectives, we use figures on live births provided by the *United Nations Department of Economic and Social Affairs*. These figures indicate that for our African sample and observation period there were approximately 36 million births per year. Assuming a risk of under-five child mortality of approximately 11 percent (as our sub-national data suggests), this implies that almost four million out of these 36 million lives end before a child reaches the age of five. Ceteris paribus, an increase of our terrorism index by 50 percent would mean an increase (in the same year) of child deaths before the age of five by 16,000 individuals [95% *CI*: 8,000; 24,000] (two-way fixed-effects OLS) or 252,000 individuals [95% *CI*: 48,000; 520,000] (IV-estimates), respectively. Similarly, the panel event-study estimates

[18] Note that such an increase in the terrorism index was indeed observed for our sample between both 2011 and 2012 and 2013 and 2014.

suggest that the beginning of sustained terrorist activity will result in 40,000 additional deaths of children under five per year [95% *CI*: 24,000; 56,000].

That is, according to our back-of-the-envelope calculations, all econometric approaches point to potentially substantial annual increases in child mortality that can be linked to increases in terrorist activity. Given that the direct effects of terrorism (e.g., in terms of its lethality and destruction of public health infrastructure) tend to be very small, we are reasonably confident that increases in child mortality primarily emerge through the behavioral response of economic agents (e.g., parents, doctors, medical staff, aid workers and policymakers) to terrorism. Exploring the role of several potential mediators in the terrorism-child mortality nexus, we indeed show that higher levels of terrorist activity unfavorably correlate with several proximate causes of child mortality: the incidence of malaria and diarrhea, vaccination rates and malnourishment. Future work could examine these behavioral responses and mediators in more detail. For instance, it could be interesting to examine – using appropriate micro-level or survey data – whether it is parents, doctors, politicians or other economic agents that respond especially unfavorably (in terms of the consequences for child health) to terrorism.

Recent years saw encouraging advances in reducing child mortality in Africa. Our empirical analysis, however, suggests that terrorism produced some conspicuous setbacks with respect to this general downward trend. Domestic and international policymakers are thus called upon to counter terrorism as well as to mitigate behavioral responses to terrorism that are to the detriment of children's health, e.g., through information and education campaigns that adjust perceptions about terrorism and the risk it entails.

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	(1)	(2)	(3)	(4)	(5)	(6)	(7)	
Dependent Variable →	Mort. U5	Mort. U5	Mort. U5	Mort. Neo	Mort. U1	Mort. U5	Mort. U5	Mort. U5
Terrorism Index	0.048***	0.088***	0.087***	0.014**	0.058***			0.028*
	(0.016)	(0.020)	(0.019)	(0.005)	(0.011)			(0.015)
Terrorism Index $_{t-1}$								0.071***
								(0.015)
Terrorism Index $_{t-2}$								0.125***
								(0.015)
[Sum of Coefficients]								[0.225]***
[Standard error]								[0.037]
Terrorist Incidents p.c.						0.167***		
						(0.030)		
Terrorism Casualties p.c.							0.071***	
							(0.021)	
Nightlights			-0.225**	-0.163***	-0.219***	-0.225**	-0.226**	-0.133
			(0.106)	(0.024)	(0.053)	(0.107)	(0.107)	(0.089)
Distance to City			0.411***	0.170***	0.120**	0.413***	0.411***	0.266***
			(0.099)	(0.027)	(0.055)	(0.099)	(0.099)	(0.094)
Urban			-1.778***	-0.326**	-0.560	-1.767***	-1.775***	-1.918***
			(0.649)	(0.149)	(0.342)	(0.649)	(0.649)	(0.644)
Female Education			-0.443***	-0.145***	-0.190***	-0.444***	-0.442***	-0.275***
			(0.038)	(0.011)	(0.022)	(0.038)	(0.038)	(0.042)
Temperature			0.023**	-0.001	0.007	0.023**	0.023***	0.021***
			(0.009)	(0.002)	(0.004)	(0.009)	(0.009)	(0.008)
Precipitation (SPEI)			0.003	0.004***	0.001	0.003	0.003	0.019***
			(0.003)	(0.001)	(0.002)	(0.003)	(0.003)	(0.003)
Observations	142,473	101,089	101,089	101,089	101,089	101,089	101,089	87,528
Adjusted R^2	0.95	0.96	0.96	0.95	0.95	0.96	0.96	0.96

Notes: Mort. U5= Mortality risk of children under the age of five. Mort. Neo= Mortality risk of children in first 28 days after birth. Mort. U1= Mortality risk of children under the age of one. Grid- and year-fixed effects always included. Standard errors clustered at the grid-level in parentheses. *p<0.1, **p<0.05, ***p<0.01.

Table1: Two-Way Fixed-Effects Estimates

	(1)	(2)	(3)
Terrorism Index	1.252**		
	(0.518)		
Terrorist Incidents p.c.		2.346**	
		(1.044)	
Terrorism Casualties p.c.			1.594**
			(0.689)
Nightlights	-0.138	-0.137	-0.122
	(0.111)	(0.115)	(0.114)
Distance to City	0.381***	0.411***	0.361***
	(0.107)	(0.112)	(0.110)
Urban	-2.040***	-1.887***	-2.137***
	(0.688)	(0.685)	(0.707)
Female Education	-0.453***	-0.467***	-0.449***
	(0.039)	(0.041)	(0.040)
Temperature	0.012	0.012	0.011
	(0.010)	(0.010)	(0.011)
Precipitation (SPEI)	0.001	-0.001	0.001
	(0.004)	(0.004)	(0.004)
First-Stage Results and IV-Diagnostics			
Social Protest	0.090***	0.048***	0.071***
	(0.022)	(0.015)	(0.019)
Endogeneity Test (p-value)	(0.00)***	(0.00)***	(0.00)***
First-Stage F-Statistic	17.25	10.68	14.48
AR-Test (p-value)	(0.00)***	(0.00)***	(0.00)***
AR Confidence 90% Interval	[0.512; 2.405]	[0.994; 5.155]	[0.655; 3.221]
Observations	101,089	101,089	101,089

Notes: Dependent variable=Mortality risk of children under the age of five. Grid- and year-fixed effects always included. Standard errors clustered at the grid-level in parentheses. Instrument for terrorism=Number of social protests that do not involve organized violence (inverse hyperbolic sine transformation). Results for first-stage regression for instrumented reported (results for other controls omitted). *p<0.1, **p<0.05, ***p<0.01.

Table 2: Instrumental-Variable Estimates

	(1)	(2)	(3)
Terrorism Variable →	Index	Incidents	Casualties
Grids in zero first-stage group	5,485	5,485	5,762
(Number of Observations)	(82,104)	(82,104)	(86,259)
Grids in remaining sample	1,266	1,266	989
(Number of observations)	(18,985)	(18,985)	(14,830)
Panel A: Effect of Social Protest on Child Mortality (Reduced Form)			
Full Sample	0.113***	0.113***	0.113***
	(0.039)	(0.039)	(0.039)
Zero first-stage group	-0.054	-0.054	-0.062
	(0.065)	(0.065)	(0.058)
Remaining sample	0.189***	0.189***	0.238***
	(0.044)	(0.044)	(0.051)
Panel B: Effect of Terrorism on Child Mortality for Full Sample (95% Confidence Interval)			
2SLS regression	[0.237; 2.267]	[0.301; 4.392]	[0.243; 2.945]
Plausibly exogenous regression	[0.628; 2.533]	[1.159; 4.577]	[0.779; 3.211]
Value of γ for plausibly exogenous regression	-0.054	-0.054	-0.062
Further Controls	Yes	Yes	Yes

Notes: OLS-estimates (Panels A) IV-estimates (Panel B) reported. All models include grid- and year-fixed effects. *Full sample* refers to the respective baseline sample; these results are reported for comparison. Zero first-stage sample refers to grids that never experience terrorist activity (Models 1-2) or terrorism casualties (Model 3) over the period of observation. Standard errors clustered at the grid-level in parentheses. *p<0.1, **p<0.05, ***p<0.01.

Table 3: Plausibly Exogenous Framework

Panel A: Correlation of Terrorism and Potential Mediators				
	(1a)	(2a)	(3a)	(4a)
Dependent Variable →	Malaria	Diarrhea	Vaccinations	Malnutrition
Terrorism Index	0.004***	0.007***	-0.002**	0.001*
	(0.001)	(0.001)	(0.001)	(0.000)
Nightlights	0.016	0.002	-0.022***	0.012***
	(0.010)	(0.007)	(0.006)	(0.002)
Distance to City	-0.059***	0.063***	0.069***	-0.001
	(0.010)	(0.009)	(0.008)	(0.003)
Urban	-0.063	-0.163***	0.232***	0.035**
	(0.062)	(0.037)	(0.042)	(0.015)
Female Education	0.015***	-0.044***	0.022***	-0.033***
	(0.004)	(0.003)	(0.003)	(0.001)
Temperature	0.007***	0.001	0.006***	0.001***
	(0.001)	(0.001)	(0.001)	(0.000)
Precipitation (SPEI)	0.003***	-0.001	-0.002***	0.000
	(0.001)	(0.001)	(0.000)	(0.000)
Adjusted R^2	0.93	0.93	0.93	0.95
Panel B: Correlation between Potential Mediators and Child Mortality				
	(1b)	(2b)	(3b)	(4b)
Mediator Variable →	Malaria	Diarrhea	Vaccinations	Malnutrition
Mediator	1.173***	1.659***	-1.481***	6.950***
	(0.071)	(0.100)	(0.129)	(0.446)
Nightlights	-0.360***	-0.233**	-0.258**	-0.312***
	(0.106)	(0.104)	(0.107)	(0.101)
Distance to City	0.535***	0.308***	0.516***	0.417***
	(0.099)	(0.099)	(0.099)	(0.095)
Urban	-1.996***	-1.490**	-1.419**	-2.006***
	(0.660)	(0.634)	(0.631)	(0.638)
Female Education	-0.443***	-0.369***	-0.409***	-0.210***
	(0.038)	(0.039)	(0.038)	(0.037)
Temperature	0.011	0.023***	0.032***	0.015*
	(0.009)	(0.009)	(0.009)	(0.009)
Precipitation (SPEI)	0.002	0.003	-0.000	0.002
	(0.004)	(0.003)	(0.003)	(0.003)
Adjusted R^2	0.96	0.95	0.96	0.96
Observations (Both Panels)	100,006	101,089	101,074	101,074

Notes: OLS-estimates reported. Dependent variable in Panel B=Mortality risk of children under the age of five. Grid- and year-fixed effects always included. Standard errors clustered at the grid-level in parentheses. *p<0.1, **p<0.05, ***p<0.01.

Table 4: Potential Transmission Channels

	N*T	Mean	SD	Min	Max
Child Mortality Risk (under 5)	101,089	11.03	4.48	1.52	30.29
Child Mortality Risk (under 28 days)	101,089	3.27	1.02	0.54	7.71
Child Mortality Risk (under 1)	101,089	6.84	2.31	1.17	16.46
Terrorism Index	101,089	0.04	0.37	0	9.58
Terrorist Incidents (absolute)	101,089	0.07	1.77	0	373
Terrorism Casualties (absolute)	101,089	0.30	7.78	0	953
Population (in millions)	101,089	0.13	0.37	0	17.26
Nightlights	101,089	0.38	2.17	0	61.51
Travel time to regional capital	101,089	7.73	5.37	1.08	43.34
Urbanization	101,089	0.16	0.69	0	24.41
Female Education	101,089	4.47	2.97	0.01	13.59
Precipitation (SPEI)	101,089	0.11	0.91	-3.45	2.83
Temperature	101,089	24.50	4.07	7.51	39.53
Social Protest (absolute)	101,089	0.03	0.86	0	175
Plasmodium falciparum incidence rate	100,006	0.63	0.54	0	1.99
Incidence rate of serious diarrhea	101,089	46.07	13.42	14.05	107.8
Diphtheria-pertussis-tetanus vaccine coverage	101,089	204.48	60.11	62.02	486.14
Risk of child under-5 years being underweight	101,074	0.24	0.10	0.03	0.73

Notes: Many variables enter the empirical model in transformed form (hyperbolic sine transformation). See the main text for a discussion.

Supplementary Table 1: Summary Statistics

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Terrorism Measure	0.050**	0.063***	0.010***	0.232***	0.088***	0.051**	0.088***	0.094***	0.087***	0.015***	0.076***	0.165***
	(0.022)	(0.015)	(0.023)	(0.052)	(0.019)	(0.021)	(0.019)	(0.025)	(0.019)	(0.001)	(0.019)	(0.047)
Nightlights	-0.228**	-0.225**	-0.225**	-0.230**	-0.194*		-0.223**	-0.103	-0.224**	-0.085***	-0.142	-0.204
	(0.106)	(0.106)	(0.107)	(0.106)	(0.105)		(0.107)	(0.121)	(0.106)	(0.007)	(0.102)	(0.175)
Distance to City	0.408***	0.411***	0.411***	0.408***	0.417***	0.378***		0.769***	0.414***	0.057***	0.251***	0.334**
	(0.099)	(0.099)	(0.099)	(0.098)	(0.099)	(0.101)		(0.119)	(0.099)	(0.008)	(0.096)	(0.132)
Urban	-1.831***	-1.783***	-1.777***	-1.851***	-1.519**	-0.597	-1.722***	-0.941	-1.786***	-0.084*	-1.554*	-1.572**
	(0.648)	(0.649)	(0.649)	(0.648)	(0.643)	(0.670)	(0.648)	(0.715)	(0.649)	(0.045)	(0.615)	(0.630)
Female Education	-0.444***	-0.442***	-0.443***	-0.443***	-0.442***	-0.476***	-0.437***	-0.552***	-0.442***	0.025***	-0.496***	-0.617***
	(0.038)	(0.038)	(0.038)	(0.038)	(0.038)	(0.039)	(0.038)	(0.046)	(0.038)	(0.003)	(0.036)	(0.042)
Temperature	0.024***	0.023**	0.023**	0.024***	0.023***	0.022**	0.023***	0.027***	0.457**	0.003***	0.020**	0.024*
	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.199)	(0.001)	(0.008)	(0.013)
Precipitation (SPEI)	0.003	0.003	0.003	0.003	0.004	0.001	0.002	-0.007*	0.003	-0.002***	-0.006*	0.131***
	(0.003)	(0.003)	(0.003	(0.003)	(0.003)	(0.004)	(0.003)	(0.004)	(0.004)	(0.000)	(0.004)	(0.014)
Additional or Alternative Control					-0.208***	-1.931***	-0.151***	-0.015				
					(0.018)	(0.314)	(0.023)	(0.052)				
Observations	101,280	101,089	101,089	101,280	101,089	96,139	101,093	77,812	101,089	101,089	101,089	26,979
Adjusted R^2	0.96	0.96	0.96	0.96	0.96	0.96	0.96	0.94	0.96	0.97	0.96	0.96

Robustness Checks: (1) terrorism index not weighted by population size, (2) terrorism index weighs the number of casualties four times stronger than the number of incidents, (3) terrorism is log-transformed (with unity added for zero-observations), (4) binary indicator, equal to unity when grid-year pair sees any terrorist activity, (5) also controls for population size (data from Bright et al. 2018), (6) controls for gross cell product (data from Nordhaus 2006) instead of nightlights, (7) controls for travel distance to market instead of city (data from Müller-Crepon 2021), (8) controls for number of excluded social/ethnic groups (data from Vogt et al. 2015), (9) uses inverse hyperbolic sine transformation of temperature and Precipitation, (10) uses inverse hyperbolic sine transformation of dependent variable , (11) lags the terrorism variable and the controls by one year, (12) uses four-year averages instead of annual data.

Notes: Dependent variable=Mortality risk of children under the age of five. Grid- and year-fixed effects always included. Standard errors clustered at the grid-level in parentheses. *p<0.1, **p<0.05, ***p<0.01.

Supplementary Table 2: Robustness Checks for Two-Way Fixed-Effects Models

	(1)	(2)	(3)
Terrorism Variable →	Index	Incidents	Casualties
Terror (+5)	-0.152*	-0.202	-0.088
	(0.088)	(0.144)	(0.088)
Terror (+4)	-0.073	-0.084	-0.119**
	(0.050)	(0.075)	(0.053)
Terror (+3)	-0.058	-0.014	-0.099**
	(0.047)	(0.068)	(0.050)
Terror (+2)	-0.019	0.042	-0.053
	(0.044)	(0.054)	(0.048)
Terror (Event)	0.197***	0.260***	0.181***
	(0.046)	(0.063)	(0.047)
Terror (-1)	0.229***	0.345***	0.217***
	(0.065)	(0.096)	(0.063)
Terror (-2)	0.305***	0.503***	0.282***
	(0.079)	(0.122)	(0.077)
Terror (-3)	0.410***	0.819***	0.373***
	(0.093)	(0.140)	(0.090)
Terror (-4)	0.392***	0.973***	0.351***
	(0.101)	(0.149)	(0.099)
Terror (-5)	-0.150	1.032***	-0.209
	(0.144)	(0.200)	(0.139)
Nightlights	-0.210**	-0.216*	-0.209**
	(0.106)	(0.111)	(0.106)
Distance to City	0.404***	0.403***	0.403***
	(0.102)	(0.105)	(0.102)
Urban	-1.695**	-1.744**	-1.647**
	(0.673)	(0.674)	(0.672)
Female Education	-0.445***	-0.453***	-0.444***
	(0.039)	(0.039)	(0.039)
Temperature	0.023**	0.022**	0.023**
	(0.009)	(0.009)	(0.009)
Precipitation (SPEI)	0.003	0.002	0.004
	(0.004)	(0.004)	(0.004)
Parallel Trends (p-value)	(0.32)	(0.22)	(0.11)

Notes: Grid- and year-fixed effects always included. (+5, +4, …) are the leads. (-5, -4, …) are the lags. Event-time coefficient at year before event normalized to be 0. Standard errors clustered at the grid-level in parentheses. *p<0.1, **p<0.05, ***p<0.01.

Supplementary Table 3: Treatment Effect Estimates

	(1)	(2)	(3)	(4)	(5)
Terror (+5)	-0.152*	-0.153	-0.135*	-0.150*	-0.590
	(0.087)	(0.096)	(0.079)	(0.087)	(0.488)
Terror (+4)	-0.073	-0.097*	-0.069	-0-078	-0.345
	(0.050)	(0.057)	(0.044)	(0.050)	(0.329)
Terror (+3)	-0.058	-0.072	-0.060	-0.064	-0.304
	(0.047)	(0.052)	(0.043)	(0.047)	(0.271)
Terror (+2)	-0.019	-0.014	-0.027	-0.024	-0.298
	(0.044)	(0.048)	(0.041)	(0.044)	(0.220)
Terror (Event)	0.196***	0.224***	0.140***	0.193***	0.387**
	(0.046)	(0.050)	(0.043)	(0.046)	(0.194)
Terror (-1)	0.228***	0.276***	0.155***	0.226***	0.544***
	(0.065)	(0.069)	(0.059)	(0.065)	(0.198)
Terror (-2)	0.304***	0.348***	0.205***	0.302***	0.546**
	(0.079)	(0.085)	(0.072)	(0.079)	(0.257)
Terror (-3)	0.409***	0.504***	0.277***	0.406***	0.814***
	(0.092)	(0.099)	(0.084)	(0.093)	(0.301)
Terror (-4)	0.391***	0.508***	0.253***	0.389***	0.743**
	(0.101)	(0.107)	(0.092)	(0.101)	(0.363)
Terror (-5)	-0.147	0.048	-0.217*	-0.143	0.373
	(0.144)	(0.148)	(0.126)	(0.144)	(0.431)
Nightlights	-0.210**	-0.214**	-0.213**	-0.238*	-0.225**
	(0.106)	(0.108)	(0.107)	(0.125)	(0.111)
Distance to City	0.404***	0.405***	0.403***	0.455***	0.406***
	(0.102)	(0.102)	(0.102)	(0.103)	(0.102)
Urban	-1.697**	-1.760***	-1.653**	-2.668***	-1.805***
	(0.672)	(0.675)	(0.672)	(0.790)	(0.678)
Female Education	-0.445***	-0.447***	-0.442***	-0.448***	-0.443***
	(0.039)	(0.039)	(0.039)	(0.040)	(0.039)
Temperature	0.023**	0.022**	0.023**	0.026***	0.024**
	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)
Precipitation (SPEI)	0.003	0.003	0.004	0.003	0.003
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Parallel Trends (p-value)	(0.32)	(0.15)	(0.36)	(0.31)	(0.37)
Observations	101,280	101,280	101,280	94,125	101,280

Alternative treatment definitions: (1) mean-grid terrorism index larger than 0.136 with at least one year of terrorist activity, (2) mean-grid terrorism index larger than 0.207 (one standard deviation) with at least two years of terrorist activity, (3) mean-grid terrorism index larger than 0.053 (sample mean) with at least two years of terrorist activity, (4) mean-grid terrorism index larger than 0.136 with at least two years of terrorist activity; grids with terrorist activity below threshold dropped, (5) mean-grid terrorism index larger than 0.136 with at least ten years of terrorist activity

Notes: Grid- and year-fixed effects always included. (+5, +4, …) are the leads. (-5, -4, …) are the lags. Event-time coefficient at year before event normalized to be 0. Standard errors clustered at the grid-level in parentheses. *p<0.1, **p<0.05, ***p<0.01.

Supplementary Table 4: Robustness Treatment Effect Estimates