Epidemics and Conflict:

Evidence from the Ebola outbreak in Western Africa

12 December 2017, 9:45-11:00, bld. 72, room 465

Ada Gonzalez-Torres (European University Institute**)**

Abstract: This paper investigates the impact of a rapidly spreading epidemic on civil violence in the context of the largest Ebola outbreak in history, in Western Africa. The identification strategy relies on the epidemiological features of the Ebola Virus Disease (EVD). We exploit the dynamics of the disease and weekly frequency data at the local level to analyze the effect of new infections on riots, protests and violence against institutional authorities. The impacts are large, localized and tied to containment efforts. The results suggest that state coercion and demand for public goods are mechanisms fueling conflict. Containing the epidemic requires a change in cultural practices which leads to social unrest, especially for groups facing higher costs of cultural adaptation, low trust in institutional authorities and depending on the response of the state. This further deepens mistrust in institutions after the epidemic, especially among these communities. The paper contributes to a growing literature on conflict by providing light into mechanisms triggering civil violence.

Epidemics and Conflict:
Evidence from the Ebola outbreak in Western Africa

Job Market Paper

Ada Gonzalez-Torres* and Elena Esposito †

This Version: November 24, 2017

Link to the most current version

Abstract

This paper investigates the impact of a rapidly spreading epidemic on civil violence in the context of the largest Ebola outbreak in history, in Western Africa. The identification strategy relies on the epidemiological features of the Ebola Virus Disease (EVD). We exploit the dynamics of the disease and weekly frequency data at the local level to analyze the effect of new infections on riots, protests and violence against institutional authorities. The impacts are large, localized and tied to containment efforts. The results suggest that state coercion and demand for public goods are mechanisms fueling conflict. Containing the epidemic requires a change in cultural practices which leads to social unrest, especially for groups facing higher costs of cultural adaptation, low trust in institutional authorities and depending on the response of the state. This further deepens mistrust in institutions after the epidemic, especially among these communities. The paper contributes to a growing literature on conflict by providing light into mechanisms triggering civil violence.

Keywords: conflict, epidemics, Africa, collective action, state coercion, public goods, aid

*Job Market Paper. ada.gonzalez-torres@berkeley.edu, Visiting research scholar at UC Berkeley, 2016-2018; PhD Candidate in Economics at the European University Institute, 2013-present. I am indebted to Jérôme Adda, Edward A. Miguel and Michèle Belot for invaluable support and guidance. I am grateful to Michael Anderson, Peter R. Hansen, Andrea Ichino, David K. Levine and Noam Yuchtman for their constant guidance. We are indebted with Matteo Cervellati and Uwe Sunde for detailed discussions. I am thankful to Chris Blattman, Ernesto dal Bò, Ylenia Brilli, Katherine Casey, Marc Dordal, Benjamin Faber, Frederico Finan, Gabriela Galassi, Paul Gertler, Manuel Gehrke Ryff Moreira, Jonas Hjort, Solomon Hsiang, Michael Jannson, Ibrahima Kaba, Supreet Kaur, Madina Kurmangaliyeva, Nicholas Li, John Loeser, Andreas Madestam, Magne Mogstad, Tomás Monarrez, Ben Morse, Nathan Nunn, Gerard Roland, Raul Sánchez de la Sierra, Christian Traeger, Manuela Travaglianti, Maarten Voors, Joachim Voth, Yang Xie, Guo Xu for helpful feedback and insights. Many thanks to participants in seminars or workshops at Columbia Univ., EUI, UC Berkeley, Pacific Development Conference at UC Riverside and WGAPE at University of British Columbia. I gratefully acknowledge funding from the Spanish Ministry of Education, the EUI and for data collection in Guinea from the Royal Netherlands Embassy in Ghana.

†elena.esposito.1@unil.ch, University of Lausanne (DEEP-HEC)

1 Introduction

The last decades have seen the emergence or re-emergence of infectious disease outbreaks throughout the world, with devastating consequences for developing countries, especially affecting the young, and causing worldwide alarm fearing contagion[1]. While the immediate effect of epidemics are clearly illness and death, their economic and political consequences are more complex and are at the center of the debate on why poor countries are poor (Bloom and Sachs, 1998, Acemoglu and Johnson, 2007). Developing countries are not only poorer and have weaker institutions than rich countries, they also have a higher risk of emerging infectious disease outbreaks and higher rates of civil violence[2]. Epidemics can lead to a poverty trap, as the social, economic and institutional disruption they carry can lead to social upheaval, especially in weak institutional settings, potentially further debilitating the state. On the other hand, depending on the nature of civil violence and the capacity of the state to respond to a disease outbreak, epidemics can also create an opportunity for technological and institutional change[3]. Understanding the drivers of civil violence and the state response to an epidemic is therefore critical to our understanding of economic development.

In this paper we seek to identify the impact of an epidemic on civil violence and the role of the state response and trust in authorities in determining this relationship. In doing so we aim to learn about determinants of conflict and in particular the part played by perceived state coercion and demand for public goods in fueling social unrest. We also study the consequences for long-run trust in institutions.

Numerous acts of civil violence are reported in newspapers following the spread of epidemics throughout the African continent today[4]. Mobs, riots and attacks often target government authorities, health personnel and social workers aiming to contain an epidemic[5]. Since these episodes are especially likely to happen in weak institutional settings, under the presence of latent conflict, and poverty and conflict can facilitate the spread of disease, establishing causal evidence is difficult, yet of great policy relevance. Identifying the drivers of civil violence in the

[1]The 1918 influenza pandemic took the lives of 50 million people, HIV/AIDS killed over 35 million. While other epidemics, such as severe acute respiratory syndrome (SARS) in 2003, H1N1 in 2009 or the Ebola epidemic in 2014-15, had lower death tolls, they led to huge social and economic disruption (GHRF, 2016).

[2]Civil violence is perpetrated collectively by citizens or civilian organizations. Interpersonal and collective violence kills 12.3 people in 100'000 per capita in low income countries, compared to 2.6 people in 100'000 per capita being killed by interpersonal or collective violence in high income countries (WHO, 2015). On institutional determinants of poverty across countries vid. North (1990), Acemoglu et al. (2005, 2009); on evidence of emerging infectious disease outbreaks increasing mostly in the developing world vid. Jones et al. (2008).

[3]The Plague in 16th century Europe created an opportunity to develop better institutions, through its effect on raising the salience of public goods (Dittmar and Meisenzahl, 2016).

[4]We have identified riots, protests and violence against civilians following the spread of cholera, malaria, Ebola, HIV/AIDS, or unidentified diseases, in Congo, DRC, Kenya, Nigeria, Mozambique, Uganda, Tunisia, Somalia, South Africa, from newspaper reports in the Armed Conflict Location and Event Data Project, 1997-2015.

[5]Examples are provided in the Supplementary Appendix Table B.3.

context of an epidemic and the type of state response that fuels or mitigates this effect is ever more important for policy makers, as infectious disease outbreaks are on the rise in developing regions and greater humanitarian aid is needed to respond to them (Jones et al., 2008).

The main contribution of this paper is to develop strategies to identify both the impact of an epidemic on the likelihood of conflict and precise channels underlying this effect. We also show long-run consequences by studying its impacts on trust in institutions. We study the state response or emergency assistance and provide new evidence on drivers of civil violence. We do this in the context of the Ebola epidemic in Western Africa in 2014-15, described as the largest, most severe and most complex outbreak in the history of the disease (WHO, 2015). The outbreak was a major shock that generated a great influx of state capacity[6], through foreign aid, and required the adoption of new medical technologies, as well as a change in cultural practices to halt the spread of the disease. Numerous riots, protests and violence against government officials, medical personnel and social workers were reported in newspapers. We exploit detailed data available at weekly and localized level on Ebola infections, conflict events and intervention measures, combined with the precise dynamics of the Ebola Virus Disease (EVD) and the timing of distinct intervention measures to identify the impact of the epidemic on civil violence for different containment efforts in the countries most affected by the epidemic, namely Guinea, Liberia and Sierra Leone. The setting further allows us to track the impact of the epidemic shock on civil violence under varying levels of ethnic diversity, religious beliefs and trust and measure long-run impacts on trust in institutions.

Epidemics affect the relationship between civilians or between civilians and the state. These changes can lead to social unrest. We conjecture that an epidemic in which the state intervenes or is expected to intervene changes citizens' perception of the state and demands from it in at least three ways. First, it leads the state to adopt coercive measures, in order to halt contagion. Second, it generates a demand for public goods, as people need health treatment. Third, it requires a change in cultural practices, such as burial practices, and these are induced by state authorities. We hypothesize that these changes are important drivers of civil violence targeted against institutional authorities in the context of an epidemic, and whether they are perceived as threats and ultimately lead to civil conflict depends on beliefs, trust in institutions and the

[6]Including financial resources, medical technology and infrastructure and military aid, coordinated under the auspices of the World Health Organization (WHO). The total worth of direct and in-kind contributions to WHO for the Ebola response was US$459 million from over 60 donors between March 2014 and 22 April 2016, `www.who.int/csr/disease/ebola/funding/en` updated April 2016. We see this as a permanent shock in state capacity, since it led to an improved public health system that is now ready to contain future Ebola outbreaks. Moreover, community responses were key in containing the epidemic and this experience can have permanent effects on containing future disease outbreaks.

response of the state. Epidemics also tear families apart and affect the relationship between citizens, fearing contagion. In this paper we highlight the importance of institutional channels, those that are influenced by policy makers, through the choice of a particular emergency response. These mechanisms mean that epidemics are more likely to lead to civil conflict in weak institutional settings[7]. Moreover, they can deepen mistrust in institutions, therefore further weakening the state.

We test our hypotheses empirically and approach this by combining several data sources. The explanatory variable, the number of Ebola infections, are patient records from the World Health Organisation (WHO) and the National Ebola Response Ebola Center (NERC) in Sierra Leone. This data was collected in Guinea for a related paper (Gonzalez-Torres, 2017) and for Sierra Leone it was shared by Fang et al. (2016). For Liberia we use publicly available data and additional data scraped from Situation Reports from the Liberian Ministry of Health. Our outcome variable are riots, protests and violence against civilians that are large enough to be recorded in local, national and international newspapers, collected by the Armed Conflict Location and Event Data Project[8]. The intervention measures were published during the Ebola outbreak to facilitate containment efforts by the United Nations Office for the Coordination of Humanitarian Affairs (UNOCHA), the Red Cross and the WHO. We also use two rounds of Afrobarometer survey data, pre and post-epidemic.

We use two main sources of variation to identify the impact of the epidemic on conflict incidence. First, the epidemic had a clear beginning, in December 2013, and a clear end, in April 2016. The timing and location of the first human being infected with the virus in Guinea is an exogenous shock, created by the very unlikely event of animal-to-human transmission. The geographic spread throughout the region, prior to significant control efforts is largely due to pre-existing road and trade networks, mobility patterns and population density. Despite enormous efforts by Medecins Sans Frontieres (MSF) to control the epidemic, their capacity was largely overwhelmed by the speed of the disease spreading throughout the region until the international community committed a large amount of resources eight to nine months after the patient zero was infected at the borders in the intersection of three countries. We exploit this in a difference-in-difference design to look at the overall impact of the epidemic on conflict incidence. We look at the change in civil violence taking place before and after the start of the epidemic, comparing

[7]With low access to public goods, low trust in leaders and settings in which state coercion is perceived as illegitimate.

[8]We also did the same exercise using as outcome variable social resistance data, which was scraped from Situation Reports during the Ebola outbreak collected in Guinea for a related paper (Gonzalez-Torres, 2017). We also find a positive impact of Ebola infections on social resistance using this measure. At this stage they are omitted from the paper.

places hit by Ebola with different levels of intensity measured by the cumulative number of cases at the end of the outbreak. The identification relies on a parallel trends assumption and the exogeneity of the first index case. We strengthen our identification to address the possibility of ex-post selection into treatment by instrumenting the end-level of Ebola in a given location with the geographic distance to the first index case.

Second, the virus spread from human-to-human through body fluids under precise dynamics that we know from the epidemic models and that we observe at weekly level for a given location. We use this high-frequency panel to identify the impacts of new infections on riots in subsequent weeks, conditional on time, location and month per region fixed effects[9]. Identification relies on the arrival of new cases being random with respect to conflict, conditional on fixed characteristics and past incidence. We use two instrumental variables strategies to strengthen our identification. First, we instrument the number of new infections in a given location by the turning on and off of the epidemic in neighboring locations, to ensure that our findings are not driven by time-varying confounders with persistent effects in a given location. Secondly, we construct a predicted Ebola measure from the medical literature that relies on the geographic position of each location, infections several periods in the past and fixed characteristics, with the aim to address the possibility of non-standard measurement error in infections.

To study the role of the state response in determining civil violence, we exploit an exogenous shock in state capacity, following the emergency assistance package. It included military aid, new medical technologies and improved public health systems[10]. This gives us precise timing and location of containment efforts that allow us to study the role of perceived state coercion and demand for public goods in generating civil conflict. The impact of military district quarantines is studied in a difference-in-difference setting relying on the timing being independent of predicted conflict and on a parallel trends assumption[11]. The role of demand for public goods is measured by looking at the differential impact of new Ebola infections on conflict incidence for locations with varying levels of demand for public goods, before and after their implementation[12].

[9] This is robust to controlling for infections in contiguous weeks, cumulative infections or past conflict incidence.

[10] We see this as a permanent shock in state capacity, since the public surveillance and response system that was put in place in West Africa is now ready to contain future outbreaks. Moreover, community responses were key in containing the epidemic and this required mobilizing local communities, as well as learning, halting the spread of rumors and changing burial practices, salutation and improving hygiene. This experience can have permanent effects on containing future disease outbreaks.

[11] We provide evidence that this is the case by showing parallel trends prior to their implementation. We also restrict our sample to ever quarantined districts in an event study. The results are robust to conditioning on areas with sufficient level of epidemic incidence.

[12] The decision of the international community to intervene serves as a timing event that is exogenous with respect to the local spread of Ebola and conflict incidence. The end-level of public good provision is a measure of the ideal level of health centers if the great amount of financial aid was available at the beginning of the outbreak. We use this to study the impact of new infections on conflict incidence for varying levels of public good provision at the end of the outbreak, before and after the arrival of aid.

The role of trust and beliefs in influencing the likelihood of civil violence in the context of an epidemic is studied by looking at the impact of new infections on conflict incidence for areas with varying levels of trust, religiosity, ethnic diversity and other expected correlates of civil violence. We hypothesize that religious beliefs are a predictor of civil violence in the context of an epidemic, as it requires a change in cultural practices, which is more costly these groups.

Finally, we study the effect of the epidemic on trust in institutions. We compare trust levels before and after the Ebola outbreak for locations with different levels of Ebola incidence and we also study how this varies by religiosity[13]. If changes in cultural practices are a driver of civil violence in the context of the epidemic, we expect trust in institutional authorities to drop especially for communities with strong religious beliefs.

The results show that one new Ebola infection in 100'000 per capita increases the likelihood of conflict in the next period by 10% in a given location[14], from a baseline mean incidence of 0.013 at two-week level, for a given location[15]. Our estimates mean that moving from no cases to the average Ebola incidence for locations that were hit by the epidemic in the first year doubles conflict incidence in a matter of weeks[16]. The type of conflict that arises is subversive violence, since the object of attack are institutional and medical authorities.

The impacts of this epidemic shock on civil violence are localized and its biggest impacts occur at the peak of the international intervention. Military district quarantines have a large impact on increasing the likelihood of riots and protests, beyond the impact of new infections. Prior to the arrival of a significant amount of emergency assistance, areas with more end-level of public goods have higher conflict incidence as a consequence of new Ebola cases, and the opposite effect thereafter[17]. The results indicate that civil conflict is fueled by perceived state coercion and demand for public goods. Lower trust in leaders and strong religious beliefs makes civil violence more likely to arise as a consequence of new Ebola cases[18].

The epidemic further deepens mistrust in institutional authorities. Two years after the outbreak there are lower levels of trust across measures compared to pre-epidemic levels. In particular the epidemic led to lower trust levels for locations that were hardest hit by the epidemic, especially for strong religious communities, which face larger costs of cultural adaptation.

[13] We study religiosity as measured prior to the outbreak.

[14] Conflict is measured as the number of conflicts in 100'000 per capita in a given location. Ebola is measured at county, chiefdom and sub-prefecture level and conflict is measured at district, chiefdom and sub-prefecture level, for Liberia, Sierra Leone and Guinea, respectively.

[15] In our tables we look at the incidence of conflict in one million per capita to let the reading of the tables be easier, so the baseline incidence in that case is 0.13.

[16] The average Ebola incidence for locations that were already hit in 2014 is 8 cases in 100'000 per capita.

[17] This holds when controlling for cumulative cases or allowing for heterogeneous effects for locations with varying end-level Ebola.

[18] Areas with strong religious beliefs do not have higher rates of civil violence at baseline.

Literature

We seek to contribute to several sets of literature. This paper adds to the empirical literature on the determinants of conflict, thoroughly reviewed by Blattman and Miguel (2010)[19]. We contribute to recent evidence studying the impact of endemic diseases, pathogens and infectious diseases on conflict incidence. Cervellati et al. (2016) provide first evidence of vector-borne endemic diseases as drivers of civil conflict[20], using cross country variation in exposure to human pathogens. They suggest that the impact of endemic diseases on conflict incidence is a direct effect of health. Cervellati et al. (2017, 2018) investigate the effect of malaria on civil conflict in the African continent using data at sub-national grid-cells, studying long-term impacts, as well as effects at monthly frequency exploiting variation in climatic conditions favorable for the transmission of malaria[21]. They suggest a health shock and a negative income shock as potential mechanisms and find exploratory evidence on the role of anti-malarial policies in lowering conflict incidence. This paper contributes to the evidence on the impacts of infectious diseases on civil conflict, methodologically, as well as more deeply by providing a novel mechanism underlying this effect. Ebola is a virus, does not require a vector of transmission and it is not driven by climatic variation[22]. In this way we isolate the impact of an epidemic from climate shocks driving conflict (Hsiang et al., 2011, 2013, Harari and La Ferrara, 2013). The Ebola epidemic is sufficiently large to generate social distress, but it did not affect the population size significantly[23]. In our context we are therefore able to rule out the potential effect of the epidemic acting through population changes (Acemoglu et al., 2017). Most importantly, the Ebola epidemic provides a unique opportunity to study a new epidemic to a large region[24], track it from the first index case to the last contagion, study the role of the state response to it and provide precise mechanisms linking

[19]This paper is most related to a large literature studying economic determinants of conflict (Fearon and Laitin, 2003, Miguel et al., 2004, Bellows and Miguel, 2006, Burke et al., 2009, Brückner and Ciccone, 2010, Bazzi and Blattman, 2014), as well as a literature studying the role of ethnic diversity as a correlate of civil violence, collective action and the provision of public goods (Easterly and Levine, 1997, Posner, 2004, Habyarimana et al., 2007, Esteban and Ray, 2008, Eifert et al., 2010, Glennerster et al., 2013).

[20]In some cases they are recurrently epidemic, such as malaria in some regions. Vector-borne diseases require vectors of transmission, which are living organisms that can transmit infectious diseases between humans or from animals to humans; for example mosquitoes.

[21]They exploit the fact that the specific features of the malaria epidemiology imply temporary spikes in malaria transmission risk that are related to weather conditions and confined in time and space.

[22]Humidity can help its transmission, but the spread of the virus is driven by the contact with the body fluids of an infected person. After the first index case from animal-to-human, the spread is exclusively through to human-to-human contact and is largely driven by proximity to the epicenter

[23]The deceased population is 0.05% of the total population in the three most affected countries over 2-3 years, compared to an annual population death rate of 1% in Guinea and Sierra Leone or 0.7% in Liberia (CIA World Factbook, 2017). The total cumulative number of Ebola infections was 0.13% of the total population, and 2% is the maximum cumulative percentage of infected people in one location over the 2-3 years.

[24]The three most affected countries in West Africa comprise a total population of 23 million people. Discovered in the 1970s, the Ebola virus has caused around twenty outbreaks to date, all in Africa, but this was its first time turning into an epidemic and it was the first time it hit West Africa, therefore constituting an unknown disease to the population affected.

the impact of a major epidemic outbreak to civil conflict. We suggest that epidemics trigger civil conflict as they affect the relationship between civilians or between civilians and the state. In the case of the Ebola outbreak the major influx of emergency assistance allows us to study the role of the state response in fueling civil violence. In particular, we provide evidence of perceived state coercion and demand for public goods as drivers of subversive violence, contributing to the discussion on the role of state capacity as a determinant of conflict (Fearon and Laitin, 2003). This further suggests that perceived state coercion and little sensitivity to local customs is a complementary explanation to the impact of humanitarian aid on civil conflict, other than aid stealing by armed groups (Nunn and Qian, 2014). We also highlight the importance of religious beliefs in triggering civil violence in the context of an epidemic, due to a greater cost of changing cultural practices. This is consistent with the role of religious beliefs in spreading rumors or exerting violence (Miguel, 2005). Finally, we show that the epidemic lowers trust in institutional authorities, especially for religious communities. This provides further evidence of epidemics triggering civil conflict through their impact on changing citizens' perception of the state and adds to the evidence on determinants of trust (Nunn and Wantchekon, 2011).

Secondly, this work contributes to a literature on the role of the spread of diseases for social and institutional change (Acemoglu and Robinson, 2001, Acemoglu et al., 2003, Young, 2005, Alsan, 2014). We zoom into the short-run dynamics of institutional change driven by an epidemic. Dittmar and Meisenzahl (2016) find that Plague outbreaks in 16th century Europe shifted local politics in a few years, creating salience for public goods, and this paper increases our understanding of this process in the short-run, showing how riots and protests might arise immediately to demand public goods. This is related to work on protests and institutional change that finds an effect of changing beliefs on protests and collective action (Barbera and Jackson, 2016, Cantoni et al., 2017). Our findings are consistent with evidence of low health uptake in developing contexts (Dupas, 2011, Greenwood et al., 2013, Alsan and Wanamaker, 2016) and resistance to adopting new medical technologies (Caprettini and Voth, 2017). By studying the behavioral response to emergency assistance, we contribute to evidence of institutional determinants of the spread of infectious diseases (Adda, 2016, Morse et al., 2016).

Finally, this paper contributes to a long discussion among historians, on the role of epidemics as social toxins. Historians have noted two distinct types of civil violence that emerged as a consequence of epidemic outbreaks. Some epidemics have led to violence against civilians, such as ethnic violence or targetted at victims of the disease, notoriously the Black death in 14th century Europe and to some extent also later Plagues, the US smallpox epidemic or the HIV/AIDS epidemic in the early 20th century (Cohn, 2016). Epidemics in such contexts have

sparked ethnic violence that lasted for centuries through cultural persistence (Voigtländer and Voth, 2012). Other epidemics have led to subversive violence, targetting government authorities, medical personnel and social workers. This is the case of the Ebola outbreak in Western Africa, there is ample historical evidence of similar violence arising during epidemic outbreaks in Europe, Russia or North America[25], and is recurrent throughout epidemic outbreaks in the African continent today[26]. Evans (1988) notes that "the general coincidence of cholera epidemics with years of upheaval and revolution has proved too obvious to ignore", however the direction of causality remains to be demonstrated. This paper gives proof of an epidemic leading to subversive violence and precise channels underlying this effect[27]. We provide empirical evidence of institutional mechanisms linking epidemics to civil violence in weak institutional settings and its long-run effects on trust.

The paper is structured as follows. In the following Section 2, we provide a brief background on the Ebola outbreak. The data sources are described in Section 3. The empirical set-up and results are presented in Section 4. First, we search for causal evidence linking the epidemic to civil violence, Subsection 4.1. Second, we provide empirical evidence on drivers of civil violence, Subsection 4.2. Third, we show long-run impacts of the epidemic on trust, Subsection 4.3. We conclude in Section 5.

2 Background

The Ebola virus disease (EVD) is a severe disease with a fatality rate varying from $25-90\%$ at different stages of the outbreak. The virus is transmitted by physical contact with the blood, organs, secrecations, or other body fluids of infected humans or animals, such as fruit bats or primates, as well as infected objects, such as needles and syringes. The disease is characterised by initial flu-like symptoms, which rapidly progress into vomiting, diarrhea, stomach pain and haemorrhage[28]. The incubation period, i.e. the time from infection with the virus to the onset of symptoms, is estimated at an average of 8-12 days in the 2014 Western African Ebola outbreak (Van Kerkhove et al., 2015), but it can potentially take up to 21 days. The virus can only be detected after symptoms arise, even in the laboratory, and it is hard to detect at

[25]Cholera riots similar to the Ebola riots in Western Africa were the norm during the cholera outbreaks in 19-20th century Europe, North America and Russia. On further historical evidence of epidemics fueling civil violence, vid. Evans (1988), Voigtländer and Voth (2012), Cohn (2016), Richards (2016)

[26]See examples in the Supplementary Appendix Table B.3.

[27]Understanding what makes ethnic rather than subversive violence more likely to arise is an interesting question for future work. We do not have the counter-factual of ethnic violence in the case of the Ebola outbreak. Which type of civil violence arises could depend on the response by the state, as this can affect the salience of the out-group. A relevant theoretical framework is provided by Esteban and Ray (2008, 2011b).

[28]`www.who.int/ebola` or `www.cdc.gov/vhf/ebola`

early stages[29]. Infectiousness increases at later stages, with deceased bodies being the most contagious. Patients die within one or two weeks after onset of symptoms or recover becoming immune. Ebola survivors suffer with persistent medical conditions after recovery, including joint pain, lost of sight, headaches, and other chronic health issues, as well as social stigma.

Discovered in the 1970s, the Ebola virus has caused around twenty outbreaks to date, all in Africa, but this was its first time turning into an epidemic[30]. The 2014 West African Ebola epidemic is the largest in history, causing over over 28,600 infections and over 11,300 deaths, between December 2013 and April 2016[31]. Within less than a year the disease spread through Guinea, Liberia and Sierra Leone, small outbreaks reached Nigeria, Mali and a few cases were exported to Europe and the US. Halting an Ebola outbreak requires a great effort to treat symptomatic individuals, isolate infected people, trace their contacts, ensure safe burials and change population behaviors towards protective habits (Fast et al., 2014). This proved to be especially difficult in the present context of weak state capacity, slow international response, unfamiliarity with the disease and religious or cultural habits that facilitated the spread, especially through traditional burials.

Evidence suggests that the first index case[32] occurred in the Forest region in Guinea in December 2013 at the borders of Liberia and Sierra Leone. Subsequent cases spread exclusively through human-to-human contact. For eight to nine months these countries with very weak health systems and state capacity, tried to deal with the outbreak, with soaring death rates. Medecins Sans Frontieres (MSF), who scaled up their intervention, called it an 'unprecedented Ebola epidemic' already by end of March 2014. It was not until August 2014 that the World Health Organisation (WHO) declared it an 'international public health emergency', followed by financial aid from international donors. By the time international aid reached the affected countries, over 4,000 cases had been confirmed. International aid coordinated by the WHO implemented specialized medical infrastructure, contact tracing, surveillance systems and awareness raising campaigns. The peak of the outbreak due to the effectiveness of the interventions was reached by the end of 2014. The outbreak came to an end mid-2015, except for Guinea, which had a significant amount of cases until end of 2015. New infections still appeared in April 2016,

[29] "Diagnosing Ebola in a person who has been infected for only a few days may be complicated. The early symptoms of Ebola infection are difficult to distinguish from other, more common infectious diseases such as such as malaria, influenza, and typhoid fever. Ebola virus is detected in blood only after onset of symptoms, most notably fever, which accompany the rise in circulating virus, however, it may take up to 3 days after symptoms begin for the virus to reach detectable levels." from Centers for Disease Control and Prevention `http://www.cdc.gov/vhf/ebola/healthcare-us/laboratories/specimens.html`

[30] Definition of *epidemic* : affecting or tending to affect a disproportionately large number of individuals within a population, community, or region at the same time

[31] `http://www.who.int/csr/disease/ebola/en/`, accessed April 1, 2017.

[32] The first contagion to humans is zoonotic, i.e. entering in contact with a reservoir host, such as bats, for instance by eating rare bush meat.

but at that point new medical infrastructure and surveillance systems were in place to avoid another major outbreak and the epidemic was officially declared to an end in the summer of 2016.

Numerous riots, protests and violence erupted to counter medical interventions that were opposed by the civilian population. A few examples of violence during the Ebola outbreak include (Cohn and Kutalek, 2016) :

> "[In Guinea], Macenta on 5 April 2014, urban youth attacked the towns first [Ebola] clinic constructed a week earlier, and threatened fifty or more of the centres personnel. The protesters claimed [Ebola] did not exist or was spread by outsiders [...]. Then on 16 September, at Womey, West Africa experienced its worst [Ebola] atrocity when 8 members of a high-level delegation of doctors, politicians, and journalists were killed and their bodies dumped in a latrine [...]. At Matainkay, east of Freetown, Sierra Leone, on 20 September 2014, villagers assailed health workers while they buried [Ebola] victims, and in December, the Red Cross reported further attacks on their burial teams with damages to their vehicles. In Liberia at Westpoint, a poor township in Monrovia, an angry mob overran a health care facility, brought out all patients isolated there and looted the clinic."

Containment efforts were opposed by the civilian population either because they were coercive, such as military quarantines or forced detainment in Ebola treatment units (ETUs); the intentions of emergency assistance were misunderstood, through the spread of rumors; or they went against people's most fundamental beliefs, surrounding the burial of the deceased family members. This allows us to identify three main sources of distress associated to an epidemic.

The first source of distress is military or police action. Their activity included maintaining checkpoints, patrolling country and locality borders, enforcing village-level and district-wide quarantines, and taking punitive measures against individuals found in violation of government mandates for burials, case reporting, and caregiving[33]. Military district-quarantines, curfews or area blockades, for instance, meant that people could not leave a given area, while knowing that they are surrounded by other people infected with Ebola. The population size of an area blockaded varied. It could affect a village, chiefdom or a whole district[34]. The decision is taken in some cases at government level, sometimes at chiefdom level.

The second source of distress is demand for health treatment, on one hand, and rumors

[33] In some cases security forces were doing required public health actions, in others they showed an abuse of power. In either case, these activities received a variety of responses, from acceptance to outright resistance (Hofman and Au, 2017).

[34] In our dataset the average size of a geographic unit quarantined is 50'000 people.

surrounding it, on the other. Ebola patients need to be isolated to avoid contagion and treatment requires special equipment, including the use of personal protective equipment (PPE) by doctors. This meant that patients could not be treated in hospitals and instead required the establishment of ad-hoc health centers, known as Ebola treatment units (ETUs). According to our interviews with social workers in Guinea, the biggest source of rumors and conspiracy theories was around what was happening inside ETUs[35]. Given that Ebola was a new, unknown disease to the region, that early Ebola symptoms are very similar to other endemic diseases in the region and that people died in ETUs with a very high fatality rate at the beginning at the outbreak, it is not surprising that rumors spread[36]. At later stages of the outbreak, when the public health systems were improved, including the establishment of laboratories that allowed for rapid testing, death rates went down and the benefit of seeking treatment in an ETU became prevalent. In some cases an alternative method emerged, known as Community care centers (CCCs), for first patient care of suspect patients. These were opened spontaneously by the communities and led by traditional caregivers, rather than professional staff.

Third, changes in cultural practices leads to social unrest. Traditional burial practices involved washing the deceased by close family members. Since the Ebola virus disease is most infectious in dead bodies, changing this practice was a first priority. In Liberia cremations were imposed (Richards, 2016) and in the other countries the first attempt was to impose safe burials, which did not allow for traditional or religious customs. The Red Cross and Red Crescent Movement, who were in charge of conducting safe burial practices, received significant violent opposition. In Guinea alone, reported attacks against Red Cross volunteers averaged ten per month in the last six months of 2014[37]. Safe burials were progressively adapted to local customs, such as involving religious leaders, leading to what became known as safe and dignified burials.

Social unrest or resistance against the health interventions, on the other hand, can lead to lower health uptake and affect the spread of the disease. Anthropology studies, medical reports (Moon, 2015) and own-collected evidence from interviewing health personnel, social workers and public officials in Guinea, suggest that informing, persuading and involving communities, religious and local leaders in containment activities was key for the success of the intervention. Gonzalez-Torres (2017) shows that places that had access to a prolonged campaign from local radios designed to stop the spread of Ebola rumors in Guinea had a decrease in social unrest

[35]According to their accounts, an important way to halt the spread of rumors was for family members to come and see what was happening inside the ETUs.

[36]In pure observational probabilistic terms entering an ETU was a death sentence at the start of the outbreak, especially for patients that arrived when symptoms were already advanced.

[37]IFRC, website. The had to use personal protective equipment (PPE) to avoid their own contagion with the virus, which has a very impressive effect, as the whole body is covered and you are not able to see the face of the person.

and a drop in Ebola cases seven months after the start of the campaign, compared to other areas with access to the same campaign from distant communities or to areas with access to national or private radio stations.

3 Data

The explanatory variable, the number of Ebola infections, originates from patient records from the World Health Organisation (WHO) and the National Ebola Response Ebola Center (NERC) in Sierra Leone. The number of Ebola infected cases was collected in Guinea for a related paper (Gonzalez-Torres, 2017) and for Sierra Leone it was shared by Fang et al. (2016). For Liberia we use publicly available data from WHO and additional data scraped from Situation Reports from the Liberian Ministry of Health. Ebola cases can be either suspect, probable or confirmed, depending on the stage at which patients have been identified, based on symptoms or laboratory testing. We exclude suspect cases, since these are cases that have not been evaluated by a clinician[38]. Infections are reported at weekly level and the date corresponds to the actual or estimated date of symptom onset. Hence, these are contagions that necessarily occurred prior to the week of report, most likely in a time window of 1-2 weeks before. We collapse the data to two-week windows in order to take into account the possible time-span from contagion to symptom onset. Cases are recorded at the level of sub-prefecture for Guinea, chiefdom for Sierra Leone and county for Liberia. Our main measure of infections are new Ebola infections in 100'000 per capita in a given geographical unit (507) over two weeks (115) from January 2012 - May 2016.

Our outcome of interest, conflict incidence, is constructed using the Armed Conflict Location and Event Data Project[39] Version 6 - 2015 and Realtime data 2016, which collects data on intergroup conflict from local and international newspapers, coding the exact date, geographic coordinates, type of conflict event, actors involved, number of fatalities, news source and description found in the newspaper[40]. We consider all conflict types, irrespective of the number of fatalities involved. During the sample period, the most common conflict events are intra-state conflict, i.e. civil conflicts, including riots, protests and violence against civilians. We aggregate conflict events at the level of sub-prefecture for Guinea, chiefdom for Sierra Leone and district

[38]This is strategy is consistent with the reporting practice of the WHO, which publish data of confirmed and probable cases only. In addition, for Liberia, our measure of suspect cases might contain errors in timing or double counting, since these are scraped from Situation Reports, published at the time, rather than from the Patient database. The results are qualitatively similar when adding suspect cases.

[39]www.acleddata.com

[40]We also did the same exercise using as outcome variable social resistance data, which was scraped from Situation Reports during the Ebola outbreak collected in Guinea for a related paper (Gonzalez-Torres, 2017). We also find a positive impact of Ebola infections on social resistance using this measure. At this stage they are omitted from the paper.

for Liberia. For Liberia, this is a more disaggregated level than the number of Ebola cases. Since we use population weights in all our specifications, results are unchanged if we aggregate the number of conflicts to county level for Liberia. The advantage to use the lowest possible level of disaggregation, is that we have more variation in pre-determined characteristics to study heterogeneous effects. Our main outcome variable is the number of conflicts in 1'000'000 per capita in a given geographic unit (573) over two weeks (115) from January 2012 - May 2016.

Military district-quarantines and movement restrictions at given dates and locations were collected by the Red Cross and Red Crescent Movement. We consider that a given location is quarantined if the chiefdom or district is quarantined. Given this definition, the average size of a quarantined area in our dataset is 50'000 people, compared to an average population of 40'000 people. The implementation of laboratories and health treatment units, such as Community care centers (CCCs) and Ebola Treatment Units (ETUs) are from the WHO and the United Nations Office for the Coordination of Humanitarian Affairs (UNOCHA). Election dates and results to study the role of political grievances in fueling civil violence were scraped from newspapers. To study the possibility of economic mechanisms being at play we use food price statistics collected by Glennerster et al. (2016).

Population data is taken from the 2014 census in Guinea and Sierra Leone. We projected the 2008 census to estimate the population in 2014 for Liberia, based on a combination of prior population growth rates for each district and the growth rate for Liberia overall from 2008 to 2014. To study heterogeneous effects we use predetermined covariates prior to the start of the outbreak. Household survey data are taken from the Afrobarometer, Rounds 5, 2012-2013 and 6, 2015-2016. We construct averages for each variable by sub-prefecture, chiefdom and district for the respective countries.

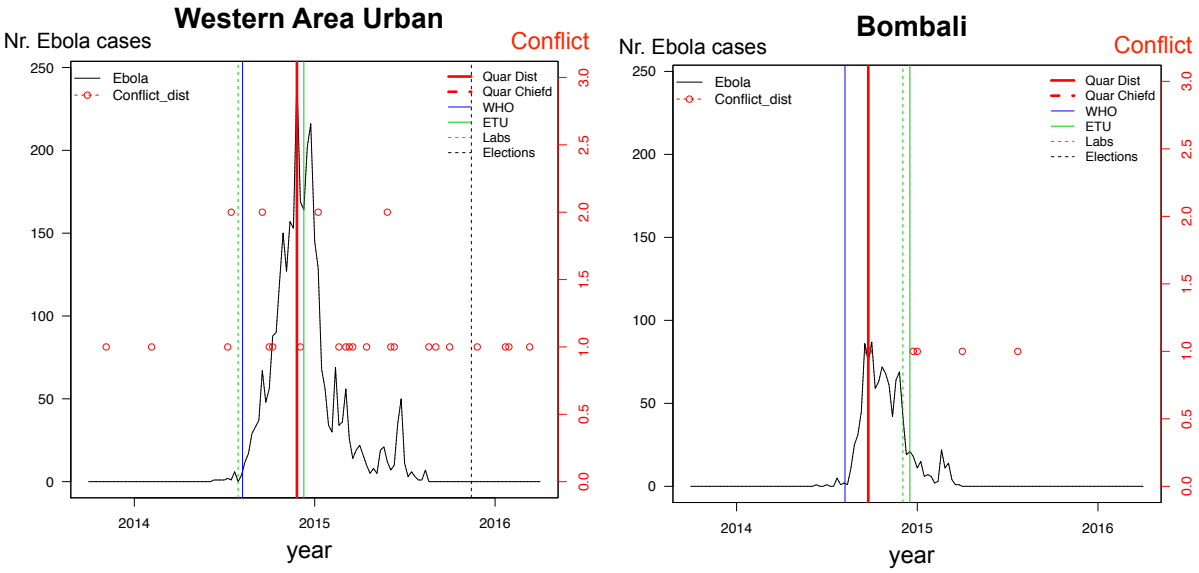
Descriptives

We first give a graphical representation of the data in Figure 1. The Figure plots the timeline of conflict events and number of new Ebola infections per week for two highly populated districts in Sierra Leone. The vertical lines show the timing of the WHO intervention and containment efforts. Across districts in our sample we observe a higher frequency of conflict events at times and districts with more Ebola infections. Oftentimes, a conflict event follows an intervention. Figures for all other district are found in Figures B.1-B.4. The geographic spread of conflict is shown graphically in Figures A.1- A.2.

Descriptives for the aggregate number of Ebola cases are shown in Table 1. Ebola incidence varied widely across the three most affected countries[41]. The first index case occurred through

[41]In this paper we are not able to explain these differences. Population, population density, mobility patterns

Figure 1: Weekly Ebola and Conflict incidence, Sierra Leone



Notes: Weekly timeline of the number of new Ebola infections (left-axis, black line) and of conflict events (right-axis, red dots) for two highly populated districts in Sierra Leone, Western Area Urban (left), and Bombali (right). The vertical lines show the first time the WHO declared Ebola a global health emergency (blue), the imposition of military district or chiefdom quarantines (red), the establishment of Ebola treatment units (ETUs) and of Laboratories (green).

a contact with an animal reservoir of the Ebola Virus, in Guinea in December 2013. We observe the first cases after January 2014 and in the main descriptive statistics the sample is split into the two years prior to the start of the outbreak and the two years and a half since then, January 2012-May 2016.

Descriptives for the aggregate number of conflicts, namely riots, protests and violence against civilians, are shown in Table 2. A simple accounting of conflict incidence in the two years after the start of the outbreak shows a 50% increase in conflict compared to two years prior to the start of the outbreak. This is driven by the countries with highest rates of Ebola cases, namely Liberia and Sierra Leone.

To illustrate the type of conflict events in our data, we show here one such example:

> "Thousands of protesters marched on the main Ebola hospital in Kenema and threatened to burn it down and remove the patients after a rumour spread about "cannabalistic rituals" occurring there; police fired tear gas to disperse the crowd." Kenema, Sierra Leone, *Reuters*, July 2014

and distance to the first case are predictors of the spread of Ebola and epidemiologists have shown that the intervention was key in halting the spread of the outbreak. While the emergency assistance was initially led by Medecins Sans Frontieres (MSF) and then coordinated under the auspices of the WHO in all three countries, there were significant differences in the Ebola response across countries due to distinct national institutions and international donors taking the lead in each response.

Table 1: Descriptives - Aggregate Ebola cases

	Number of Ebola cases				
	Confirmed + Probable	Total incl. Suspect	Deaths	Population	Pop./km^2
Guinea	3,814	3,814	2,544	12.3 mn	41
Liberia	5,044	13,416	4,810	4.4 mn	35
Sierra Leone	8,358	11,903	3,956	6.3 mn	79
TOTAL	17,166	29,133	11,310	23 mn	

Notes Total number of cases: confirmed, probable and suspect cases. In our main analysis we use the sum of confirmed and probable cases, since these have been evaluated by a clinician. WHO-Definitions: *Suspect case:* (1) any person alive or dead, suffering or having suffered from a sudden onset of high fever and having had contact with a suspect case or a dead or sick animal; or (2) with sudden onset of high fever and at least three Ebola symptoms; or (3) with inexplicable bleeding; or (4) with sudden, inexplicable death. *Probable case:* suspect case that has been evaluated by a clinician or with an epidemiological link with a confirmed case. *Confirmed case:* suspect or probable case with a positive laboratory result.

Table 2: Descriptives - Aggregate Conflict events

	Pre-Ebola 2012-2013	During Ebola 2014-2016/5	During Ebola 2014-15	All 2012-2016/5
Number of Conflicts				
Guinea	89	91	73	180
Liberia	72	168	146	240
Sierra Leone	18	53	43	71
TOTAL	179	313	262	491
Likelihood of Conflict per year per obs. unit (573)				
TOTAL	0.15	0.22	0.23	0.19

Notes: Number of conflict events reported in newspapers throughout a given period in each country. Conflict events are riots, protests and violence against civilians.

Our outcome variable is the sum of all such events occurring every two weeks in a given chiefdom. A list of examples on Ebola and non-Ebola related civil violence during the Ebola outbreak is reported in Supplementary Appendix Tables B.1-B.2.

Summary statistics to each of our empirical strategies are provided in Tables A.1-A.4.

4 Empirical strategy

In this Section we search for a causal relationship between epidemics and conflict, Subsection 4.1, precise drivers underlying this effect, Subsection 4.2, and impacts of an epidemic on long-run trust, Subsection 4.3. Robustness checks are given in Subsection 4.4.

4.1 Epidemic spread and civil violence

To identify the effect of Ebola infections on the likelihood of conflict, namely riots, protests and violence against civilians, we use two main sources of variation. The first source of variation comes from the first index case of Ebola, due to the contagion from animal to human being a random and extremely rare event. We exploit this first index case as a random timing event defining our pre-treatment period in a difference-in-difference strategy, as well as geographic variation in total disease incidence. The second source of variation comes from the spread of Ebola at high frequency. We exploit the short-run dynamics of the disease to study the impact of new infections on conflict incidence in a panel at high frequency with location, time and region per month fixed effects.

4.1.1 Difference-in-Differences

In order to study the overall impact of the epidemic on civil violence, we study the change in civil violence after the start of the outbreak, comparing locations hit by Ebola with varying levels of intensity. We do this in a difference-in-difference design with continuous treatment given by the total cumulative number of Ebola cases at the end of the outbreak. The first Ebola case gives us a random timing event defining our pre-treatment period.

$$conflict_{i,\tau} = \beta\, ebolaTotal_i \times PostEpidemic_\tau + \lambda_\tau + \alpha_i + \nu_{i,r,\tau} \tag{1}$$

Equation (1) describes this first identification strategy. $conflict_{i,\tau}$ is the number of conflicts in one million per capita in location i in yearly quarter τ. The cumulative number of Ebola infections in location i measured at the end of the outbreak is given by $ebolaTotal_i$. $PostEpidemic_\tau$ defines the post-treatment period, taking value 1 at the first quarter of the year 2014 and 0 before that[42]. Standard errors are clustered at the level of a location i, or a group of locations, such as regions or districts r, to allow for serial and spatial dependency[43]. The coefficient of interest is β. It measures the change in conflict incidence after the start of the outbreak for one additional Ebola case in 100'000 per capita.

The identification relies on a parallel trends assumption and the exogenous timing of the event. To allow for the effect to vary over time, we use a more flexible specification than (1),

[42]The epidemic started at the end of the last quarter of the year 2013. The first index case is believed to be a child infected on December 26, 2013. We take this into account in our flexible estimates specification. The results do not change if we take December 2013 as the start of the outbreak and in fact, given our flexible specification results, this seems to be a more conservative exercise.

[43]The sample analyzed are 507 locations in the 3 most affected countries, Guinea, Liberia and Sierra Leone. In particular, each location refers to chiefdoms in Sierra Leone, sub-prefectures in Guinea and districts in Liberia. A group of locations or region is a larger unit of analysis, namely districts in Sierra Leone, prefectures in Guinea and counties in Liberia.

using yearly quarter time dummies. This also allows us to provide graphical evidence of parallel trends in conflict incidence for areas with varying levels of Ebola, prior to the start of the outbreak. The specification is shown in equation (2).

$$conflict_{i,\tau} = ebolaTotal_i \sum_{\tau=2012:2}^{2016:1} \beta_\tau \; I^\tau + \lambda_\tau + \alpha_i + \nu_{i,r,\tau} \tag{2}$$

Compared to the previous equation, we replaced the post-treatment dummy with several time dummies I^τ for each yearly quarter, ranging from 2012 to 2016, with the first quarter of 2012 as omitted category. The coefficient β_τ gives us the difference in conflict incidence at each quarter of a year due to one additional Ebola case in 100'000 per capita.

A potential concern is the possibility that post-treatment selection into high Ebola incidence is driven by factors that also change conflict incidence over time. Most of the changes occurring in the months after the first index case are driven by the epidemic and we interpret them as channels underling the impact of an epidemic on civil violence. For instance, an unequal distribution of new health centers or military aid can affect both Ebola incidence and conflict events. This is one of the main channels we stress in this paper driving the impact of an epidemic on civil violence. To address the possibility of time-varying conditions affecting both the spread of Ebola and conflict incidence, such as movement of police forces unrelated to the epidemic, we instrument the end-level of Ebola in each location with the geographic distance to the first index case, also known as epicenter. In particular we use the linear and quadratic geographic distance interacted with the post-treatment dummy and an indicator variable for each country, to allow for non-linear effects in distance and heterogeneity across countries[44]. Geographic distance measures to the first index case are a predictor of the total cumulative number of Ebola infections and it is used in other contexts to study impacts of epidemics, such as (Oster, 2005) for the HIV/AIDS epidemic. The exclusion restriction is that time-varying unobservables arising after the start of the outbreak are not driven by the distance to the epicenter. We show that the incidence of conflict prior to the start of the outbreak is uncorrelated with distance to the

[44]We add the first stage shown in equation (3) to our equation (1) above.

$$\begin{aligned} ebolaTotal_i \times PostEpidemic_\tau = & \sum_{c=Country} \gamma^c \times I(c) \times DistEpicenter_i \times PostEpidemic_\tau \\ & + \sum_{c=Country} \gamma^c \times I(c) \times DistEpicenter_i^2 \times PostEpidemic_\tau + \lambda_\tau + \alpha_i + \nu_{i,r,\tau} \end{aligned} \tag{3}$$

Where $DistEpicenter_i, DistEpicenter_i^2$ are the linear and quadratic geographic distance to the epicenter. Note that there are only three countries. The quadratic term allows for non-linear effects in distance and provides a more flexible specification. The indicator variable for each country, $I(c)$, allows the effect to vary for each country. Other specifications, such as using only the linear distance interacted with each country and the capital lead to similar predictions. Using the simple distance is not predictive of the total cumulative number of Ebola cases, which suggests that there are non-linear effects in distance and heterogeneity across countries.

epicenter, Table A.6. Expected correlates of civil violence that appear statistically associated with the distance to the epicenter are added as controls, interacted with the post-treatment dummies.

Results on the impact of the epidemic over yearly quarters

Descriptive statistics in yearly quarters are shown in Table A.1. The difference-in-difference results are shown in Table 3 for all countries. Columns (1) and (2) show no differential trend in conflict incidence prior to the start of the outbreak in areas with higher Ebola incidence in comparison to areas with lower incidence.

Table 3: Difference in Differences relative to the first index case in West Africa

Outcome: conflict(quarter)	Pre-Ebola		Pre/Post Ebola			
	(1) OLS	(2) 2SLS	(3) OLS	(4) 2SLS	(5) 2SLS	(6) 2SLS
ebolaTot × Trend	0.0001	-0.0001				
	(0.0001)	(0.0002)				
ebolaTot × PostEbola			0.0055***	0.0089**	0.0100**	0.0092*
			(0.0017)	(0.0036)	(0.0040)	(0.0043)
N	4672	4672	10512	10512	3870	3870
Time FE	Y	Y	Y	Y	Y	Y
Chiefd FE	Y	Y	Y	Y	Y	Y
Controls					Restr.	Y

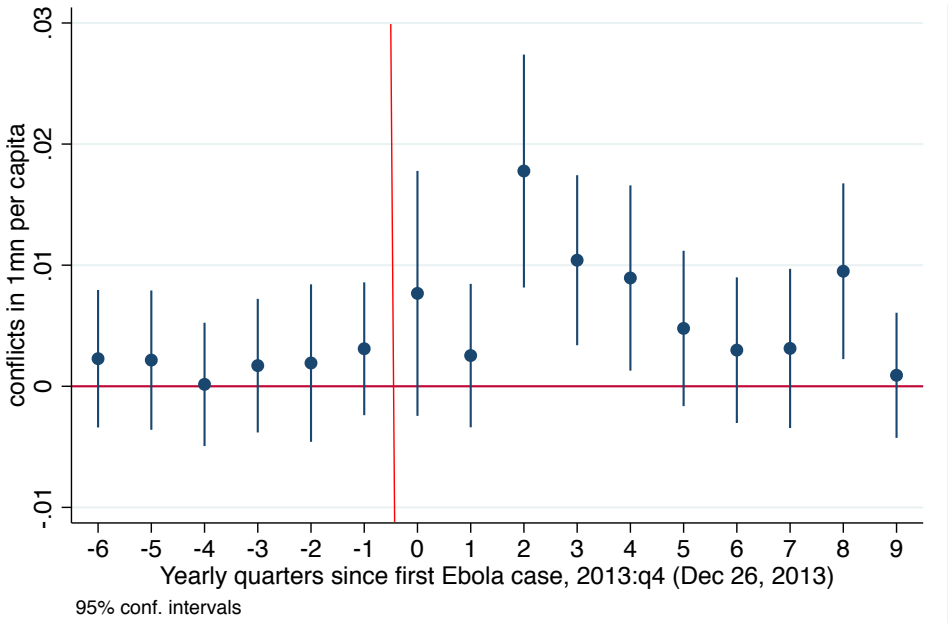
(Clustered SE) by Dist; Controls Restr.: sample restricted to locations with household survey data.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: *conflict* is the number of conflicts in each yearly quarter in 1 million per capita. *ebolaTotal* is the total cumulative number of Ebola cases measured at the end of the outbreak for each location in 100'000 per capita. *PostEbola* is a post-treatment dummy taking value 1 from 2014 on, after the first Ebola case is observed.
2SLS results use the geographic linear and square distance to the first index case as instrument for the total cumulative number of Ebola infections. First stage F-Statistic: 10.24, Table A.5.
Controls: Traditional religion, trust in leaders and votes or preferences for the incumbent measured pre-epidemic and interacted with the post-treatment dummy. The choice of these controls is based on the fact that they are correlated with the geographic linear and square distance to the epidemic, potentially affecting the likelihood of conflict, Table A.6.

The difference-in-difference results are reported in Columns (3)-(5). They indicate that one additional Ebola case in 100'000 per capita increases the likelihood of civil violence by $0.006-0.01$ or by $0.8-1.3\%$ from a baseline mean (standard deviation) incidence of conflict in one million per capita of 0.76 (8.64) over a quarter of a year. With a mean incidence of 50 Ebola cases in 100'000 per capita this means that a location moving from no cases to the average level of infections increases the likelihood of conflict by $40-66\%$ or by $0.3-0.5$ additional conflict events in one million people per capita within three months. In some locations the Ebola incidence was as high as 2105 in 100'000 per capita. The 2SLS results are similar in magnitude compared to the

OLS results. The slightly larger coefficient is plausibly due to measurement error in the number of infections[45]. The first stage is shown in Table A.5. Areas that are closer to the epicenter have higher Ebola incidence, but there is a non-linear effect in distance. Adding pre-determined covariates expected to be correlates of civil violence, interacted with the post-treatment dummy does not change our coefficient, compared to the sample restricted to locations with household survey data used to construct those covariates[46]. Our preferred specification is the simple OLS, given that both strategies lead to similar results, that we observe parallel trends in conflict incidence prior to the outbreak and that most unobservables are interpreted as mechanisms driving civil violence in the context of the epidemic.

Figure 2: Difference in Difference relative to first case in West Africa - All countries



Notes: Coefficients on the total end-number of Ebola cases in one location × dummy for a yearly quarter. Calendar time since first case.

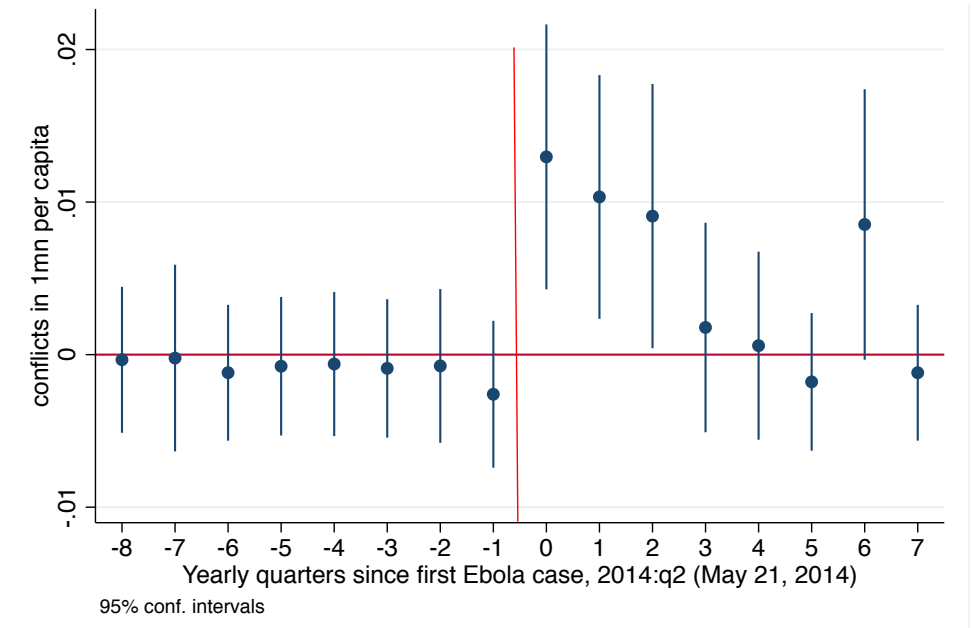
The difference-in-difference design with flexible time coefficients is shown graphically in Figure 2 for all countries and Figure 3 for Sierra Leone. The Appendix Figures A.5-A.6 summarize results for Liberia and Guinea. We find evidence of parallel trends in conflict incidence prior to the start of the outbreak and a spike immediately following the first cases arriving in Liberia, March 2014, and Sierra Leone, May 2014. This does not hold overall for Guinea, which had a

[45]We have patient records for Sierra Leone and Guinea, but for Liberia our measure is from the WHO website and it is aggregated at county level. There was a large number of suspect cases in Liberia that have not been classified yet.

[46]Controls: Traditional religion, trust in leaders and votes or preferences for the incumbent measured pre-epidemic and interacted with the post-treatment dummy. The choice of these controls is based on the fact that they are correlated with the geographic linear and square distance to the epidemic, potentially affecting the likelihood of conflict, Table A.6.

much smaller infection rate as percentage of the population. However, a similar pattern arises for Guinea when we use the arrival of the epidemic in each prefecture as a reference point to study its impact on each sub-prefecture, Figure A.7[47]. The effects for each country indicate an increase in conflict incidence ranging from $0.01 - 0.04$ or $1.3 - 5.2\%$ from a baseline mean incidence of conflict in one million per capita of 0.76 per quarter of a year, with the larger effect representing Liberia[48]. We see the largest impacts during the last three quarters of 2014, namely throughout the core of the outbreak. The effects fade over time, indicating potential non-linearities or a change as the state response evolves over time. We can think of periods 0-2 in Figure 2 as pre-intervention periods. Strictly speaking, the pre-intervention period corresponds to periods 0-1 only, before the epidemic is officially declared in Guinea in March, 2014 following a laboratory test in Lyon, France, that confirmed the Ebola virus. However, MSF was quickly overwhelmed and the states had little capacity to respond to the outbreak until the international aid package started arriving in August 2014, namely period 3. We observe the first military district-quarantines in that month too.

Figure 3: Difference in Difference relative to first case in country - Sierra Leone



Notes: Coefficients on the total end-number of Ebola cases in one location (chiefdom) × dummy for a yearly quarter. Calendar time since first case in Sierra Leone.

[47]We do the same for Sierra Leone, using the first arrival of the epidemic in a district to study its impact on each chiefdom, Figure A.8; but not for Liberia, since we do not have Ebola data at lower than county level.

[48]See interpretation in the paragraph above.

4.1.2 High frequency Panel data

Next we exploit the short-run dynamics of the disease to study the impact of new infections on civil violence at high frequency. The main panel data specification is summarized in equation (4).

$$conflict_{i,t} = \beta\ ebola_{i,t-1} + \mathbf{X}_{i,t-1}\Gamma + \alpha_i + \lambda_t + \mu_{r,\tau} + \epsilon_{i,r,t} \tag{4}$$

The time dimension is at two-week level t. We aggregate our weekly data at that level to reduce possible measurement error in the number of infections due to the improvement of medical and testing conditions changing over time. We choose two weeks because Ebola symptoms arise on average within 8-12 days after infection with the virus. In this specification $conflict_{i,t}$ is the number of conflicts in one million per capita in location i in a two-week period t. $ebola_{i,t-1}$ is the number of new Ebola infections in location i in period $t-1$. In our main specification we do not add any covariates. We will consider basic covariates $\mathbf{X}_{i,t-1}$, namely past infections, cumulative infections and past conflict incidence, for robustness. Our specification conditions on location α_i, time λ_t and month×region fixed effects[49] $\mu_{r,\tau}$. Standard errors are clustered at the level of a group of locations r, to allow for serial and spatial dependency[50]. The coefficient of interest is β. It measures the change in conflict incidence in the next two weeks for one additional Ebola case in 100'000 per capita in the past two weeks.

Our main identification strategy is a simple OLS. We rely on the spread of new Ebola infections in the past two weeks being exogenous at two-week level, conditional on location, time, region×month fixed effects, and possibly other time-varying covariates[51]. Most time-varying confounders we can think of are related to the epidemic and are interpreted as channels. For instance, if the imposition of safe burial practices in areas with more Ebola incidence leads to significant opposition against preventive measures, this could generate new infections in the future and drive conflict incidence. In fact this paper suggests that a state response with little sensitivity to local customs is an important reason explaining why Ebola drives subversive violence.

We discuss a number of alternative strategies in Subsection 4.4 on Robustness checks. In particular we use two instrumental variables strategies to strengthen our identification. First, we instrument the number of new infections in a given location by the turning on and off of the

[49] Note that region is necessarily at a higher level of aggregation than our location. Instead of month we will consider three time periods, corresponding to six weeks.

[50] When we look at each country separately or when we condition our sample to regions for which we have household survey data, we cluster at location level instead, in order to avoid having too few clusters

[51] OLS identifying assumption: $E(\epsilon_{t,i}|ebola_{i,t-j}, X_{i,t-j}, \alpha_i, \lambda_t, \mu_{r,\tau}) = 0$ for all j.

epidemic in neighboring locations, to ensure that our findings are not driven by time-varying confounders with persistent effects in a given location. Second, we construct a predicted Ebola measure from the medical literature that relies on the geographic position of each location, infections several periods in the past and fixed characteristics, with the aim to address the possibility of non-standard measurement error in infections. We also estimate the model by Generalized Method of Moments (GMM), allowing for dynamic effects. The results are similar across specifications and our preferred specification is the simple OLS.

We next motivate the particular the choice of $ebola_{i,t-1}$ at time $t-1$ as our measure of interest. In theory new infections could cause distress in any future time period. We conjecture that Ebola has an impact in matter of few weeks, since people die or recover within four weeks after infection, that is, within two weeks after symptom onset. However, we can answer this question empirically. We estimate the equation above (4) several times for conflict incidence in different time periods in the future, conditional on Ebola and conflict incidence in the past. The logic follows that of a local linear projection proposed by Jordà (2005)[52]. The strategy is summarized in equation (5).

$$conflict_{i,t+h} = \beta_h \, ebola_{i,t} + \sum_{j=1}^{J} \gamma_j \, ebola_{i,t-j} + \sum_{j=1}^{J} \rho_j \, conflict_{i,t-j} + \alpha_i + \lambda_t + \epsilon_{i,r,t} \qquad (5)$$

for $h \in [0, H]$.

The coefficient of interest β_h measures the impact of new infections on civil violence h periods from now, net of other things affecting Ebola or conflict incidence in the past $J = 9$ periods. We plot β_h for each time horizon $h \in [0, 10]$[53]. These plots give us an impulse response function indicating that new infections in 100'000 per capita impact the likelihood of civil violence only in the next period. This provides an empirical justification to our main specification (4).

Results on the impact of the epidemic at high frequency

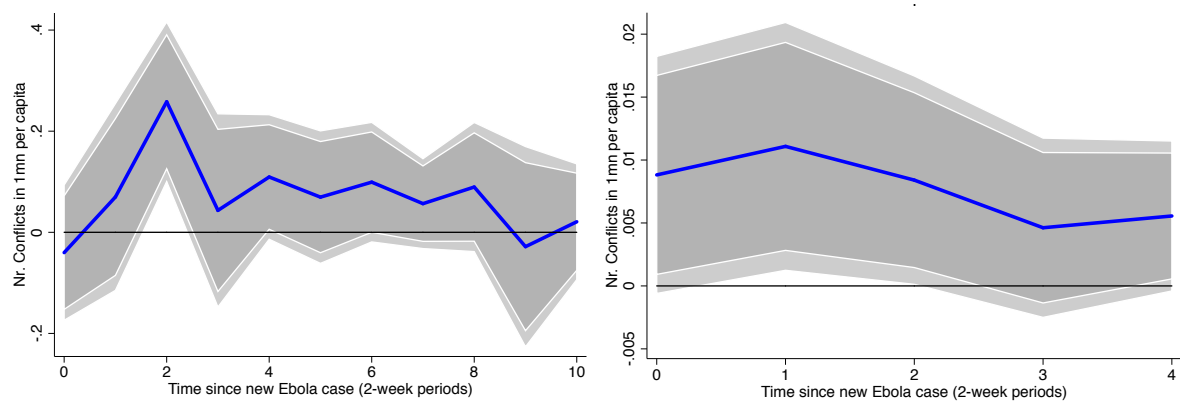
Descriptive statistics for our panel specification are shown in Table A.2.We first give a graphical representation of impulse responses for the extensive margin and intensive margins. The extensive margin tells us whether the presence of the epidemic, $ebola \in \{0, 1\}$, has an impact on civil conflict, Figure 4, left-hand-side. The impacts are concentrated in the two periods after new infections emerge. Locations hit by the epidemic have on average 0.26 more conflict in one million per capita in the following four weeks compared to locations not hit by the epidemic in

[52]This is similar to controlling for different lags in infections, but provides a more complete analysis of the impact of Ebola infections on conflict incidence. We also show results for our baseline specification (4) and controlling for several lags in Ebola, discussed under robustness checks.

[53]We choose $H = 10$ future time periods and $J = 9$ lags and the results are robust to different choices of H, J

that time period. The intensive margin is the impact of new Ebola infections in 100'000 per capita on conflict incidence, Figure 4[54], right-hand-side. The effect is statistically significantly different from zero one period after new infections hit a given location, motivating our specification (4). The figure shows that one new case in 100'000 per capita increases the likelihood of conflict incidence by 0.01 from a baseline mean incidence of 0.117 pre-epidemic or 0.128 for the whole period, thus a $7.8 - 10\%$ increase in the likelihood of conflict incidence in one million per capita. There is suggestive evidence of persistence, but the long-run effects are not statistically significant.

Figure 4: Impulse response for the presence of the epidemic, or extensive margin (LHS), and for the number of Ebola infections (RHS), on conflict incidence.



LHS graph: Impulse response for the presence of the epidemic, or extensive margin, on conflict incidence for 10 future time periods. The coefficients of $ebola_{t-1} \in \{0, 1\}$ in equation (5) are plotted, with 90% and 95% confidence intervals. We condition on 9 lags in ebola and conflict (two-week frequency).
RHS graph: Impulse response for local projections for the impacts of ebola infections in 100'000 per capita (intensive margin) on conflict incidence for 4 future time periods. The coefficients of $ebola_{t-1}$ in equation (5) are plotted, with 90% and 95% confidence intervals. We condition on 9 lags in ebola and conflict (two-week frequency). The effects for the 10 future periods are shown in Figure A.9.

The main results on the impact of new infections on conflict incidence are summarized in Table 4, which includes several specifications discussed in detail Subsection 4.4 under Robustness checks. The epidemic generates a large short-run immediate effect on increasing the likelihood of conflict. Conflict in one million per capita in the next two week rises by $0.012 - 0.013$ in a given location as a consequence of one new infected person in that location[55]. This supposes a $9 - 10\%$ change in the incidence of conflict for one additional Ebola infection in 100'000 per capita, from a baseline incidence of 0.117 pre-epidemic or 0.128 for the whole period. The results

[54] We show the effects for the 10 future periods in the Appendix, Figure A.9

[55] Ebola is measured at county, chiefdom and sub-prefecture level and conflict is measured at district, chiefdom and sub-prefecture level, for Liberia, Sierra Leone and Guinea, respectively. All regressions are population-weighted.

Table 4: High-frequency panel: Summary of Results for All countries

Outcome: conflict(t)	All countries					
	(1) OLS	(2) OLS	(3) OLS	(4) GMM	(5) IV-eb(t-2)	(6) IV-ebOthers(t-2)
ebola(t-1)	0.0138***	0.0146***	0.0123**	0.00756**	0.0162***	0.0238***
	(0.0039)	(0.0041)	(0.0049)	(0.0036)	(0.0047)	(0.0076)
conflictOthers(t-2)						0.0971
						(0.0754)
N	66576	66574	65990	66576	65404	61320
R2	0.125	0.143	0.144		0.145	0.130
Time FE	Y	Y	Y		Y	Y
Chiefd FE	Y	Y	Y		Y	Y
Reg × Month FE		Y	Y		Y	
Clusters	Dist	Dist	Dist		Dist	Dist
Controls						
ebolaCum			Y		Y	
ebola(t-2)			Y			

(Robust SE)

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. $ebolaCum$ is the cumulative number of Ebola infections over time. Column (4) estimates a dynamic panel of conflict on past Ebola using all possible lags as GMM-type instruments for the difference equation. Column (5) uses only $ebola(t-2)$ as instrument for $ebola(t-1)$. Column (6) uses the presence of the epidemic in neighboring locations in periods $\{t-2, t-3, ..., t-10\}$ interacted with country, capital and district-capital as instruments for $ebola(t-2)$. Results using only $\{t-2\}$ as instrument are shown in the Appendix, Table A.11. $conflictOthers(t-2) \in \{0, 1\}$ is the presence of conflict in the region except the own. First stage: $R^2 = 0.21$, $F - Statistic = 84.12$.

are therefore identical to the local projections in which we control for 9 lags in Ebola and conflict. The average number of Ebola infections for locations that were already hit by the epidemic in 2014 is 8 cases in 100'000 per capita in a two-week period. The maximum is 500 cases. These estimates mean that moving from no cases to the average level of Ebola during the first year of the outbreak increases the likelihood of civil violence by 0.112 within the next two weeks, thus doubling the mean incidence of conflict in a matter of weeks. We find only a suggestive impact of cumulative Ebola cases, much smaller in magnitude and less precisely estimated than the impact of new infections, Table A.8. This provides further evidence that the main effects are immediate. The results are robust to different specifications. Robustness checks and details on all specifications are discussed in detail in Subsection 4.4.

4.2 Drivers of civil violence

Once we have established that the epidemic leads to civil violence, in the previous Subsection, we now test our hypotheses on precise channels underlying this effect.

Epidemics affect the relationship between citizens or between citizens and the state. They change citizens' perception of the state and demands from it in at least three ways. First, they move the state to adopt coercive measures, in order to halt contagion. This supposes an immediate threat to citizens, which leads to social unrest. If this coercion is regarded as illegitimate, or excessive, it can increase the benefits from a riot aiming at countering these measures. Second, they generate a demand for public goods, as people need health treatment. This lowers the opportunity cost of engaging in violence, or creates a benefit of doing so if this eventually led to the establishment of treatment centers. Third, they require a change in cultural practices, such as burial practices, which are induced by state authorities. Changing them is more costly for people with strong religious beliefs and the potential benefits of adopting new cultural practices are less clear if there is low trust in institutional authorities. We hypothesize that these changes are important drivers of subversive violence in the context of an epidemic. Epidemics also tear families apart and affect the relationship between citizens, fearing contagion. In this paper we highlight the importance of institutional channels, those that are influenced by policy makers, through the choice of a particular emergency response.

To study these potential channels we first look at the main patterns in our data. What type of conflict was affected by the epidemic, who were the actors involved? We then study how the shock propagates over time and space. We also analyze whether the impacts vary over different time periods corresponding to distinct intervention phases and by country. We interpret results from these exercises as suggestive. Secondly, we study the impact of the state response and emergency assistance that followed. Finally, we study the role of trust and religious beliefs as determinants of civil violence in the context of an epidemic.

4.2.1 Main patterns

We start from the observation that the Ebola outbreak was associated with a particular kind of conflict, namely subversive violence, such as riots, protests and violence against institutional authorities. Tables 5-6 suggest that most conflict events are violent, but also non-violent protests arise, they are directly related to the epidemic outbreak and they involve either civil actors or both civil and state actors, in line with medical records and anthropologists accounts of violence emerging as a consequence both of social unrest and the state response to it.

The impact of an epidemic on violence is very localized, as shown in Table A.17, and spillovers

Table 5: Types of Conflict

	(1) conflict(t)	(2) Viol.a.Civ.	(3) Riots	(4) Protests	(5) Riots/Protests	(6) Viol.	(7) Non-Viol.	(8) EVD/Health	(9) Non-EVD
ebola(t-1)	0.0141***	0.00106	0.00836**	0.00448**	0.0128***	0.00927**	0.00478**	0.0111***	0.00298
	(0.00388)	(0.00104)	(0.00383)	(0.00191)	(0.00387)	(0.00388)	(0.00203)	(0.00406)	(0.00202)
Obs.	65990	65990	65990	65990	65990	65990	65990	65990	65990
R^2	0.144	0.035	0.091	0.082	0.132	0.096	0.088	0.056	0.141

(Clustered SE) by Dist, with Time FE, Chiefd FE, Month x Reg FE; Cond. cumEbola(t-2)

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. We measure different types of conflict events: violence against civilians, riots, protests, riots and protests, violent conflict, non-violent conflict, Ebola or Health-related conflict, Non-Ebola related conflict.

Table 6: Actors in Conflict

	(1) conflict(t)	(2) State	(3) Civilians	(4) State/Civil	(5) CivilOnly	(6) StateOnly
ebola(t-1)	0.0146***	0.00838**	0.0131***	0.00837**	0.00473***	0.0000164
	(0.0041)	(0.00398)	(0.00458)	(0.00398)	(0.00151)	(0.000217)
Observations	66574	66574	66574	66574	66574	66574
R^2	0.143	0.089	0.104	0.084	0.053	0.028

(Clustered SE) by Dist, with Time FE, Chiefd FE, Month x Reg FE

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. A given conflict can have one or several types of actors. At least a state actor, at least civilians, both the state and civilians are involved, only civilians or only state actors are involved.

from neighboring areas are not significantly different from zero, which is in line with the geography of the countries, in which chiefdoms, sub-prefectures and counties or districts are relatively far from each other[56].

We study how the shock evolves over time, according to the different intervention phases. This is shown in Table 7. The results show that the epidemic generates violence in all phases, pre and post-intervention. They provide suggestive evidence of these being largest in August and September 2014, which are the most convulsive months as emergency assistance arrives and a major state response is set in place. The difference is not significantly different from the main effect, when comparing each period separately, column (3). In search for a structural break we study whether the effect changes in August, September or October, columns (4)-(6). We find that the impact of a new Ebola case on conflict incidence is significantly reduced in the post-October period, compared to the period before. This shows that the epidemic generated civil violence at early stages, when the epidemic was unknown, prior to the huge influx of emergency assistance, column (4). It also provides suggestive evidence in favor of numerous accounts by anthropologists and people involved in the Ebola response, that as the state response evolved, taking into account local customs, social unrest was diminished, column (6). A competing explanation is that social unrest subsides after the peak of the epidemic outbreak is reached. In Table A.18 we provide evidence against this competing explanation. The structural break is not driven by the peak of the epidemic, defined as the period in which a region hits the maximum of Ebola cases, column (6). The shock does fade over time column (7). This is consistent with both the state response adapting to people's culture and people's beliefs and customs evolving, a channel shown in (Gonzalez-Torres, 2017) using exogenous variation in exposure to radio campaigns. There are some non-linearities, but they are of small magnitude, for instance including a square term or studying the impact of cumulative number of Ebola cases, Table A.19. In fact, whether there is any Ebola case in a given region, generates a lot of distress, Table A.18, column (5).

The epidemic generates different effects for each country. The impacts are largest for Sierra Leone and a bit smaller for Liberia, in both cases statistically significant, Table A.20. For Guinea the mean effect is small and not statistically significant, as in our difference-in-difference estimates. Guinea is also the country with least number of cases and fatalities and the role of information campaigns in halting social unrest and the spread of Ebola is studied in (Gonzalez-Torres, 2017).

[56]Corresponding to our measures of location for Sierra Leone, Guinea and Liberia, respectively

Table 7: Conflict throughout the outbreak

Outcome: conflict(t)	No omitted cat.		Vs Before			
	(1)	(2)	(3)	(4)	(5)	(6)
ebola(t)	0.0137***		0.0135**	0.0104**	0.0182**	0.0205***
	(0.0043)		(0.0058)	(0.0049)	(0.0071)	(0.0067)
ebola(t) × preJuly2014		0.0135**				
		(0.0058)				
ebola(t) × Aug2014		0.0274**	0.0139			
		(0.0135)	(0.0136)			
ebola(t) × Sep2014		0.0203**	0.00675			
		(0.0091)	(0.0109)			
ebola(t) × postOct2014		0.00594**	-0.00759			-0.0146**
		(0.0025)	(0.0059)			(0.0062)
ebola(t) × postAug2014				0.00380		
				(0.0067)		
ebola(t) × postSep2014					-0.00610	
					(0.0085)	
Mean	0.187	0.187	0.187	0.187	0.187	0.187
N	66576	66576	66576	66576	66576	66576
R2	0.142	0.142	0.142	0.142	0.142	0.142

(Clustered SE) by Dist; Time, Chiefd, Reg × Month FE

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: The period before the huge emergency assistance package corresponds to pre-July 2014. Our prior is that August and September 2014 are the most convulsive months as emergency assistance arrives and a major state response is set in place. This response evolves in the months following October 2014. The epidemic peaks in September in Liberia, in November in Sierra Leone and in December in 2014.

4.2.2 State response

We now study the role of the state response, or emergency assistance, in fueling social unrest. In particular we test whether perceived state coercion and demand for public goods are drivers of civil conflict and study the effect of military district quarantines and the establishment of health treatment centers on conflict incidence. This has direct policy relevance, since they depend on the particular type of emergency assistance that is provided.

Epidemics change the relationship between citizens and the state and the particular state response can in principle dampen or fuel social unrest. Halting the outbreak requires treating patients and preventing further contagion. Some of the measures adopted by the state are coercive, they can are misunderstood, considered illegitimate, or in the context of weak state capacity, they are insufficient, such as limited amount of health facilities.

Perceived state coercion, such as military presence, can lower the probability of success of a mob, discouraging it in the first place. This is a similar argument to the role of military and police resources discouraging insurgency in Fearon and Laitin (2003). However, the role of state coercion in this context can potentially further undermine the state, rather than strengthening it. Quarantines are in general a form of coercion, which could have public benefits[57], but they have private costs. People that are not infected or whose infectiousness is not confirmed with a laboratory result have restricted movement and are often exposed to other infected people. Military district quarantines are an extreme form of state coercion in the context of an epidemic (Moon, 2015, Richards, 2016, Hofman and Au, 2017). With low trust in authorities, this can impact the spread of rumors and low uptake of other protective measures, such as resistance against the imposition of safe burial practices or contact tracing, isolation and patient care.

This means that the opening health treatment centers could lead to a rise in social unrest, due to this previous experience with the state. This is especially the case if people are uninformed about the benefits of treatment centers or if they are in fact very low because there is under-capacity or soaring death rates. The opening of treatment centers should lead to a rise in civil violence in that case. However, the epidemic generates an exogenous demand in public goods, due to the need for health treatment. We should expect a drop after the establishment of treatment centers, if people trust the state, they are informed about the benefits of treatment centers and there is enough access to them. In that case, we should see a drop in riots. This depends in principle on how the state response is perceived, which relies on previous experience with the state. Finally, this can have long-term effects on trust, further undermining weak

[57] Whether they are optimal depends on the type of quarantine and on the citizens response to it. If they lead to civil violence and low health uptake, they can be very counterproductive (Moon, 2015, Richards, 2016, Hofman and Au, 2017).

states.

Empirical strategies to disentangle these channels are discussed next. Descriptive statistics for the state responses we observe are shown in Tables A.3-A.4.

State coercion

To study the role of perceived state coercion as a driver of civil violence we measure the impact of military district-quarantines on civil violence. Our empirical strategies are in the spirit of a difference-in-difference design. First, we look at the change in conflict after a quarantine, comparing locations that have one to those that do not. We restrict the sample to locations that have sufficient level of Ebola incidence, to have comparable locations. Secondly, we look at the conflict incidence during the time of a quarantine in a standard panel specification with location and time fixed effects. Thirdly, we restrict the sample to ever-quarantined districts and compare the incidence in conflict at different points in time for these districts. Finally, we corroborate our results with graphical evidence in an event study design. The main strategy is summarized in equation 6.

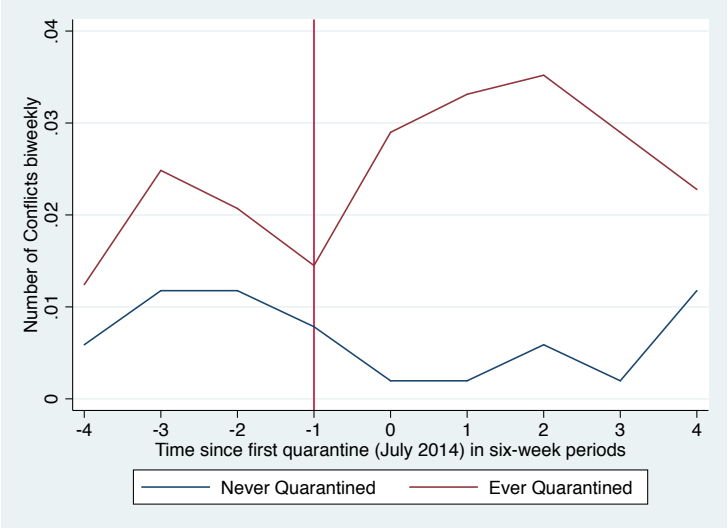
$$conflicts_{i,t} = \gamma_0 PostQuar_{i,t} \times EverQuar_i + \mathbf{X}_{i,t}\Gamma + \alpha_i + \lambda_t + \epsilon_{i,t} \tag{6}$$

The treatment dummy $EverQuar_i$ indicates whether a location i has ever been quarantined. $PostQuar_{i,t}$ is a treatment dummy that takes value 1 if a location i has already been quarantined and 0 otherwise. Note that $EverQuar_i$ is redundant, we add it here for clarification. Only time-varying factors or only location-varying factors are captured by the fixed effects. γ_0 gives us the change in conflict incidence for quarantined locations after they are quarantined, compared to non-quarantined locations. Alternatively we will replace this by a dummy $WithinQuar_{i,t}$ that takes value 1 only if the location i is in a quarantine in at time t. In that case the γ_0 measures the change in conflict incidence for quarantined locations during the quarantine, compared to other periods without the quarantine. We run the simple specification without covariates and then include Ebola as covariate to ensure that the results are not driven by the spread of Ebola.

Identification relies on parallel trends and the assumption that quarantines are set up by the state to stop the epidemic outbreak, not to solve conflict incidence. We provide evidence of no trend differential for quarantined locations prior to the establishment of the quarantine, compared to other locations, Figure 5. Additional graphical evidence for parallel trends and immediate impacts of the quarantine in several event studies using either calendar time since first quarantine or actual time that a location is quarantined are shown in Figures 5-A.15. Table A.21 shows that quarantines are established in locations based on potential disease spread, such

as distance to the epicenter, and access to roads. There is no evidence of strategic placement of quarantines to hurt opposition members or other strategic motives, Table A.22. However, they are placed in locations with low trust in the army, in leaders and in local institutions, hence potentially further exacerbating trust in the long-run.

Figure 5: Parallel trends in Quarantined vs. Non-Quarantined locations



Notes: Conflict incidence for ever-quarantined and never-quarantined locations.

The main results are summarized in Table 8. We restricted the sample to locations that have at least 20 cumulative cases of Ebola in order to have comparable units. There is no significant trend difference prior to the establishment of a quarantine, even after controlling for prior Ebola cases, columns (1)-(2). While more conflictual locations are more likely to have a quarantine, there are parallel trends in conflict incidence prior to their establishment and we see a rise in conflict incidence thereafter. The results indicate a huge rise in civil violence in weeks in which area blockades are in place, compared to other weeks, columns (3)-(4). We see the same effect when looking at the change in conflict incidence for the whole period after the establishment of a quarantine, columns (5)-(6). The point estimates indicate that having a quarantine in place means $0.3 - 0.5$ higher conflict incidence than not having it. This effect is $1.5 - 2.5$ times larger than the effect of being hit by the epidemic in a given week, column (5) in Table A.18. The results also indicate that the impact of new Ebola infections is stable and statistically significant. We provide results restricted to locations that were ever quarantined in Table A.23. The results are identical. Graphical evidence is provided in Figure A.12. It shows the change in conflict incidence over time due to an additional quarantine in 100'000 per capita. We see a spike in conflict during the six weeks following the first time quarantines were imposed, in June/July 2014. Similar strategies using the time of the quarantine in each location show similar results.

Table 8: Military district-quarantines - OLS results (including all locations)

Outcome: conflict(t)	Pre-Quar		Pre/Post-Quar			
	(1)	(2)	(3)	(4)	(5)	(6)
WithinQuar			0.296*	0.468**		
			(0.1540)	(0.1784)		
PostQuar					0.371*	0.508**
					(0.1992)	(0.2227)
PostQuar × Trend	0.00473	0.00595*				
	(0.0028)	(0.0030)				
EverQuar × Trend	-0.000642	0.0127				
	(0.0046)	(0.0247)				
ebola(t-1)		0.00828***		0.00755**		0.00806***
		(0.0027)		(0.0029)		(0.0026)
ebolaCum(t-2)		-0.00143**		-0.00162**		-0.00140**
		(0.0006)		(0.0006)		(0.0006)
N	2218	2000	2218	2000	2218	2000
R2	0.288	0.313	0.287	0.311	0.288	0.312
Time FE	Y	Y	Y	Y	Y	Y
Chiefd FE	Y	Y	Y	Y	Y	Y
cumEbola> 20	Y	Y	Y	Y	Y	Y

(Clustered SE) by Dist

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. $ebolaCum$ is the cumulative number of cases in 100'000 per capita. $WithinQuar$ is a dummy variable taking value 1 if the location is currently under a military quarantine and 0 otherwise. $PostQuar$ takes value 1 if the location has already been quarantined once and 0 otherwise. $EverQuar$ takes value 1 if the location will ever be quarantined and 0 otherwise.

They are shown in Figures A.13-A.14, conditioning on having at least 5 and 10 cumulative Ebola cases. We interpret these results indicating that perceived state coercion generates social unrest, with potential impacts on health uptake of other measures. They show that the immediate threat of state coercion is stronger than its potential benefit from discouraging civil violence.

Public goods

To learn about the role of the demand for public goods as a driver of conflict, we measure the impact of the epidemic on conflict incidence for varying access to Ebola treatment units (ETUs), community care centers (CCCs) and laboratories. Ebola patients were treated in Ebola treatment units (ETUs), which were established first by MSF and then under the auspices of the WHO and created ad-hoc for treatment purposes. Laboratories were set in place for rapid testing of the virus and their establishment rapidly increased with the influx of emergency assistance. They were also decided at national and international level. Community care centers were local hospitals that turned into transit centers for first care of Ebola patients. They were opened

spontaneously by local communities, as opposed to Ebola treatment units and laboratories. We therefore see an opportunity to study the implementation of ETUs and laboratories as a shock to study its impact on civil violence. On the other hand, we will also ask whether conflict incidence throughout the epidemic predicts the amount of CCCs that opened. Since this last exercise is not causal we interpret the results as suggestive.

The role of demand for public goods is measured as a heterogeneous treatment effect of new Ebola cases on conflict incidence. The main specification is summarized in equation (7)

$$\begin{aligned} conflict_{i,t} = {} & \beta_0\, ebola_{i,t-1} + \beta_1\, ebola_{i,t-1} \times PostEmergency_t \\ & + \gamma_0\, NearETU_i^{end} \times PostEmergency_t \\ & + \gamma_1\, ebola_{i,t-1} \times NearETU_i^{end} + \gamma_2\, ebola_{i,t-1} \times NearETU_i^{end} \times PostEmergency_t \\ & + \mathbf{X}_{i,t}\Gamma + \alpha_i + \lambda_t + \epsilon_{i,t} \end{aligned} \tag{7}$$

Table A.21 shows that public goods are established in locations based on potential disease spread, such as distance to the epicenter, population density and access to roads. CCCs are established in locations with prior access to health centers, mechanically. There is no evidence of strategic placement of public goods to benefit members of the incumbent group, Table A.22.

Identification relies on the fact that treatment centers were new, established ad-hoc to counter the spread of Ebola and decided externally by international organizations, with their greatest availability following the influx of money nine months after the start of the outbreak, when the international community realized the epidemic was a global health concern. The decision of the international community to intervene serves as a timing event that is exogenous with respect to the local spread of Ebola and conflict incidence. $PostEmergency_t$ takes value 1 after September 2014. The end-level of public good provision is a measure of the ideal level of health centers if the great amount of financial aid was available at the beginning of the outbreak. This is given by $NearETU_i^{end}$, which is the normalized inverse distance to the closest treatment center ever available to a given location. Our main effects of interest are first γ_1, which measures the impact of new infections on conflict incidence for locations with high potential demand for public goods, before the arrival of the main package. Secondly, γ_2, which gives us the effect for these locations, after the arrival of health centers. We expect $\gamma_1 > 0$ if there is a high demand for those public goods and or there are rumors surrounding the few treatment centers that are present. Moreover, we expect $\gamma_2 < 0$, if the arrival of public goods lowers the incidence of social unrest.

A potential confounder is the epidemic itself. If only locations with a sufficient level of Ebola have conflict incidence, access to treatment centers could be picking up this effect. We therefore control for the cumulative level of Ebola and also add another term to our specification, which would capture differential effects for high and low infected regions, $ebola_{i,t-1} \times HighEbola_i^{end}$ and $ebola_{i,t-1} HighEbola_i^{end} \times PostEmergency_t$, as well as $HighEbola_i^{end} \times PostEmergency_t$ for consistency with a fully specified model.

The main results are summarized in Table 9. There is a lower incidence of conflict due to the epidemic after a significant amount of aid arrives in the ground, column (1). Prior to this moment, the epidemic causes civil violence in locations with a high demand for Ebola Treatment Units (ETUs), column (2). The effect is three times larger for locations with high demand of public goods, than the average effect, 0.037 compared to 0.012. However, after this, there is a drop in civil violence. The estimates are stable when we control for cumulative Ebola cases or allow for heterogeneous effects for high and low incidence areas, Tables A.25-A.26. They are also stable when controlling for other public goods, column (5) in Table 9. The impacts are of similar magnitude and sign for Laboratories, but not statistically significant, column (3). This is consistent with the role of rumors in fueling distress around ETUs before there are enough of them. Moreover, ETUs are for patient care and suppose a more immediate benefit, whereas laboratories are for prevention of future spread, therefore implying benefits that are deferred. Community Care Centers (CCCs) have the opposite sign but are not precisely estimated. These were local hospitals that were spontaneously turned into first patient care for Ebola patients. We interpret this as suggestive evidence, that wherever the state or the international organizations cannot reach, this leads to social unrest and potentially allows people to organize to develop own strategies within the local community (Richards, 2016).

A simple correlational exercise studying whether conflict throughout the epidemic predicts access to ETUs, laboratories or Community care centers, shows that areas with higher conflict incidence during the Ebola outbreak also had higher access to CCCs. As expected, conflict incidence is not predictive of higher ETUs or laboratories, which are established by international organizations with the objective to halt the spread of Ebola. These results are summarized in Table A.27. We interpret the combination of these findings as indicative of subversive violence arising in demand of public goods.

Table 9: Public Goods: Heterogeneous effects with Health Centers

Outcome: conflict(t)	(1) conflict(t)	(2) conflict(t)	(3) conflict(t)	(4) conflict(t)	(5) conflict(t)
ebola(t-1)	0.0220***	0.00214	0.0195**	0.0301	0.0140
	(0.0071)	(0.0039)	(0.0079)	(0.0192)	(0.0098)
ebola(t-1) × PostEmerg	-0.0177***	-0.00343	-0.0157***	-0.0309	-0.0172*
	(0.0059)	(0.0039)	(0.0056)	(0.0194)	(0.0102)
ebola(t-1) × NearETU^{end}		0.0368***			0.0376***
		(0.0052)			(0.0093)
ebola(t-1) × PostEmerg × NearETU^{end}		-0.0209***			-0.0226**
		(0.0055)			(0.0087)
ebola(t-1) × NearLab^{end}			0.0270		0.00133
			(0.0360)		(0.0369)
ebola(t-1) × PostEmerg × NearLab^{end}			-0.0188		0.00789
			(0.0378)		(0.0386)
ebola(t-1) × NearCCC^{end}				-0.0102	-0.0154
				(0.0211)	(0.0099)
ebola(t-1) × PostEmerg × NearCCC^{end}				0.0178	0.0175*
				(0.0209)	(0.0101)
N	36480	33516	33516	36480	33516
R2	0.010	0.013	0.012	0.010	0.014

(Clustered SE) by Dist; Excl. capital; only Epidemic period; Time FE, Chiefd FE, Reg × Month FE
Omitted: ETU*PostEmerg, Lab*PostEmerg, CCC*PostEmerg
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. *ebolaCum* is the cumulative number of cases in 100'000 per capita. $PostEmerg$ is a post-treatment dummy taking value 1 after September 2014, when a great amount of emergency assistance is released. $NearETU_i^{end}$ is the normalized inverse distance to the closest Ebola treatment unit (ETU) ever available to a given location. $NearLab_i^{end}$ is the normalized inverse distance to the closest Laboratory for rapid testing of the Virus ever available to a given location. $NearCCC_i^{end}$ is the normalized inverse distance to the closest Community Care Center (CCC) ever available to a given location.

4.2.3 Trust levels and Religious beliefs

Here we study role of trust and religious beliefs as determinants of civil violence in the context of an epidemic. The epidemic requires a change in cultural practices and the adoption of new medical technologies to halt the spread of an outbreak. This moves institutional authorities to respond and induce a behavior change. Civil disobedience targeted at doctors or social workers, trying to stop the outbreak, suggests not only a role for state coercion and demand for public goods driving civil violence in the context of an epidemic, but also of misinformation or mistrust in authorities. Whether the state succeeds in inducing the required changes to halt the outbreak, depends on costs and benefits of cultural change. The imposition of safe burial practices has a larger cost for communities with strong religious beliefs. The perceived benefits of the change in practices depend on trust in authorities.

We approach this by looking into heterogeneous effects of new Ebola cases on conflict incidence for varying levels of trust or religiosity in a given location[58]. The effect of interest are new infections interacted with each of our covariates. We control for new infections interacted with the distance to the epicenter and square distance in order to diminish the threat that we are capturing differences in cultural traits due to the random event of the first index case arising in a particular location, or differences due to the epidemic generating more distress for locations that were hit first[59]. We construct summary index statistics to group several variables of interest, following Anderson (2008). The covariates are constructed from survey data from the Afrobarometer and measured pre-epidemic and aggregated at the the level of our unit of observation, i.e. chiefdom, district and sub-prefecture for Sierra Leone, Liberia and Guinea, respectively. The results should be interpreted with caution due to the small sample size per cell, namely around 28 surveyed individuals on average each of our locations. We adjust our p-values for multiple inference correction following Anderson (2008), Tables A.31-A.32.

The main results are presented in Table 10. The most robust result across specifications is that locations with low trust in leaders have higher rates of civil violence as a consequence of the epidemic, columns (4)-(5). This holds across specifications and controls. This is consistent with low trust in institutional authorities leading to lower health uptake. We see a positive coefficient on high trust in local institutions in column (4), but this effect depends on the specification. It does not hold when we control for other variables, column (5). Trust in local institutions is potentially correlated with social cohesion in local communities, which can drive riots as a collective action. A lower significance level induced in stronger religiosity and trust in local institutions in column (5) is consistent with this interpretation.

[58]To study heterogeneous effects we start from our baseline high frequency panel specification. We are interested in teasing out patterns in the data, based on pre-existing covariates. Equation (8) summarizes the specification.

$$conflict_{i,t} = \beta_0 ebola_{i,t-1} + ebola_{i,t-1} \times \sum_{k=1}^{K} \beta_k Covariate^k + \gamma_1\, ebola_{i,t-1} \times DistEpic_i + \gamma_2\, ebola_{i,t-1} \times DistEpic_i^2 + \alpha_i + \lambda_t + \mu_{r,\tau} + \epsilon_{i,t} \tag{8}$$

We are interested in β_k, the impact of new infections in areas with a greater share of covariate k. The overall effect of new infections on civil violence is $\sum_{k=0}^{K} \beta_k + \sum_{j=1}^{2} \gamma_j$.

[59]We used distance and square distance as an instrument for the total cumulative number of infections in Subsection 4.1 when looking at the overall effect of the epidemic on conflict incidence, in the difference-in-difference strategy. In this case we are in our high frequency panel specification looking at impacts of new Ebola infections every two weeks, conditional on fixed characteristics. The distance to the epicenter is a fixed characteristic that is predictive of the spread of Ebola in the long-run. The interaction of new Ebola infections with the distance to the epicenter will capture this potential for long-run spread. By controlling for it, we are studying the impact of new Ebola infections on conflict incidence for locations varying under a given dimension, conditional on their potential long-run spread.

Strong religious beliefs are associated with a greater likelihood of civil violence as new Ebola cases emerge, columns (1), (2), (5). These areas are not more violent at baseline, however, Table A.29. There is no difference when looking at differences across the three most prevalent religions in the affected countries. This is consistent with the fact that people from any religion face costs to cultural changes[60].

We also study other expected correlates of civil violence, such as ethnic diversity and potential income channels. Ethnic polarization is associated with a higher likelihood of civil violence, columns (3) and (5) in Table 10. We construct a summary index statistic for ethnic salience grouping questions on ethnic rather than national identification and perceived discrimination of the own ethnic group. We find that ethnic salience has no effect, columns (3) and (5). Note that ethnic fractionalization and polarization are measures of the distribution of ethnic groups and they do not necessarily capture ethnic cleavages[61]. Prior conflicts and political grievances have little explanatory power in this context, as we see from studying prior voting outcomes, expressed political views for the incumbent or the opposition or conflict incidence during the Civil Wars in Liberia and Sierra Leone, columns (3) and (5), or Table A.30. The prize of the riot is a public good, for instance it can defeat a military quarantine. In this case polarization should matter more than fractionalization according to a theory proposed by Esteban and Ray (2011a).

To rule out that possible income channels are driving the main results, we look at the impact of the epidemic on conflict incidence for varying levels of wealth and we also study whether food prices drive our results. The aim of this exercise is to rule out that pre-existing wealth or varying prices are the only drivers of civil violence in the context of an epidemic. The impact of the epidemic on civil violence does not change by the inclusion of contemporaneous food prices, Table A.28. This suggests that changing economic conditions measured (imperfectly) in price levels of two important commodities for food consumption (imported rice and palm oil) are not driving the results, for areas for which we have data on prices. We also study the role of pre-existing infrastructure in interaction with Ebola incidence and see that worse infrastructure is not associated with more civil conflict, Table A.30.

[60]Safe burial practices that were imposed could be more costly for traditional African beliefs, or Muslims, due to the custom of family members washing the deceased, but also cremations, traditionally against Christian views, were imposed in Liberia, which has a Christian majority.

[61]Fractionalization is a Hirschman-Herfindahl index, given by $F = \sum_{i=1}^{m} n_i(1-n_i)$, where m number of groups, (n_i, n_j) size of each group. It ranges from 0, with all people belonging to the same group, to 1, total diversity. The simplest measure of polarization is given by $P = \sum_{i=1}^{m} n_i^2(1-n_i)$. It takes its maximum value when two equally sized groups face each other. They are negatively correlated at low values of fractionalization, not correlated at intermediate values and positively correlated at positive values.

Table 10: Correlates of civil violence

	(1)	(2)	(3)	(4)	(5)
ebola(t)	0.0133**	0.0132**	0.0218***	0.0195***	0.00320
	(0.0053)	(0.0054)	(0.0062)	(0.0031)	(0.0052)
ebola(t-1) × Strongly Relig.	0.00314*	0.00358**			0.00557**
	(0.0016)	(0.0018)			(0.0028)
ebola(t-1) × Tradit. Relig		0.0000767			0.0000973
		(0.0001)			(0.0001)
ebola(t-1) × Ethnic Fractional.			-0.0519**		-0.0333
			(0.0231)		(0.0231)
ebola(t-1) × Ethnic Polariz.			0.0546**		0.0841***
			(0.0256)		(0.0284)
ebola(t-1) × Ethnic Salience			0.00942		0.0108
			(0.0103)		(0.0111)
ebola(t-1) × Trust Leaders				-0.0199**	-0.0323***
				(0.0075)	(0.0106)
ebola(t-1) × Trust Local Instit.				0.0230***	0.0161
				(0.0064)	(0.0101)
ebola(t-1) × Trust President				-0.00479	-0.00706
				(0.0058)	(0.0058)
ebola(t-1) × Trust Opposition				0.00247	-0.00740
				(0.0098)	(0.0126)
ebola(t-1) × Trust Army				-0.0228***	0.00411
				(0.0081)	(0.0126)
ebola(t-1) × Trust People				0.000379	0.00187
				(0.0014)	(0.0015)
Mean	0.0968	0.0968	0.0968	0.0968	0.0968
N	24852	24852	24852	24852	24852
R2	0.0508	0.0508	0.0510	0.0518	0.0528
Time FE	Y	Y	Y	Y	Y
Chiefd FE	Y	Y	Y	Y	Y

(Clustered SE) by Dist; Time, Chiefd; Control: ebola × DistEpic, ebola × DistEpic^2; Excl. capital

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. The covariates are Afrobarometer data measured pre-epidemic and aggregated at the level of our unit of observation, i.e. chiefdom, district and sub-prefecture for Sierra Leone, Liberia and Guinea, respectively. Ethnic salience is a summary index statistic grouping questions on ethnic rather than national identification and perceived discrimination of the own ethnic group. Strongly religious is the perceived importance of religion. Traditional religion is a religion that is a traditional African religion and is neither a branch of Islam nor of Christianity. Trust in people is a summary index statistic grouping trust in neighbors, in other citizens, family members.

4.3 Long-run Impacts on Trust

The epidemic changes the relationship with the state, affects beliefs, and a costly change can potentially undermine trust in institutions. We study the long-term impacts on trust in institutions comparing two rounds of Afrobarometer survey data, pre and post-Epidemic. The effects on long-run trust are studied in a simple difference-in-difference strategy, comparing trust before and after the Ebola outbreak for locations with varying levels of Ebola incidence. If the epidemic affects conflict incidence through a change in citizens' perceptions of the state, we expect a drop in trust levels. We look at the impact of the epidemic on trust for areas with strong religious beliefs in third differences. If cultural change is a major source of social distress, we should be expect a further drop in trust levels for groups with high costs of cultural adaptation.

Results are shown in Table 11. Simple correlational evidence shows that there are lower levels of trust in leaders, in local institutions and in the President after the epidemic, compared to one or two years prior (first row). In difference-in-differences we find that areas that were harder hit by the epidemic experience a drop in trust in the President, column (3). We further look at interaction effects with levels of religiosity as measured prior to the outbreak, Table 12. The epidemic leads to a drop in trust in leaders for areas that are strongly religious, third row in column (1).

A potential implication of these findings is that epidemics affect weak institutional settings through their impact on social unrest and trust levels, especially for groups with significant costs of cultural adaptation. State coercion and weak public good provision exacerbates this effect and since lower trust in authorities is associated with more civil violence, especially among these groups, this widens the cleavage between them and the state authorities.

4.4 Robustness checks

We provide a number of robustness checks on our high-frequency panel specification (4). Firstly, the main concern to our OLS specification is unobserved serial correlation in conflict or the possibility of conflict affecting the future spread of Ebola. We address these concerns with a number of strategies leading to similar results.

In all our specifications we cluster standard errors at a given region or location, allowing for time dependency. For robustness we add cumulative Ebola and past Ebola cases and lagged conflict as control $X_{i,t-j}$ and show that our coefficient of interest β is stable. Given that we have many time periods and that we study the impact of infections, conditional on several lags

Table 11: Trust Levels Pre/Post Ebola

Outcome: Trust in	(1) Leaders	(2) Local Institutions	(3) President	(4) Opposition	(5) Army
PostEbola	-0.730***	-0.206***	-0.203***	0.0378	-0.126**
	(0.153)	(0.044)	(0.057)	(0.052)	(0.055)
EbolaTotal^{pc} × PostEbola	-0.000254	0.0000145	-0.000811**	0.0000942	0.000210
	(0.001)	(0.000)	(0.000)	(0.000)	(0.000)
Mean	2.55	1.47	1.85	1.29	1.73
N	457	457	457	457	457
R2	0.67	0.80	0.81	0.70	0.71

(Clustered SE) by Chiefd*Year; Chiefd FE

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: We compare two rounds of Afrobarometer data, pre and post-Epidemic. $EbolaTotal^{pc}$ is the total cumulative number of Ebola cases measured at the end of the epidemic in 100'000 per capita. *PostEbola* is a post-treatment dummy taking value 1 from 2014 on.

Table 12: Trust Levels Pre/Post Ebola - interaction with Religiosity

Outcome: Trust in	(1) Leaders	(2) Local Institutions	(3) President	(4) Opposition	(5) Army
PostEbola	-0.855***	-0.241***	-0.431***	0.197***	-0.168***
	(0.168)	(0.067)	(0.091)	(0.060)	(0.059)
EbolaTotal^{pc} × PostEbola	0.000536	0.000142	-0.000312	-0.000310	0.0000808
	(0.001)	(0.000)	(0.000)	(0.000)	(0.000)
StronglyRelig × PostEbola	0.244	0.0690	0.439***	-0.307***	0.0814
	(0.290)	(0.093)	(0.106)	(0.084)	(0.108)
EbolaTotal^{pc} × StronglyRelig × PostEbola	-0.00229**	-0.000268	-0.000483	0.000559	0.000783
	(0.001)	(0.000)	(0.001)	(0.001)	(0.001)
Mean	2.55	1.47	1.85	1.29	1.73
N	457	457	457	457	457
R2	0.68	0.80	0.83	0.72	0.72

(Clustered SE) by Chiefd*Year; Chiefd FE

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: We compare two rounds of Afrobarometer data, pre and post-Epidemic. $EbolaTotal^{pc}$ is the total cumulative number of Ebola cases measured at the end of the epidemic in 100'000 per capita. *PostEbola* is a post-treatment dummy taking value 1 from 2014 on.

in our explanatory variable, the inclusion of a lagged dependent variable is unlikely to affect our coefficient of interest significantly[62]. The Jorda local projection specification also addresses these concerns including several lags in Ebola and conflict incidence and clustering standard errors at a given location.

We also run an OLS regression of conflict on Ebola incidence with flexible coefficients on past and future infections to show the full dynamics. The strategy is shown in equation (9).

$$conflict_{i,t} = \sum_{j=-J}^{J} \beta_j \, ebola_{i,t+j} + \alpha_i + \lambda_t + \mu_{r,\tau} + \epsilon_{i,r,t} \tag{9}$$

Since $ebola_{i,t+j}$ for $j \geq 0$ are post-treatment variables affected by our explanatory variable of interest, $ebola_{i,t-1}$, they are bad controls and our coefficient of interest β_{-1} is likely to be biased under this specification. Therefore, our main specification does not include future Ebola incidence. We provide OLS results for equation (9) as a placebo exercise strengthening our results. In particular it allows us to test whether future Ebola is predictive of conflict today, when we condition on past Ebola incidence.

Furthermore, we test whether conflict incidence generates new Ebola infections in the future by estimating equation (10).

$$\begin{aligned} ebola_{i,t} = \rho \, ebola_{i,t-1} + \beta_0 \, conflict_{i,t-1} \\ + \beta_1 \, conflict_{i,t-1} \times ebola_{i,t-1} + \alpha_i + \lambda_t + \mu_{r,\tau} + \epsilon_{i,r,t} \end{aligned} \tag{10}$$

In particular, conflict cannot generate new infections unless there are already Ebola infections, since all infections after the single first index case were through human-to-human transmission. We also know that this needs to occur within the two weeks in which an infected individual has symptoms. Therefore our coefficient of interest is β_1 and we expect β_0 not to be statistically significantly different from zero.

On the other hand, we can also allow for the possibility of dynamic effects estimating a dynamic panel by General Method of Moments (GMM). Causal inference in a dynamic model relies on the weaker assumption that new infections are pre-determined with respect to conflict. That is, while conflict could cause future infections without challenging our identification, the important identifying assumption is that, conditional on controls, conflict is not correlated with new infections in the past, for reasons unrelated to the epidemic. The identifying assumption in a dynamic model with serial correlation in conflict up to $l > 0$ lags is summarized in equation

[62]The limited influence of lagged dependent variables on other covariates of interests in long panels has been shown using Monte Carlo simulations by Judson and Owen (1999), Beck and Katz (2004).

(11). It gives us a long list of moment conditions used to estimate equation (4) by GMM.

$$E(\epsilon_{t,i}|ebola_{i,t-j}, X_{i,t-j}, \alpha_i, \lambda_t, \mu_{r,\tau}) = 0 \text{ for } j \geq l \text{ (but not for } j < l) \tag{11}$$

Secondly, we provide an empirical strategy that addresses the possibility of time-varying unobservables driving the correlation between new infections and conflict incidence within the same location. The number of new infections in a given location is instrumented with the presence of the epidemic in neighboring locations. In particular, our instrument is a dummy variable taking value 1 if there is any case in neighboring chiefdoms and 0 otherwise. We measure this at time $t-2$, a period prior to the Ebola cases of interest and also at times $\{t-2, t-3, ..., t-10\}$, to allow for more flexibility in the impacts changing over time. We allow for the effect to vary for distinct countries, for capitals, capital-districts and other locations. The exclusion restriction is that the presence of the epidemic in neighboring areas does not drive conflict in a given area either than through its impact on the epidemic. We provide evidence to support this hypothesis by showing that there are no spillover effects when looking at the number of Ebola cases in neighboring chiefdoms, conditioning or not for own cases. We add a dummy variable indicating whether there is conflict or not in the region except the own as control in our 2SLS specification. Although unobserved spillovers are always possible, this instrument addresses the possibility that time-varying unobservables in the own location are driving our results.

Thirdly, to address the possibility of non-standard measurement error in infections, we use a second instrumental variables strategy. In particular, we instrument new infections with past infections or with predicted infections from the medical literature. The predicted number of cases is created following Fang et al. (2016)[63]. The model takes into account the location and time of the first index case and the position of each chiefdom in the geographic network, to estimate the parameters determining the rates of infection between distant, neighboring and own chiefdoms in Sierra Leone by Maximum likelihood. Infections follow a Poisson process and a number of covariates enter non-linearly multiplying the exponent of the risk ratio. We modify their code to do out-of-sample predictions in Ebola infections and remove potential confounders. The covariates we include are either fixed over time and measured prior to the start of the outbreak, or only vary over time for the whole country[64]. Our location and time

[63]We thank Fang et al. (2016) for sharing their code and data to replicate the Poisson transmission model in their paper. We modify the code in order to remove potential confounders and to generate out-of-sample predictions in Ebola infections.

[64]The original covariates included in the model are population density, weekly average temperature and relative humidity (varying at national level only), treatment centers, distances to nearest primary roads, secondary roads, railroads, distances to the nearest hospital, coverage percentages of cropland, forest and shrub, poverty level, three broad intervention phases and primary ethnic groups. We remove obvious confounders from the model.

fixed effects would remove their main effect. The identifying variation in this instrument comes from the position in the network of each chiefdom and the arrival of past Ebola infections[65]. We instrument $ebola_{i,t-1}$ in (4) with the resulting measure of predicted ebola cases. Since the exact timing of Ebola infections are hard to predict, even ex-post, we need past Ebola cases to predict future cases. The most conservative predictor is using Ebola infections four weeks in the past to predict current infections. That is, the exclusion restriction of the instrument is a weaker version of the fact that past infections should not affect civil violence in the future either than through its impact on current infections. Given the short-run dynamics of EVD and that we find only immediate impacts of Ebola on future conflict incidence using the actual number of Ebola cases for a variety of specifications, we think that this instrument addresses at least our main concern. Namely it allows us to address measurement error, reverse causality and serial correlation in conflict incidence.

Finally, newspaper bias is a potential problem when working with the widely used ACLED dataset. We think that the high-frequency panel specification significantly reduces the threat of newspaper bias driving the results. In particular, journalists would have to perfectly predict the epidemic spread[66] and move strategically every two weeks into areas with high Ebola incidence, in order to generate a positive correlation between infections from medical records and reported conflict at two-week frequency. We give a full list of newspapers reporting in the two year prior (2012-2013) and during the epidemic (2014-2015), Supplementary Appendix Tables B.4-B.6. There is some turnover of newspapers reporting comparing both periods. Our main empirical strategy addresses this potential concern by exploiting high-frequency variation within locations. Shut-down of newspapers is taken into account with our time fixed effects. Region $\times$ month fixed effects further take out the variation that might arise at regional level at monthly periods.

[65]The model is explained in the Appendix to Fang et al. (2016). We provide here a summary. Let $Y_i(t)$ be number of symptom onsets in chiefdom i during week t. $Z_i(t) = \sum_{d=1}^{D} \omega_d Y_i(t-d)$ are infectious cases. These are cases that have not developed symptoms yet are therefore not able to infect other people. The probability of infectiousness being d, i.e. within 21 days is given by ω_d. The number of Ebola cases we observe are $Y_i(t)$. They follow a Poisson process, $Y_i(t) \sim Poisson(N_i \gamma_i(t))$. Where N_i is population size in chiefdom i and together with $\gamma_i(t)$ gives us the average probability of a new case arriving in chiefdom i at time t. It depends on cases arriving in the own chiefdom i, neighboring chiefdoms j and distant chiefdoms, as well as on the transmission rate and either pre-determined covariates or if time-varying only changing at national level. We remove other covariates. The model used is $\gamma_i(t) = \{\gamma_0 + \gamma_1[Z_i(t) + \theta \sum_{j \in B_i} Z_j(t)]\} e^{\beta' X_i(t)}$. The model is estimated by maximum likelihood. We choose initial values for γ_0^0, γ_1^0, θ^0, and estimate $\hat{\beta}^k$ after k iterations. Given β^k, we maximize the Poisson likelihood, to obtain $\hat{\gamma_0}^k$, $\hat{\gamma_1}^k$, $\hat{\theta}^k$.

[66]This is very hard, even ex-post for epidemiologists.

Results on Robustness Checks

The main results to various specifications discussed above are summarized in Table 4 for all countries. The results are stable across specifications, positive and statistically significant. We have slightly smaller results in our GMM estimation strategy and slightly larger results from the 2SLS specification with neighboring chiefdoms, but similar in magnitude. The point estimates vary from $0.008 - 0.02$, implying a $6 - 15\%$ increase in the likelihood of conflict incidence over the next few weeks due to one additional Ebola case in 100'000 per capita. The reduced form estimates and first stage results using the presence of the epidemic in neighboring locations in the last period only are shown in Table A.11. We find no geographic spillovers, Table A.17.

A simple difference-in-difference strategy exploiting the arrival of the first case in each location and the total end-number of Ebola cases in that location shows an overall effect in conflict incidence of 1% per two-week period for one single additional Ebola case in 100'000 per capita, Table A.10. A similar strategy to the difference-in-difference design exploiting the first case in each country is done at sub-national level in Figure A.8, using the time of arrival in a district and the total Ebola incidence in a given chiefdom[67].

Results for Sierra Leone are given in Table 13, for which we have constructed predicted Ebola infections as instrument following the medical literature (Fang et al., 2016)[68]. The impacts are consistent across specifications, with point estimates varying from $0.010 - 0.017$. The baseline incidence of conflict is much smaller in Sierra Leone and these point estimates imply a $21 - 36\%$ increase in the likelihood of conflict from a baseline mean (standard deviation) incidence throughout the epidemic of 0.047(1.30), or much larger in comparison to pre-epidemic incidence, 0.012(0.50). The first stage fit for column (5), using the predicted Ebola from the medical literature, is shown by plotting the raw data of new Ebola cases and predicted Ebola, Figure A.10.

We interpret the slightly larger 2SLS coefficients found with instruments using distinct types of variation, such as the distance to the epicenter in the difference-in-difference strategy, the presence of the epidemic in neighboring locations and predicted Ebola from the medical literature as an indication that OLS results are biased downwards due to measurement error in the number of infections. Our preferred specification are the OLS results, since they are more conservative estimates and they reflect the impact of confirmed and probable cases on civil violence. As we will discuss in the Section on drivers, the effect of the epidemic on civil violence changes over time and space as the state response evolves. The smaller effect in the GMM strategy could

[67] This is also shown for Guinea, for which we have sub-prefecture level data in Ebola cases, Table A.7, but not for Liberia, for which we only have county-level data in Ebola incidence (14 counties).

[68] We are still working on extending it to Guinea and Liberia

Table 13: High-frequency panel: Summary of Results for Sierra Leone

Outcome: conflict(t)	Sierra Leone				
	(1) OLS	(2) OLS	(3) GMM	(4) IV-eb(t-2)	(5) IV-Pred.eb
ebola(t-1)	0.0141***	0.0128**	0.0102**	0.0153***	0.0172**
	(0.0054)	(0.0052)	(0.0109)	(0.0058)	(0.0081)
N	15369	15218	15369	15218	15369
R2	0.050	0.052		0.050	0.050
Time FE	Y	Y		Y	Y
Chiefd FE	Y	Y		Y	Y
Clusters	Chiefd	Chiefd		Chiefd	Chiefd
Controls					
ebolaCum		Y			

(Robust SE)

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. $ebolaCum$ is the cumulative number of Ebola infections over time. Column (3) estimates a dynamic panel of conflict on past Ebola using all possible lags as GMM-type instruments for the difference equation. Column (4) uses $ebola(t-2)$ as instrument for $ebola(t-1)$. Column (5) uses predicted Ebola following (Fang et al., 2016). The First stage is shown graphically in Figure A.10.

mean that there are feedback effects, in the sense that conflict amplifies Ebola incidence in the future. We will study this now in detail.

Results for a regression including several lags and leads in Ebola, equation (9), are shown in Tables A.12- A.13 and Figure A.11. As mentioned this cannot be our main specification since future cases are bad controls, they are affected by our variable of interest. However, this serves as a placebo exercise to show that reverse causality is not driving our results. They indicate that infections in the past two weeks lead to a rise in civil violence and that conditional on these, leads have no bite. The F-test shows that only past cases are statistically significantly different from zero, while leads are not.

We study the possibility of conflict incidence directly affecting the spread of Ebola, Table A.15. First we show that adding a lag in conflict incidence does not affect our coefficient of interest[69]. Moreover, the lag is not significant and there is no evidence of serial correlation. Results for a Wooldridge (2002) test for autocorrelation in panel data suggest no evidence of

[69]The limited influence of lagged dependent variables on other covariates of interests in long panels has been shown using Monte Carlo simulations by Judson and Owen (1999), Beck and Katz (2004). In particular, without the covariates the bias derived by Nickell (1981) is $plim_{N\to\infty}(\hat{\rho}-\rho) \approx \frac{-(1+\rho)}{T-1}$, with ρ being the relationship between conflict at time t and conflict at time $t-1$. We have $T = 115$ two-week periods and $\hat{\rho} = 0.053$, so the bias in $\hat{\rho}$ is $\frac{-(1+0.053)}{114} \approx -0.009$. This is an upper bound, since the inclusion of covariates necessarily reduces this bias (Nickell, 1981). It affects our coefficient of interest indirectly through the first-stage correlation between lagged conflict and Ebola incidence, estimated to be 0.06. An upper bound to the bias in our coefficient β is $-0.06 \times (-0.009) \approx= 0.00054$ or 4% of our estimated $\hat{\beta} = 0.0127$ in the regression of conflict on ebola incidence with lagged conflict as control. Taking this into account, the lower bound impact of new ebola infections on conflict incidence is 0.0122.

serial correlation in conflict incidence, Table A.14. Second, we test whether the spread of Ebola is affected by conflict incidence, as explained above, equation (10). We find no evidence of this feedback effect, Table A.15.

Finally, our results are robust to using conflict incidence as a dummy variable taking values $\in \{0, 1\}$, as well as using the total number of new Ebola cases including suspect cases, Table A.16.

5 Conclusion

The emergence of infectious diseases has been increasing in the last decades and they are likely to rise in the future as globalization, population growth, environmental degradation and climate change are affecting human societies and the natural environment in ways never experienced before[70]. Extraordinary advances in the medical science have allowed the international community to intervene in such contexts. The perception of disease epidemics as a threat to global security, beyond its impacts on human health,[71] however, shapes the objectives and implementation of emergency assistance. The international community is often accused of intervening too late, once the risks of a pandemic are undeniable[72], and of implementing draconian measures, intended for containment, rather than minimizing the impact of an epidemic on the local population[73].

In this paper we provide empirical evidence that these interventions can potentially affect civil violence in developing countries. We identified numerous acts of civil violence reported in newspapers, following the spread of cholera, Malaria, HIV/AIDS, in Congo, DRC, Kenya, Nigeria, Mozambique, Uganda, Tunisia, Somalia, South Africa[74]. We take the case of the recent Ebola epidemic in Western Africa to provide empirical evidence of epidemics leading to riots, protests and civil violence against government authorities, medical personnel and social workers trying to contain the outbreak, and provide precise mechanisms underlying this effect.

The impacts are large, immediate and tied to containment efforts. Our results show that the effects are tied to the emergency response, pre-existing levels of trust and barriers to cultural adaptation. A new infection in 100,000 per capita raises conflict incidence by 10% in a given location, from a baseline mean incidence of 0.0128 in 100,000 per capita at two-week level. Moving from no cases to the average number of infections for epidemic areas at the start of

[70] (UNICEF/UNDP/WorldBank/WHO, 2004, Jones et al., 2008)

[71] (WHO, 2016)

[72] "The lack of international political will was no longer an option when the realisation dawned that Ebola could cross the ocean. When Ebola became an international security threat, and no longer a humanitarian crisis affecting a handful of poor countries in West Africa, finally the world began to wake up," Dr Joanne Liu, MSF international president.

[73] "Whilst social unrest and fears of state collapse ran rampant, we feared that our call [for civilian and military assets with expertise in biohazard containment] would be misconstrued or intentionally twisted into a call for armed stabilisation", Christopher Stokes, MSF general director.

[74] The Armed Conflict Location and Event Data Project, 1997-2015.

the outbreak roughly doubles the incidence of conflict within two weeks. Our results suggest that state coercion, demand for public goods and little sensitivity to local cultural practices are determinants of civil conflict. We find the largest impacts of the epidemic on civil violence around the beginning of the international response, when safe burial practices were imposed. Areas with low trust in leaders and strong religious beliefs, are more likely to engage in such subversive violence, as they face higher costs of cultural change. State capacity has a large impact on civil violence in this context. We find large impacts of military district quarantines on increasing the likelihood of riots and protests, beyond and independently of the impact of new infections. The results are driven by areas with high demand for health treatment and access to it lowers conflict incidence, driven by areas closer to them. The epidemic led to lower trust in institutional authorities, especially for strongly religious communities.

Epidemics in which the state intervenes or is expected to intervene alter citizens' perception and demands from the state. Halting an epidemic leads the state to adopt coercive measures, it generates a demand for public goods and it requires a change in cultural practices. These changes lead to social unrest, depending on the coerciveness of the response, the capacity of the state to contain the outbreak, trust in institutional authorities and beliefs among citizens. These channels also mean that epidemics are more likely to lead to civil conflict in weak institutional settings, with low trust, weak public health systems and state coercion that is perceived as illegitimate. Moreover, depending on the state response, they lower trust in institutional authorities, therefore further weakening the state.

These findings have policy implications, as they inform the choice of emergency assistance. In particular, coercive measures, little sensitivity to local customs and a late intervention lead to social unrest, undermine containment efforts (Gonzalez-Torres, 2017) and have long-run effects on trust.

The possibility to track a shock from the first index case to the last contagion, an exogenous shock in state capacity generated by the influx of emergency assistance, combined with our fine-grained data and knowledge of the context and circumstances leading to civil violence, allow us to advance in our understanding of conflict in weak institutional settings.

References

Acemoglu, Fergusson, and D. Johnson, 2017: Population and social conflict. *Mimeo.*

Acemoglu, D. and S. Johnson, 2007: Disease and development: the effect of life expectancy on economic growth. *Journal of political Economy*, **115**(6), 925–985.

Acemoglu, D., S. Johnson, and J. Robinson, 2003: Disease and development in historical perspective. *Journal of the European Economic Association*, **1**(2-3), 397–405.

Acemoglu, D., S. Johnson, J. Robinson, and P. Yared, 2005: Income and democracy. Technical report, National Bureau of Economic Research.

Acemoglu, D., S. Johnson, J. A. Robinson, and P. Yared, 2009: Reevaluating the modernization hypothesis. *Journal of monetary economics*, **56**(8), 1043–1058.

Acemoglu, D. and J. Robinson, 2001: The colonial origins of comparative development: An empirical investigation. *American Economic Review*, **91**(5), 1369–1401.

Adda, J., 2016: Economic activity and the spread of viral diseases: Evidence from high frequency data. *The Quarterly Journal of Economics* qjw005.

Alsan, M., 2014: The effect of the tsetse fly on african development. *American Economic Review*, **105**(1), 382–410.

Alsan, M. and M. Wanamaker, 2016: Tuskegee and the health of black men. Technical report, National Bureau of Economic Research.

Anderson, M. L., 2008: Multiple inference and gender differences in the effects of early intervention: A reevaluation of the abecedarian, perry preschool, and early training projects. *Journal of the American statistical Association*, **103**(484), 1481–1495.

Barbera, S. and M. O. Jackson, 2016: A model of protests, revolution, and information.

Bazzi, S. and C. Blattman, 2014: Economic shocks and conflict: Evidence from commodity prices. *American Economic Journal: Macroeconomics*, **6**(4), 1–38.

Beck, N. and J. N. Katz, 2004: Time-series-cross-section issues: dynamics, 2004. In *Annual meeting of the Society for Political Methodology, Stanford University.*

Bellows, J. and E. Miguel, 2006: War and institutions: New evidence from sierra leone. *American Economic Review*, **96**(2), 394–399.

Blattman, C. and E. Miguel, 2010: Civil war. *Journal of Economic literature*, **48**(1), 3–57.

Bloom, D. E. and J. D. Sachs, 1998: Geography, demography, and economic growth in africa.

Brookings papers on economic activity, **1998**(2), 207–295.

Brückner, M. and A. Ciccone, 2010: International commodity prices, growth and the outbreak of civil war in sub-saharan africa. *The Economic Journal*, **120**(544), 519–534.

Burke, M. B., E. Miguel, S. Satyanath, J. A. Dykema, and D. B. Lobell, 2009: Warming increases the risk of civil war in africa. *Proceedings of the national Academy of sciences*, **106**(49), 20670–20674.

Cantoni, D., D. Y. Yang, N. Yuchtman, and Y. J. Zhang, 2017: Are protests games of strategic complements or substitutes? experimental evidence from hong kong's democracy movement. Technical report, National Bureau of Economic Research.

Caprettini, B. and H.-J. Voth, 2017: Rage against the machines : Labor-saving technology and unrest in england 1830–32.

Cervellati, M., E. Esposito, U. Sunde, and S. Valmori, 2017: Malaria risk and civil violence. *CEPR Discussion Paper, 11496*.

Cervellati, M., E. Esposito, U. Sunde, and S. Valmori, 2018: Long-term exposure to malaria and violence in africa. *Economic Policy, forthcoming*.

Cervellati, M., U. Sunde, and S. Valmori, 2016: Pathogens, weather shocks, and civil conflicts. *The Economic Journal*.

Cohn, S., 2016: Plague and prejudice. *History Today*, **66**(3), 3.

Cohn, S. and R. Kutalek, 2016: Historical parallels, ebola virus disease and cholera: understanding community distrust and social violence with epidemics. *PLOS Currents Outbreaks*.

Dittmar, J. E. and R. R. Meisenzahl, 2016: State capacity and public goods: Institutional change, human capital, and growth in early modern germany.

Dupas, P., 2011: Health behavior in developing countries. *Annu. Rev. Econ.*, **3**(1), 425–449.

Easterly, W. and R. Levine, 1997: Africa's growth tragedy: policies and ethnic divisions. *The Quarterly Journal of Economics*, **112**(4), 1203–1250.

Eifert, B., E. Miguel, and D. N. Posner, 2010: Political competition and ethnic identification in africa. *American Journal of Political Science*, **54**(2), 494–510.

Esteban, J. and D. Ray, 2008: On the salience of ethnic conflict. *American Economic Review*, **98**(5), 2185–2202.

Esteban, J. and D. Ray, 2011a: Linking conflict to inequality and polarization. *American Economic Review*, **101**(4), 1345–1374.

Esteban, J. and D. Ray, 2011b: A model of ethnic conflict. *Journal of the European Economic Association*, **9**(3), 496–521.

Evans, R. J., 1988: Epidemics and revolutions: Cholera in nineteenth-century europe. *Past and Present*, (120), 123–146.

Fang, L.-Q., Y. Yang, J.-F. Jiang, H.-W. Yao, D. Kargbo, X.-L. Li, B.-G. Jiang, B. Kargbo, Y.-G. Tong, Y.-W. Wang, et al., 2016: Transmission dynamics of ebola virus disease and intervention effectiveness in sierra leone. *Proceedings of the National Academy of Sciences*, **113**(16), 4488–4493.

Fast, S. M., S. Mekaru, J. S. Brownstein, T. A. Postlethwaite, and N. Markuzon, 2014: The role of social mobilization in controlling ebola virus in lofa county liberia. *PLoS currents*, **7**.

Fearon, J. D. and D. D. Laitin, 2003: Ethnicity, insurgency, and civil war. *American political science review*, **97**(1), 75–90.

GHRF, C. o. G. H. R. F. f. t. F., 2016: *The Neglected Dimension of Global Security: A Framework to Counter Infectious Disease Crises*. The National Academies Press, Washington, DC.

Glennerster, R., E. Miguel, and A. D. Rothenberg, 2013: Collective action in diverse sierra leone communities. *The Economic Journal*, **123**(568), 285–316.

Glennerster, R., T. Suri, and S. Bhogale, 2016: The implications of the ebola outbreak on markets, traders, and food security in sierra leone. *Bulletin : The economic impacts of Ebola*.

Gonzalez-Torres, A., 2017: Propaganda and cultural change: Local radios and the spread of ebola in guinea. *working paper*.

Greenwood, J., P. Kircher, C. Santos, and M. Tertilt, 2013: An equilibrium model of the african hiv aids epidemic. Technical report, National Bureau of Economic Research.

Habyarimana, J., M. Humphreys, D. N. Posner, and J. M. Weinstein, 2007: Why does ethnic diversity undermine public goods provision? *American Political Science Review*, **101**(4), 709–725.

Harari, M. and E. La Ferrara, 2013: Conflict, climate and cells: A disaggregated analysis.

Hofman, M. and S. Au, 2017: *The Politics of Fear: Medecins sans Frontieres and the West African Ebola Epidemic*. Oxford University Press.

Hsiang, S. M., M. Burke, and E. Miguel, 2013: Quantifying the influence of climate on human conflict. *Science*.

Hsiang, S. M., K. C. Meng, and M. A. Cane, 2011: Civil conflicts are associated with the global climate. *Nature*, **476**(7361), 438–441.

Jones, K. E., N. G. Patel, M. A. Levy, A. Storeygard, D. Balk, J. L. Gittleman, and P. Daszak, 2008: Global trends in emerging infectious diseases. *Nature*, **451**(7181), 990–993.

Jordà, O., 2005: Estimation and inference of impulse responses local projections. *American economic review*, **95**(1), 161–182.

Judson, R. A. and A. L. Owen, 1999: Estimating dynamic panel data models: a guide for macroeconomists. *Economics letters*, **65**(1), 9–15.

Miguel, E., 2005: Poverty and witch killing. *The Review of Economic Studies*, **72**(4), 1153–1172.

Miguel, E., S. Satyanath, and E. Sergenti, 2004: Economic shocks and civil conflict: An instrumental variables approach. *Journal of political Economy*, **112**(4), 725–753.

Moon, S. e. a., 2015: Will ebola change the game? ten essential reforms before the next pandemic. *The Lancet*, **386**(10009), 2204 – 2221.

Morse, B., K. A. Grépin, R. A. Blair, and L. Tsai, 2016: Patterns of demand for non-ebola health services during and after the ebola outbreak: panel survey evidence from monrovia, liberia. *BMJ Global Health*, **1**(1), e000007.

Nickell, S., 1981: Biases in dynamic models with fixed effects. *Econometrica: Journal of the Econometric Society* 1417–1426.

North, D. C., 1990: *Institutions, institutional change and economic performance*. Cambridge university press.

Nunn, N. and N. Qian, 2014: Us food aid and civil conflict. *American Economic Review*, **104**(6), 1630–1666.

Nunn, N. and L. Wantchekon, 2011: The slave trade and the origins of mistrust in africa. *American Economic Review*, **101**(7), 3221–3252.

Oster, E., 2005: Sexually transmitted infections, sexual behavior, and the hiv aids epidemic. *The Quarterly Journal of Economics* 467–515.

Posner, D. N., 2004: The political salience of cultural difference: Why chewas and tumbukas are allies in zambia and adversaries in malawi. *American Political Science Review*, **98**(4), 529–545.

Richards, P., 2016: *Ebola: how a people's science helped end an epidemic*. Zed Books Ltd.

UNICEF/UNDP/WorldBank/WHO, 2004: Globalization and infectious diseases: A review of

the linkage. Technical report.

Van Kerkhove, M. D., A. I. Bento, H. L. Mills, N. M. Ferguson, and C. A. Donnelly, 2015: A review of epidemiological parameters from ebola outbreaks to inform early public health decision-making. *Scientific data*, **2**.

Voigtländer, N. and H.-J. Voth, 2012: Persecution perpetuated: the medieval origins of anti-semitic violence in nazi germany. *The Quarterly Journal of Economics*, **127**(3), 1339–1392.

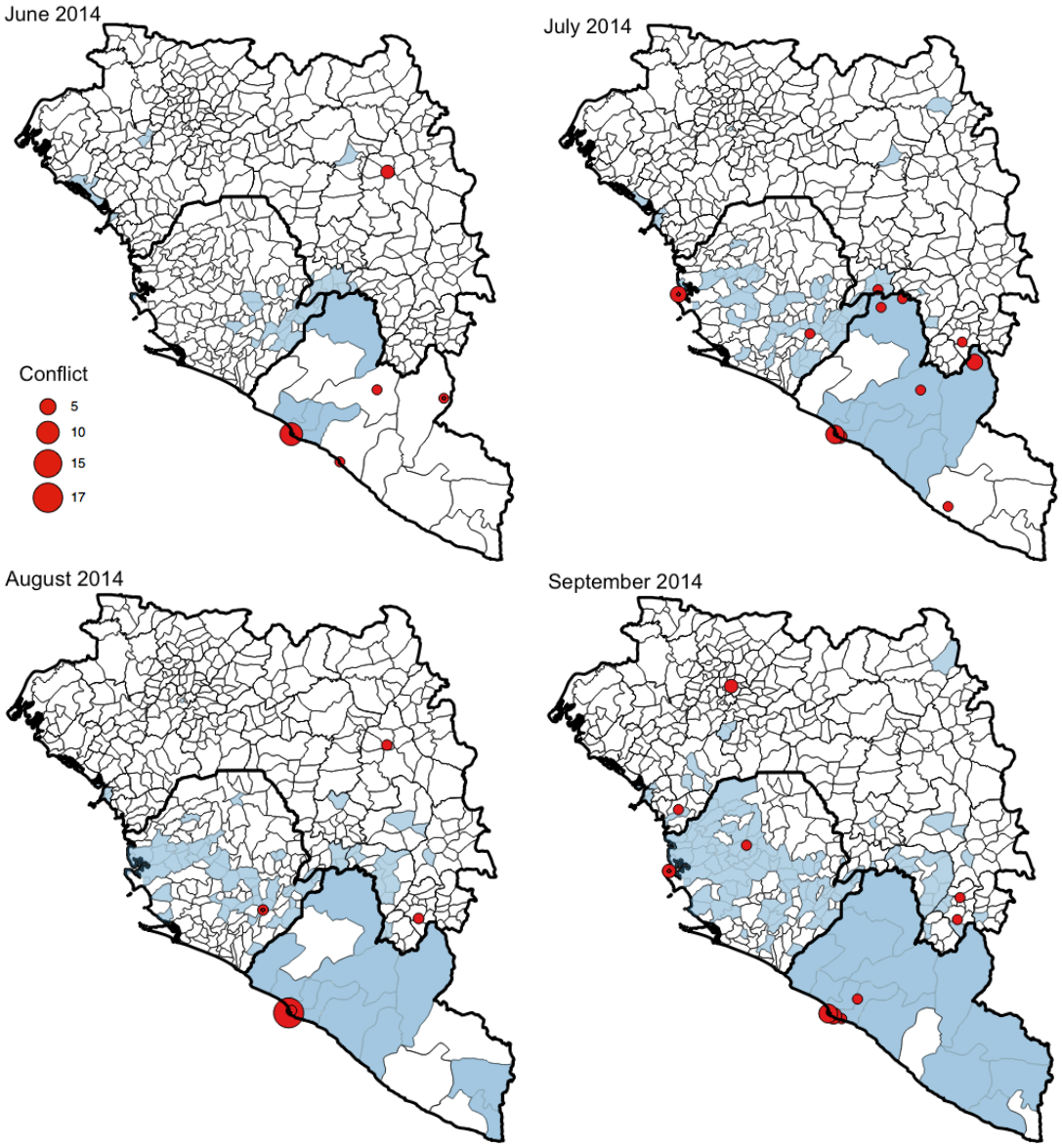
WHO, 2015: Global health estimates. Technical report.

WHO, 2016: Mapping the risk and distribution of epidemics in the who african region: a technical report. *Work Health Organization : Regional Office for Africa*.

Young, A., 2005: The gift of the dying: The tragedy of aids and the welfare of future african generations. *The Quarterly Journal of Economics*, **120**(2), 423–466.

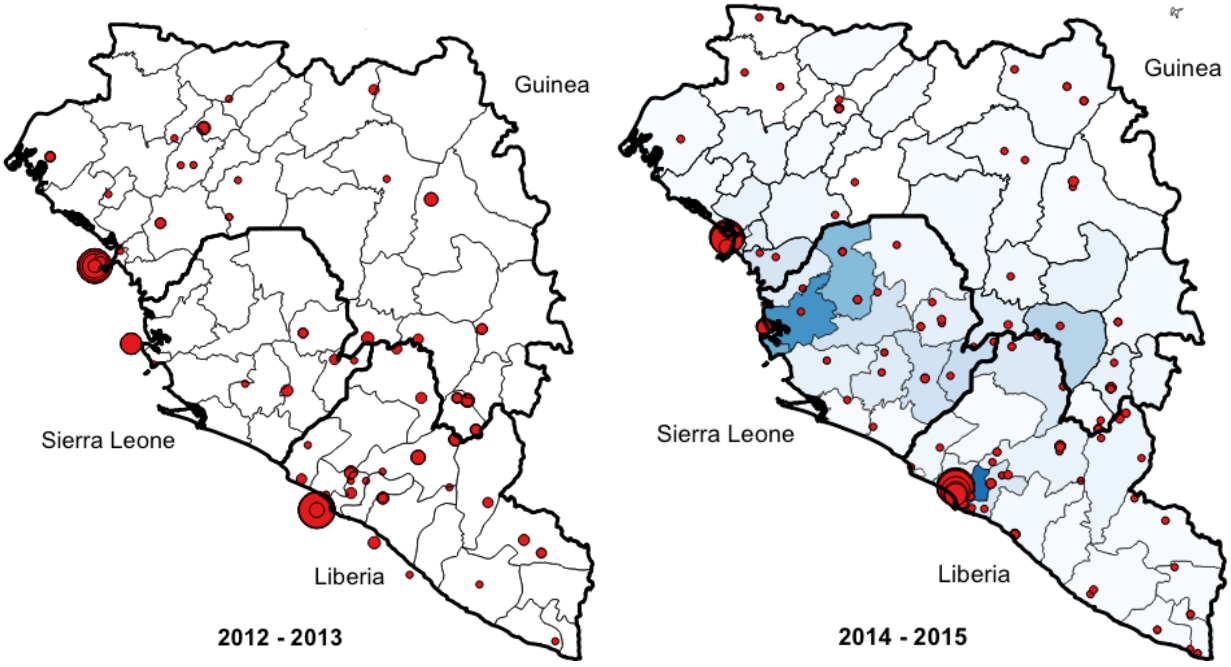
A Appendix

Figure A.1: Presence of the epidemic and conflict incidence month per month



Notes: Presence of the epidemic $\in \{0, 1\}$ (blue shade) and number of conflicts in a given location month per month (red dots).

Figure A.2: Total Ebola and Conflict incidence in West Africa, pre and post-Epidemic



Notes: Cumulative Ebola infections per district (more if darker shade) and Conflict events (red dots) weighted by number of conflicts in the same location. We see more conflict events in Sierra Leone in the post-Epidemic compared to pre-Epidemic period. We see some displacement of conflict in Guinea towards locations with more cumulative number of Ebola infections. For Liberia the effects are not evident from the raw data at cross-sectional level.

Figure A.3: Aggregate number of Ebola cases (confirmed + probable)

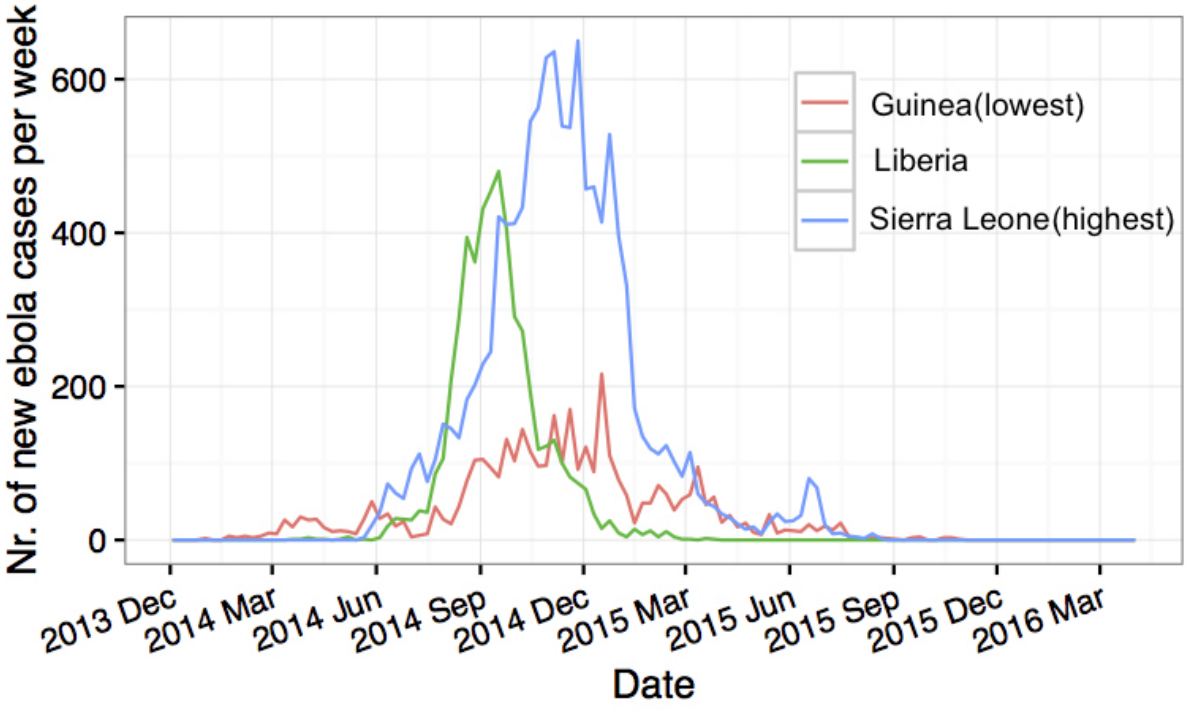


Figure A.4: Density of Riots and Protests in affected countries (above) or all Africa (below), 2010-2016

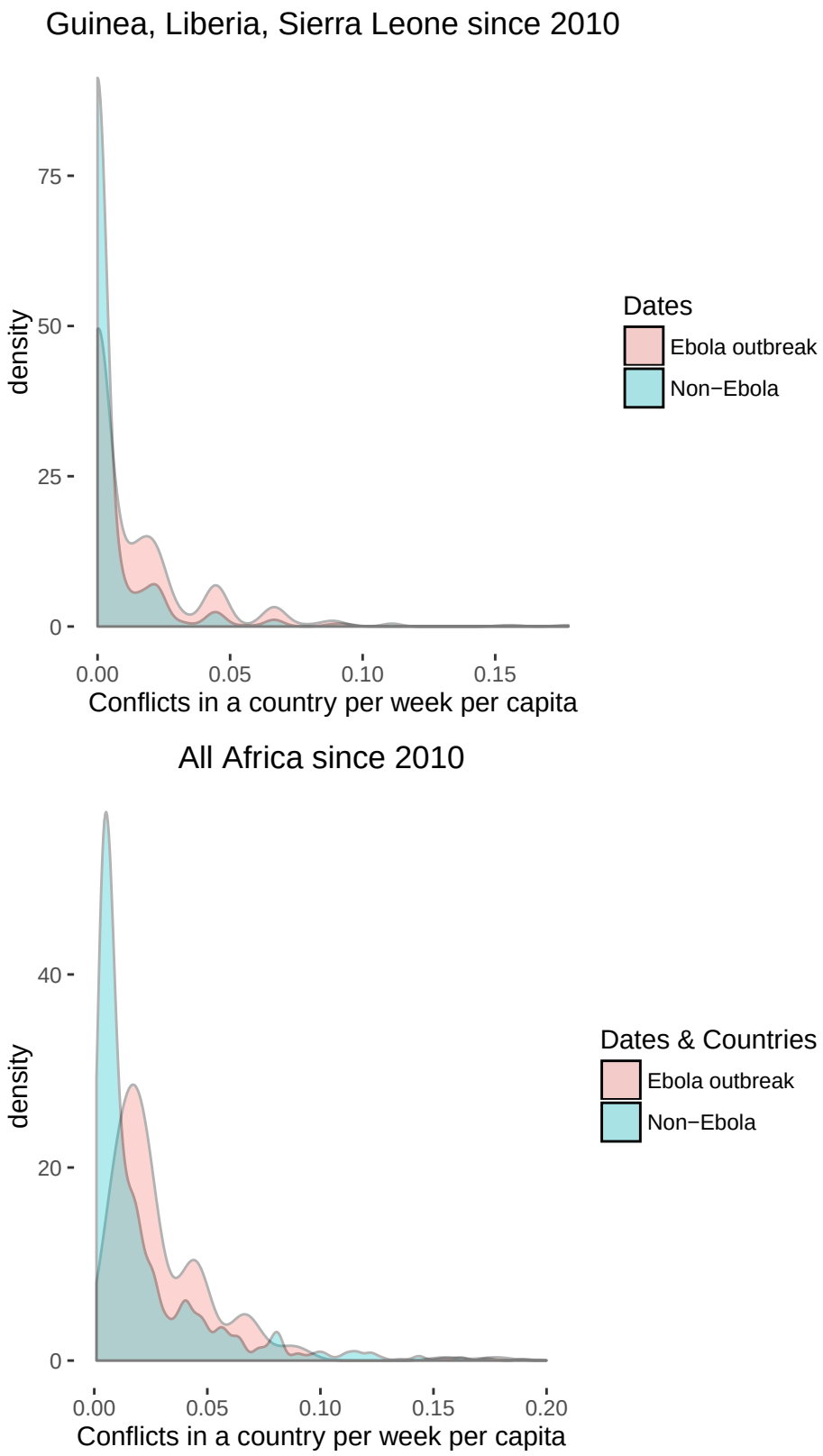


Table A.1: Descriptives in Yearly quarters from Jan 2012 to May 2016

	Pre-Epidemic 2012-2013	Post-Epidemic 2014-2016/Q2	Total	Difference Post-Pre Epid [t-test]	Total
Conflict	0.0383	0.0534	0.0467	0.0151	
	(0.393)	(0.539)	(0.479)	[1.61]	
conflict	0.763	0.856	0.815	0.0929	
	(8.641)	(7.904)	(8.239)	[0.57]	
EbolaTotal					66.83
					(283.0)
ebolaTotal					50.08
					(124.9)
Observations	4,672	5,840	10512		584

Notes: The epidemic starts at the end of December 2013 in Guinea, but we do not observe the first few cases. $Conflict$ is the number of conflicts in a given yearly quarter (riots, protests and violence against civilians) for each observational unit. $conflict$ is the number of conflict events in one million per capita in a yearly quarter. Difference in means post-Epidemic-pre-Epidemic are followed by t-test statistics in square brackets. $EbolaTotal$ are the total cumulative number of Ebola cases counted at the end of the epidemic. $ebolaTotal$ are these measured in 100'000 per capita.

Table A.2: Summary statistics

Ebola cases				**Conflict**				
	Pre-Epid. 2012-13	Epidemic 2014-16/5	Total 2012-16		Pre-Epid. 2012-13	Epidemic 2014-16/5	Total 2012-16	Difference Epid.-Pre-Epid.
ebola $\in \{0,1\}$	0	0.071	0.039					
	(0)	(0.257)	(0.19)					
confirmed	0	0.815	0.447					
	(0)	(9.631)	(7.140)					
probable	0	0.245	0.134	conflict $\in \{0,1\}$	0.00491	0.00679	0.00594	0.00189**
	(0)	(3.897)	(2.887)		(0.0699)	(0.0822)	(0.0768)	[3.17]
ebola (c+p)	0	1.061	0.581					
	(0)	(12.88)	(9.55)	Conflicts	0.00589	0.00848	0.00731	0.00259**
ebola^{pc} (c+p)	0	0.855	0.468		(0.0914)	(0.116)	(0.106)	[3.16]
	(0)	(7.49)	(5.56)					
suspect	0	1.662	0.911	**Conflicts**^{pc}	0.117	0.136	0.128	0.0185
	(0)	(15.26)	(11.33)		(0.279)	(2.85)	(2.82)	[0.84]
total	0	2.723	1.492					
	(0)	(22.59)	(16.78)					
ebola^{pc} total	0	1.553	0.851					
	(0)	(10.06)	(7.485)					
N	30,368	36,792	67160	N	30,368	36,792	67160	
chiefdom (SLE) / sub-pref (GIN) / county (LBR) 2-week windows				chiefdom (SLE) / sub-pref (GIN) / district(LBR) 2-week windows, * p<0.05, ** p<0.01, (SE), [t-stat]				

Notes: The main variables used in the regressions are $Conflicts^{pc}$, namely the number of conflicts in one million per capita and $ebola^{pc}$ (c+p), the number of confirmed and probable Ebola cases in 100'000 per capita.

Table A.3: Descriptives - State Response - provision by country (population-weighted)

	GIN	LBR	SLE	Total
EverETU	0.135	0.512	0.224	0.238
	(0.342)	(0.503)	(0.418)	(0.426)
EverLab	0.126	0.451	0.293	0.242
	(0.333)	(0.500)	(0.457)	(0.429)
EverCCC	0.133	0.190	0.620	0.294
	(0.340)	(0.394)	(0.487)	(0.456)
EverQuar	0.0290	0.825	0.762	0.412
	(0.168)	(0.382)	(0.428)	(0.493)
NearETU^{end}	0.133	0.500	0.186	0.223
	(0.338)	(0.490)	(0.385)	(0.411)
NearLab^{end}	0.123	0.445	0.256	0.228
	(0.323)	(0.494)	(0.435)	(0.415)
Observations				584

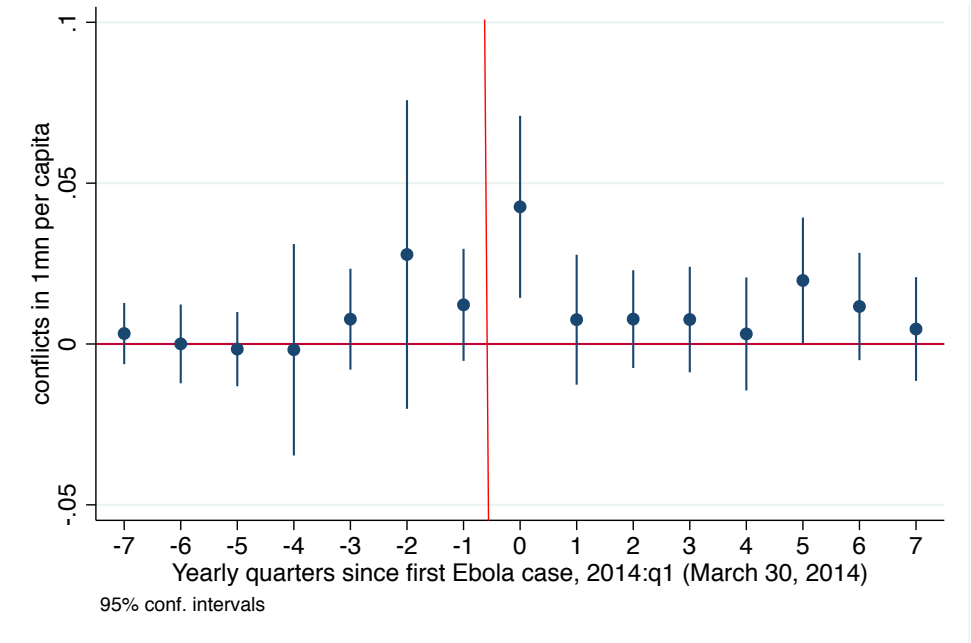
Notes: Ebola treatment Units (ETU), Laboratories, Community care centers (CCCs), Military district-Quarantines. *Ever* are dummy variables indicating whether a location had access to it or not. $Near^{end}$ is the smallest inverse distance to an ETU/Lab measured at the end of the outbreak for locations that do not have one on their own, or 0 if they do.

Table A.4: Descriptives - State Response - average population size

	ETUs	Labs	CCCs	Quarantines
Without	31,512	31,205	35,642	33,503
	(51656.8)	(51016.4)	(97315.7)	(62999.3)
With	175,189	190,972	51,227	51,487
	(349568.5)	(358544.7)	(105232.8)	(150396.6)
Total	39,138	39,138	39,138	39,138
	(99267.3)	(99267.3)	(99267.3)	(99267.3)
Observations	584	584	584	584

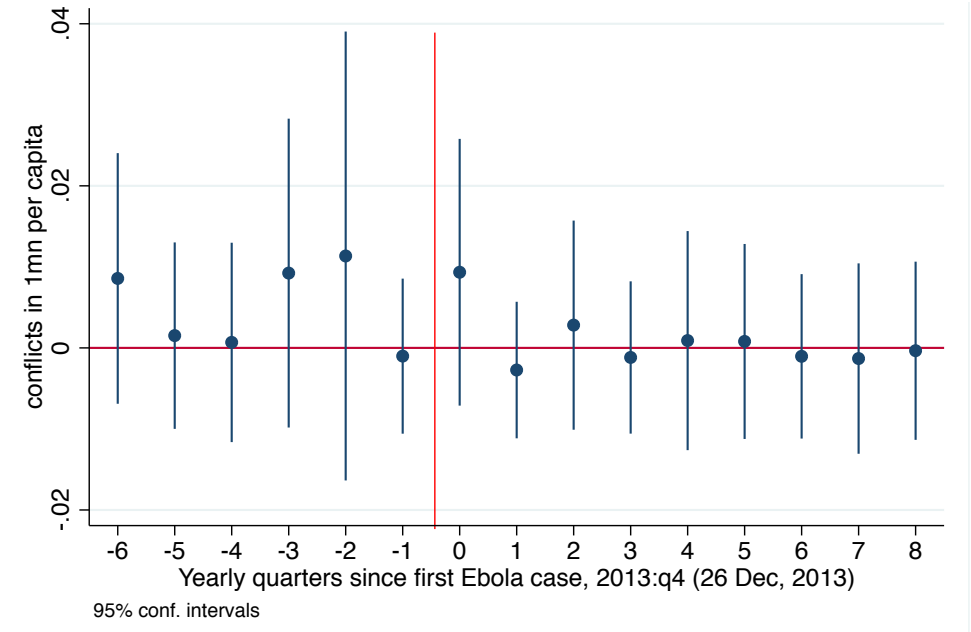
Notes: Ebola treatment Units (ETU), Laboratories, Community care centers (CCCs), Military district-Quarantines or area blockades.

Figure A.5: Difference in Difference relative to the first case in counry - Liberia



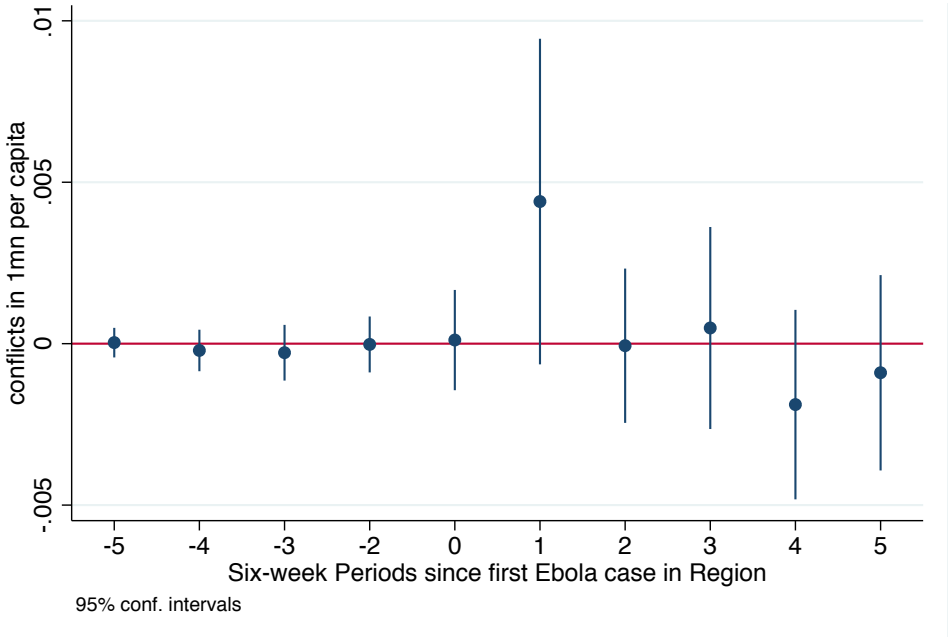
Notes: Coefficients on the total end-number of Ebola cases in one location (county) × dummy for a yearly quarter. Calendar time since first case in Liberia.

Figure A.6: Difference in Difference relative to the first case in country - Guinea



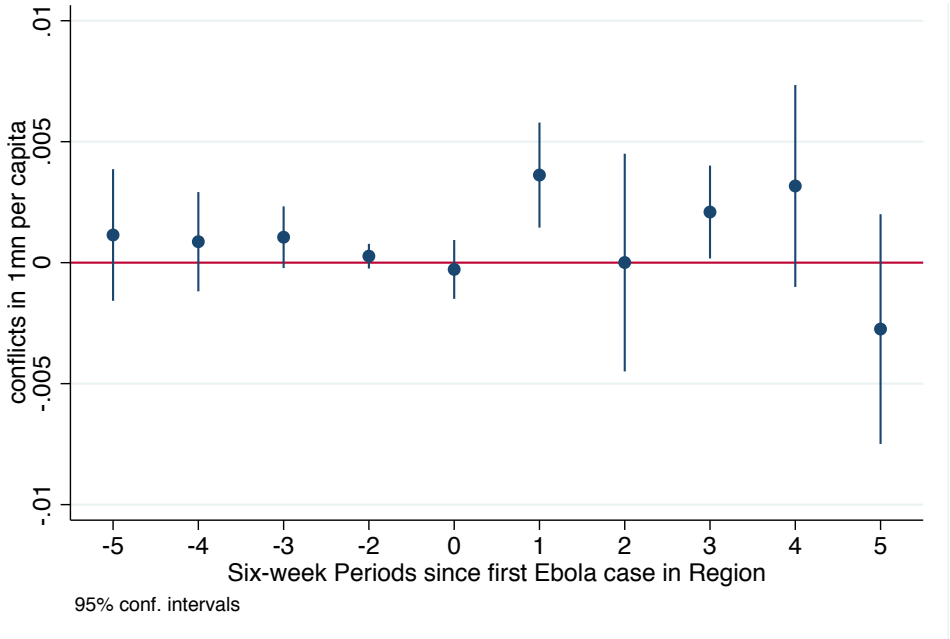
Notes: Coefficients on the total end-number of Ebola cases in 100'000 per capita in a given location (sub-prefecture) × dummy for a yearly quarter. Calendar time since first case in Guinea.

Figure A.7: Difference in Difference relative to first case in prefecture - Guinea



Notes: Coefficients on the total end-number of Ebola cases in 100'000 per capita in a given location (sub-prefecture) × dummy for a six-week period. Time relative to first case in region (prefecture).

Figure A.8: Difference in Difference relative to first case in district - Sierra Leone



Notes: Coefficients on the total end-number of Ebola cases in one location (chiefdom) × dummy for a six-week period. Time relative to first case in region (district).

Table A.5: Difference in Differences relative to the first index case in West Africa - First Stage

	First Stage	Reduced Form
	(1) ebolaTot × PostEbola	(2) conflict(quarter)
DistEpic × PostEbola	-.00101***	-1.11e-06
	(.000369)	(3.40e-06)
DistEpic × I(Guinea) × PostEbola	.000388	-6.00e-08
	(.000283)	(2.46e-06)
DistEpic × I(Liberia) × PostEbola	.0014***	.000013
	(.000427)	(7.90e-06)
$DistEpic^2$ × PostEbola	3.77e-09***	6.92e-12
	(1.07e-09)	(9.44e-12)
$DistEpic^2$ × I(Guinea) × PostEbola	-2.92e-09***	-7.93e-12
	(9.70e-10)	(8.72e-12)
$DistEpic^2$ × I(Liberia) × PostEbola	-5.26e-09***	-2.82e-11
	(1.22e-09)	(1.97e-11)
N	10512	10512
R2	0.706	0.429
Time FE	Y	Y
Chiefd FE	Y	Y

(Clustered SE) by Dist; Controls Restr.: sample restricted to locations with household survey data.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: $conflict$ is the number of conflicts in each yearly quarter in 1 million per capita. $ebolaTotal$ is the total cumulative number of Ebola cases measured at the end of the outbreak for each location in 100'000 per capita. $PostEbola$ is a post-treatment dummy taking value 1 from 2014 on, after the first Ebola case is observed. $DistEpic$ is the geographic linear distance to the first index case, $DistEpic^2$ is the square distance. $I(.)$ is an indicator variable for each country.
First stage F-Statistic: 10.24.

Table A.6: Distance to the Epicenter does not predict Conflict incidence pre-epidemic (stand. coef)

	(1) conflict(t)	(2) Ethnic Fract.	(3) Ethnic Polar.	(4) Ethnic Salience	(5) Strongly Relig.	(6) Trad. Relig.	(7) Trust Leader	(8) Trust People	(9) Infrastr.	(10) Incumbent Vote/Pref.
DistEpic	0.047	0.567	-0.308	0.305	0.464	-1.033**	-0.240	-0.466	0.226	-0.558*
	[1.40]	[1.22]	[-0.92]	[0.70]	[1.40]	[-2.39]	[-0.80]	[-1.23]	[1.42]	[-1.89]
$DistEpic^2$	-0.024	-0.393	0.388	-0.143	-0.312	0.751**	0.671**	0.511	-0.268	0.615**
	[-0.51]	[-0.81]	[1.18]	[-0.33]	[-1.03]	[2.07]	[2.35]	[1.28]	[-1.27]	[2.27]
N	30368	215	215	215	215	215	215	215	215	215
R2	0.006	0.046	0.015	0.029	0.033	0.135	0.197	0.019	0.006	0.028

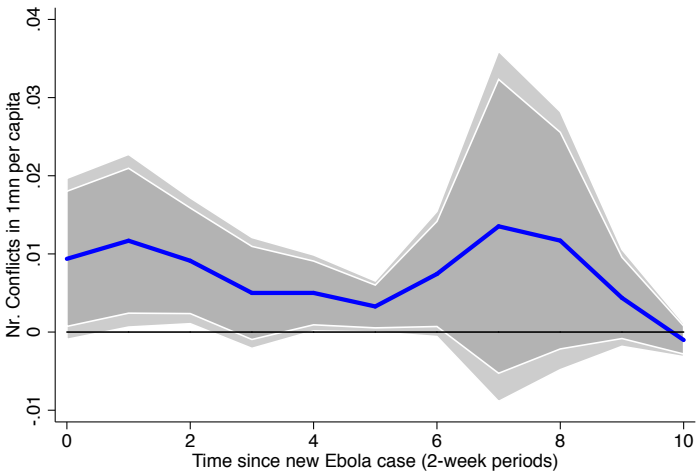
Standardized coef.; [t-stat]; Robust SE clustered by Location; Week FE
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: In column (1) t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. $DistEpic$ is the geographic linear distance to the first index case, $DistEpic^2$ is the square distance. Columns (2)-(10) measure the correlation between these and expected correlates of civil violence based on survey data from the Afrobarometer round immediately prior to the epidemic, 2012-13.

Table A.7: Descriptives - Population by country for each observational unit

	(1) all	(2) ebola
Guinea	33,119	
	(66387.9)	
Liberia	49,592	267,264.6
	(173878.1)	(294331.4)
Sierra Leone	46,361	
	(98821.1)	
Total	39,138	
	(99267.3)	
Observations	584	499

Figure A.9: Impulse response for local projections for the impacts of ebola infections in 100'000 per capita (intensive margin) on conflict incidence for 10 future time periods



Notes: The coefficients of $ebola_{t-1}$ in equation (5) are plotted, with 90% and 95% confidence intervals. We condition on 9 lags in ebola and conflict (two-week frequency).

Table A.8: High-frequency panel: Main specification

Outcome: conflict(t)	Pooled	Panel					
	(1)	(2)	(3)	(4)	**(5)**	**(6)**	**(7)**
ebola(t-1)	0.0173***	0.0138***	0.0129***	0.0116**	**0.0146*****	**0.0141*****	**0.0123****
	(0.0047)	(0.0039)	(0.0038)	(0.0049)	(0.0041)	(0.0039)	(0.0049)
ebola(t-2)				0.00195			0.00290
				(0.0041)			(0.0043)
ebolaCum(t-2)			0.000492**	0.000452*		0.000464*	0.000401
			(0.0002)	(0.0003)		(0.0003)	(0.0003)
Constant	0.176***						
	(0.0503)						
N	66576	66576	65992	65992	66574	65990	65990
R2	0.002	0.125	0.126	0.126	0.143	0.144	0.144
Time FE		Y	Y	Y	Y	Y	Y
Chiefd FE		Y	Y	Y	Y	Y	Y
Reg × Month FE					Y	Y	Y

(Clustered SE) by Dist

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. $ebolaCum$ is the cumulative number of Ebola infections over time. Our preferred specifications and our coefficient of interest are highlighted in bold.

Table A.9: Main specification - High frequency panel (standardized coef.)

Outcome: conflict(t)	Pooled	Panel					
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
ebola(t-1)	0.046***	0.037***	0.035***	0.031**	0.039***	0.038***	0.033**
	(0.0047)	(0.0039)	(0.0038)	(0.0049)	(0.0041)	(0.0039)	(0.0049)
ebola(t-2)				0.005			0.008
				(0.0041)			(0.0043)
ebolaCum(t-2)			0.013**	0.011*		0.012*	0.010
			(0.0002)	(0.0003)		(0.0003)	(0.0003)
N	66576	66576	65992	65992	66574	65990	65990
R2	0.002	0.125	0.126	0.126	0.143	0.144	0.144
Time FE		Y	Y	Y	Y	Y	Y
Chiefd FE		Y	Y	Y	Y	Y	Y
Reg × Month FE					Y	Y	Y

Stand. coef.; (Clustered SE) by Dist

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. $ebolaCum$ is the cumulative number of Ebola infections over time. Our preferred specifications and our coefficient of interest are highlighted in bold.

Table A.10: Difference-in-Difference using first index case in each location

Outcome: conflict(t)	Pre-Trends?		Diff-in-Diff				
	(1) OLS	(2) OLS	(3) OLS	(4) OLS	(5) OLS	(6) OLS	(7) 2SLS
ebolaTot × PostEbola × Trend	0.00000681	0.00000681					
	(0.000005)	(0.000005)					
ebolaTot × Trend	0.00000171	0.00000171			-0.00000425	-0.00000338	
	(0.000007)	(0.000007)			(0.000005)	(0.000006)	
PostEbola			0.0630				
			(0.061905)				
PostEbola × ebolaTot				0.000889***	0.00113***	0.00116**	0.00121***
				(0.000266)	(0.000429)	(0.000573)	(0.000396)
N	67160	67160	67160	67160	67160	67158	67160
R2	0.124	0.124	0.123	0.124	0.124	0.142	0.124
Time FE	Y	Y	Y	Y	Y	Y	Y
Chiefd FE	Y	Y	Y	Y	Y	Y	Y
Reg × Month FE		Y				Y	

(Clustered SE) by Location

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. $ebolaTot$ is the total end-number Ebola cases in 100'000 per capita. $ebolaPost$ is a dummy variable that is 0 before Ebola hits a location for the first time and then takes value 1 forever. In the last column we instrument $ebolaPost \times ebolaTot$ with the geographic linear and square distance to epicenter, interacted with $ebolaPost$ and country-dummies. First stage: $R^2 = 0.66$, $F - statistic = 12.93$.

Figure A.10: Predicted Ebola vs Ebola cases in Sierra Leone following Fang et al. (2016)

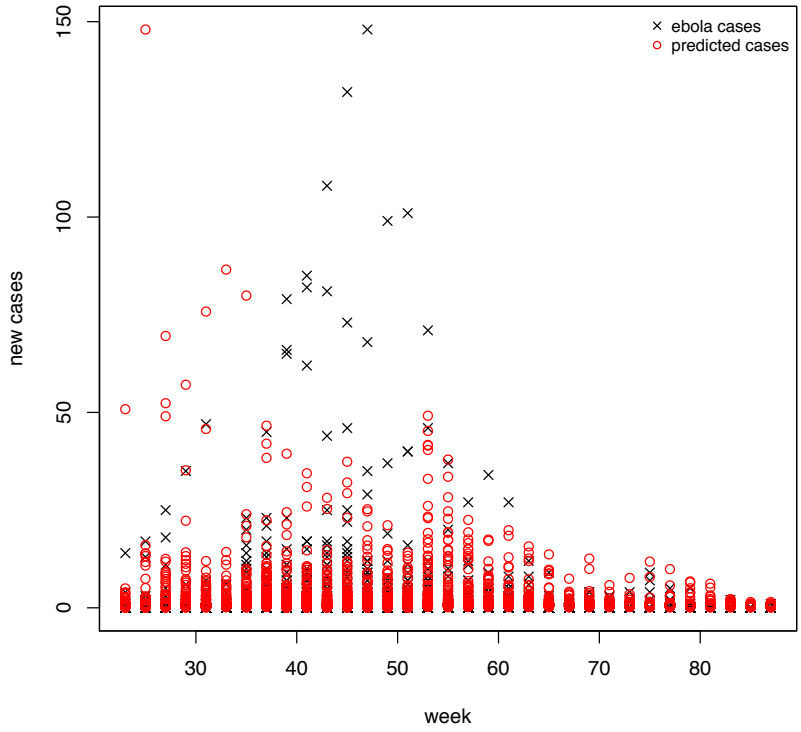


Table A.11: High frequency panel - Instrument : $ebolaNeigh(t-2) \in \{0,1\}$

	OLS	2SLS		Reduced Form	First Stage
	(1) conflict(t)	(2) conflict(t)	(3) conflict(t)	(4) conflict(t)	(5) ebola(t-1)
ebola(t-1)	0.0138***	0.0377***	0.0374***		
	(0.0039)	(0.0123)	(0.0124)		
conflictOthers(t-2)			0.0916		
			(0.0690)		
ebolaNeigh(t-2) $\times$ $I(Liberia)$				0.203	3.526***
				(0.1223)	(1.1462)
ebolaNeigh(t-2) $\times$ $I(SierraLeone)$				0.162**	4.596***
				(0.0692)	(0.8478)
ebolaNeigh(t-2) $\times$ $I(Capital)$				0.0466	1.919
				(0.1569)	(1.1676)
ebolaNeigh(t-2) $\times$ $I(District - Capital)$				-0.000360	3.765***
				(0.0934)	(1.0700)
N	66576	65992	65992	65992	65992
R2	0.125	0.123	0.123	0.125	0.192
Time FE	Y	Y	Y	Y	Y
Chiefd FE	Y	Y	Y	Y	Y

(Clustered SE) by Dist

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: $conflict(t)$ are the number of conflicts in one million per capita in the own location. $conflictOthers(t-2)$ is the number of conflict events in the region except the own location. $ebola(t-1)$ is the number Ebola cases in 100'000 per capita. The instrument is the presence of the epidemic in neighboring locations, $ebolaNeigh(t-2) \in \{0,1\}$. We allow for the effect to vary for each country, as well as the capital and main chiefdoms within a given district, using indicator variables $I(.)$. First stage: $R^2 = 0.19, F - Statistic = 40.26$.

Table A.12: Lags and Leads (regression)

	(1) conflict(t)
ebola(t)	0.00775
	(0.00605)
ebola(t-1)	**0.00566****
	(0.00230)
ebola(t-2)	0.00184
	(0.00567)
ebola(t-3)	0.00309
	(0.00383)
ebola(t+1)	-0.00165
	(0.00496)
ebola(t+2)	0.00534
	(0.00351)
ebola(t+3)	0.00447
	(0.00296)
ebolaCum(t-3)	0.000539***
	(0.000175)
N	63652
R2	0.147

(Clustered SE) by Dist; Time FE, Chiefd. FE; Reg. × Month FE
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. $ebolaCum$ is the cumulative number of Ebola infections over time.

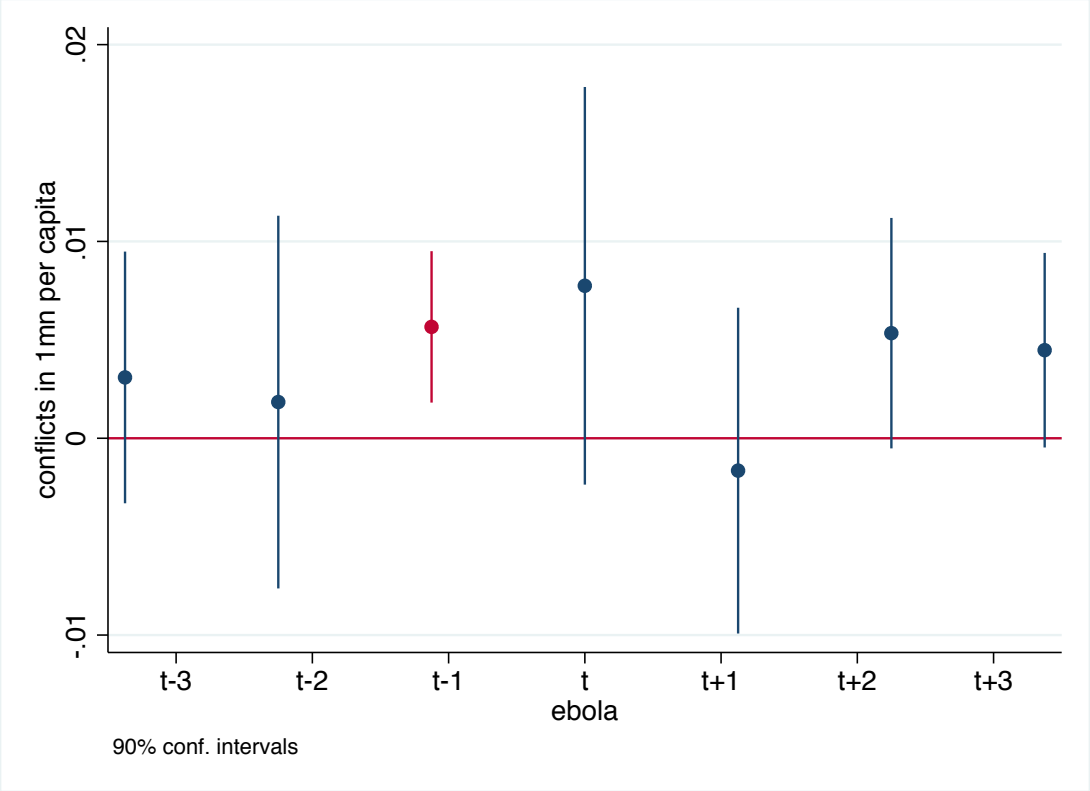
Table A.13: Lags and Leads (F-test)

	(1) p-value
ebola(t-1)=ebola(t)	0.724
ebola(t-1)=ebola(t-2)	0.594
ebola(t-1)=ebola(t-3)	0.540
ebola(t-1)=ebola(t+1)	0.242
ebola(t-1)=ebola(t+2)	0.946
ebola(t-1)=ebola(t+3)	0.763
(Lags)=0	**0.000**
SumLags=0	**0.000**
(Leads t+1, t+2)=0	0.320
SumLeads t+1, t+2=0	0.489
(LeadsAll)=0	0.271
SumLeadsAll=0	0.165

Stand. coef.; (Clustered SE) by Dist; Time FE, Chiefd. FE; Reg. × Month FE
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: F-test in a regression including new Ebola cases at three leads and lags and controlling for cumulative Ebola, Table A.12. t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. $ebolaCum$ is the cumulative number of Ebola infections over time.

Figure A.11: Lags and Leads (graph)



Notes: Coefficients on *ebola* at different time periods in a regression including new Ebola cases at three leads and lags and controlling for cumulative Ebola, Table A.12. t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. $ebolaCum$ is the cumulative number of Ebola infections over time.

Table A.14: Wooldridge (2002) test for autocorrelation in Panel data

Linear model: $conflict$ on *ebola*, $ebolaCum$

H_0: no first-order autocorrelation	
F(1, 583)	0.056
p-value	0.8130

Notes: The specification is in two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. $ebolaCum$ is the cumulative number of cases over time in 100'000 per capita.

Table A.15: Feedback effects?

	conflict(t)					ebola(t)	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
ebola(t-1)	0.0137***	0.0121***	0.0128***	0.0114***		0.636***	0.630***
	(0.0043)	(0.0040)	(0.0040)	(0.0037)		(0.0948)	(0.1076)
ebolaCum(t-1)		0.000684***		0.000645***			
		(0.0002)		(0.0002)			
conflict(t-1)			0.0534	0.0532	0.0545		0.00738
			(0.0410)	(0.0410)	(0.0409)		(0.0103)
conflict(t-1) × ebola(t-1)							0.00127
							(0.0039)
N	66576	66576	66576	66576	66576	66576	66576
R2	0.142	0.142	0.144	0.144	0.143	0.605	0.605

(Clustered SE) by Dist; cond. Time, Chiefd, Reg × Month FE

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. $ebolaCum$ is the cumulative number of cases over time in 100'000 per capita.

Table A.16: Robustness: different Conflict and Ebola measures

	(1) conflict(t)	(2) conflict(t) $\in \{0,1\}$	(3) conflict(t)	(4) conflict(t) $\in \{0,1\}$
ebola(t-1)	0.00208***	0.000907***		
	(0.00031)	(0.00003)		
ebola+suspect(t-1)			0.00145***	0.000657***
			(0.00023)	(0.00010)
Mean	0.10	0.01	0.10	0.01
N	66576	66576	66576	66576
R2	0.12	0.55	0.12	0.55

(Clustered SE) by Chiefd; Time FE, Chiefd FE

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. $conflict \in \{0,1\}$ indicates whether there has been any conflict or not in a given period. *ebola* is the number Ebola cases in 100'000 per capita. $ebola + suspect$ is the number Ebola cases including suspect cases in 100'000 per capita.

Table A.17: Spillovers in conflict incidence?

	Conflict Others		Conflict Others pre-Ebola	Neighb Ebola		
	(1) conflict(t)	(2) conflict(t)	(3) conflict(t)	(4) conflict(t)	(5) conflict(t)	(6) conflict(t)
conflict-Others(t)	5271.1					
	(3501.3081)					
conflict-Others(t-1)		592.3	1285.1			
		(861.2889)	(2718.6160)			
ebolaCum-Neighb(t-1)				0.0000296		
				(0.0000)		
ebola-Neighb(t-1)					0.000681	
					(0.0009)	
ebola-Neighb(t-1) $\in \{0,1\}$						0.0920
						(0.0678)
N	67160	66576	29784	66576	66576	66576
R2	0.124	0.124	0.172	0.124	0.124	0.124
Time FE	Y	Y	Y	Y	Y	Y
Chiefd FE	Y	Y	Y	Y	Y	Y

(Clustered SE) by Dist

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. $conflictOthers$ is the presence of conflict in the region except the own. $ebola - Neighb$ is the number Ebola cases in 100'000 per capita in neighboring locations. $ebolaCum - Neighb$ is the cumulative number of Ebola infections over time in neighboring locations. $ebola - Neighb \in \{0,1\}$ is the presence of Ebola in neighboring regions (reduced form effect).

Table A.18: Margins of response

	(1) conflict(t)	(2) conflict(t)	(3) conflict(t)	(4) conflict(t)	(5) conflict(t)	(6) conflict(t)	(7) conflict(t)
ebola(t-1)	0.0146***	0.0141***				0.0148***	0.111**
	(0.0041)	(0.0039)				(0.0053)	(0.0511)
ebolaCum(t-2)		0.000464*					
		(0.0003)					
Ebola(t-1)			0.00215***	0.00213***			
			(0.0006)	(0.0006)			
EbolaCum(t-2)				0.0000561**			
				(0.0000)			
Ebola(t-1) $\in \{0,1\}$					0.209**		
					(0.0872)		
ebola(t-1) $\times$ PostPeak						-0.000467	
						(0.0070)	
ebola(t-1) $\times$ Trend							-0.00132*
							(0.0007)
N	66574	65990	66574	65990	66574	66574	66574
R2	0.143	0.144	0.143	0.143	0.142	0.143	0.143

(Clustered SE) by Dist, Time, Chiefd, Reg $\times$ Month FE

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $ebola$ are Ebola cases in 100'000 per capita, $Ebola$ are the raw number of Ebola cases, $Ebola \in \{0,1\}$ is a dummy variable indicating whether there are Ebola cases or not in a given period. Cum refers to the cumulative. $PostPeak$ is a dummy variable indicating whether the peak of the epidemic has been reached for a given region.

Table A.19: Non-linearities?

	(1) conflict(t)	(2) conflict(t)	(3) conflict(t)	(4) conflict(t)	(5) conflict(t)	(6) conflict(t)
ebola(t-1)	0.0146***	0.0200***	0.0141***	0.0192***	0.0145***	0.0198***
	(0.0041)	(0.0061)	(0.0039)	(0.0057)	(0.0041)	(0.0058)
ebola^2(t-1)		-0.0000282**		-0.0000265*		-0.0000271**
		(0.0000)		(0.0000)		(0.0000)
ebolaCum(t-1)			0.000464*	0.000424	-0.000153	-0.000215
			(0.0003)	(0.0003)	(0.0005)	(0.0005)
ebolaCum^2(t-1)					0.000000472*	0.000000488*
					(0.0000)	(0.0000)
Observations	66574	66574	65990	65990	65990	65990
Adjusted R^2	0.109	0.109	0.109	0.110	0.109	0.110

(Clustered SE) by Dist, Time, Chiefd, Reg × Month FE

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. $ebolaCum$ is the cumulative number of cases over time in 100'000 per capita.

Table A.20: Countries (standardized coefficients)

Outcome: conflict(t)	All	Liberia	Sierra Leone	Guinea
ebola(t-1)	0.0141***	0.0297***	0.0161***	0.00341
	(0.00388)	(0.00698)	(0.00478)	(0.00271)
ebolaCum(t-2)	0.000464*	-0.000782	0.000748***	0.000386
	(0.000272)	(0.000593)	(0.000106)	(0.000905)
N	65990	10396	17061	38533
Time FE	Y	Y	Y	Y
Chiefd FE	Y	Y	Y	Y
Reg × Month FE	Y	Y	Y	Y

(Clustered SE) by Dist

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. $ebolaCum$ is the cumulative number of cases over time in 100'000 per capita.

Table A.21: State response Predictors: Infrastructure and Correlates of disease spread

	(1) Quarantine	(2) ETU	(3) Lab	(4) CCC	(5) None
log(ProxEpic)	0.162***	0.009	0.103**	0.009	-0.156***
	(0.043)	(0.048)	(0.046)	(0.056)	(0.051)
log(ProxEpic)^2	-0.014***	-0.001	-0.006	-0.002	0.014***
	(0.004)	(0.004)	(0.004)	(0.005)	(0.004)
log(Population)	-0.045	-0.118**	0.035	0.026	0.016
	(0.058)	(0.055)	(0.062)	(0.065)	(0.072)
log(Popdens)	0.039	0.179***	0.032	-0.029	-0.034
	(0.065)	(0.059)	(0.062)	(0.062)	(0.064)
Hospitals	-0.017	-0.056	0.071	0.289***	0.026
	(0.133)	(0.106)	(0.091)	(0.089)	(0.102)
Electricity	0.007	0.150	0.221	0.342**	-0.390***
	(0.206)	(0.128)	(0.210)	(0.151)	(0.144)
Water	0.124	0.197*	0.080	0.015	0.044
	(0.159)	(0.106)	(0.153)	(0.145)	(0.125)
Army	0.008	0.104	0.208	0.094	-0.136
	(0.260)	(0.103)	(0.147)	(0.172)	(0.217)
Police	-0.197	-0.193*	-0.073	-0.095	0.287
	(0.257)	(0.109)	(0.100)	(0.199)	(0.225)
Paved Roads	0.307*	0.794***	-0.203*	-0.418**	-0.012
	(0.181)	(0.083)	(0.107)	(0.159)	(0.098)
Blocked Local Roads	-2.993***	-1.165***	-0.851**	-1.663***	3.944***
	(0.473)	(0.291)	(0.341)	(0.570)	(0.305)
Mean	0.36	0.11	0.10	0.31	0.47
N	219	219	219	219	219
R2	0.52	0.28	0.22	0.27	0.43
Country FE	Y	Y	Y	Y	Y

(Clustered SE) by Dist

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: All variables are pre-determined, measured before the start of the outbreak. Outcomes: military district-quarantine, Ebola treatment units (ETUs), Laboratories, Community care centers (CCCs) $\in \{0, 1\}$. *ProxEpic* is the normalized inverse distance to the epicenter, or first index case.

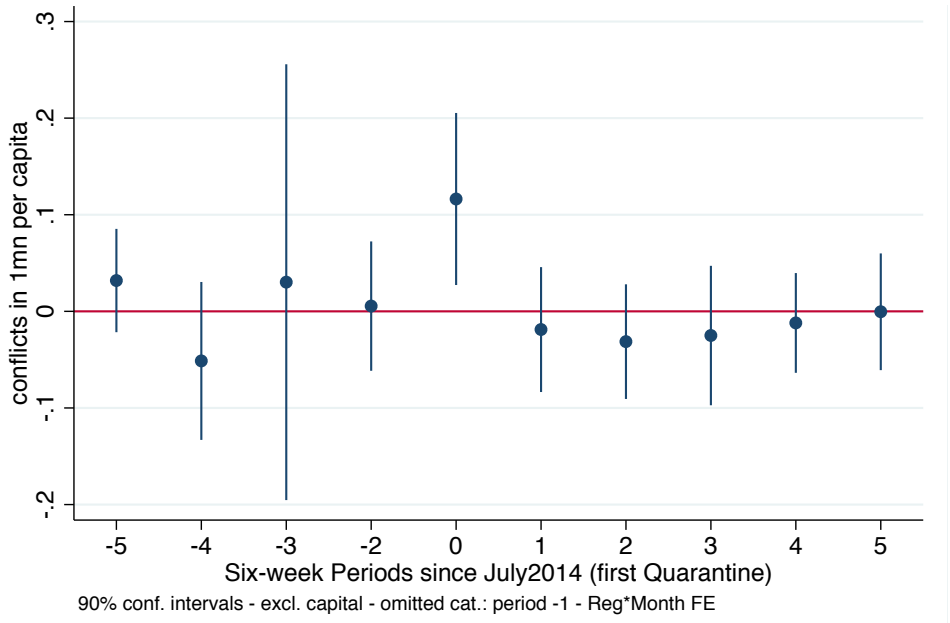
Table A.22: State response: Political motives?

	(1) Quarantine	(2) ETU	(3) Lab	(4) CCC	(5) None
Ethn Polariz.	-0.214	-0.253	-0.283	-0.158	0.031
	(0.223)	(0.218)	(0.242)	(0.264)	(0.218)
Ethn Fractionaliz.	0.532	0.744**	0.425	0.550	-0.397
	(0.334)	(0.303)	(0.327)	(0.345)	(0.271)
Ethnic Salience	0.092**	0.067**	0.047	0.008	-0.083*
	(0.040)	(0.033)	(0.036)	(0.054)	(0.046)
Strongly Relig.	0.019	0.010	0.006	-0.004	-0.006
	(0.019)	(0.023)	(0.023)	(0.030)	(0.021)
Traditional Relig.	0.002*	0.004**	0.001	-0.001	-0.001
	(0.001)	(0.001)	(0.002)	(0.002)	(0.001)
Trust People	0.001	0.011	-0.027**	-0.024*	0.009
	(0.010)	(0.012)	(0.012)	(0.014)	(0.013)
Trust President	0.319***	-0.014	0.101*	0.105	-0.155*
	(0.078)	(0.041)	(0.057)	(0.098)	(0.089)
Trust Opposition	0.208***	-0.087	0.092	0.073	-0.119
	(0.073)	(0.084)	(0.069)	(0.088)	(0.079)
Trust Local Instit.	-0.329***	0.003	-0.155**	-0.066	0.223**
	(0.083)	(0.059)	(0.062)	(0.105)	(0.092)
Trust Army	-0.178*	-0.034	0.016	0.002	0.050
	(0.104)	(0.070)	(0.071)	(0.078)	(0.091)
Trust Leaders	-0.246***	-0.131	-0.006	-0.164	0.287***
	(0.065)	(0.082)	(0.073)	(0.122)	(0.092)
Mean	0.36	0.11	0.10	0.31	0.47
N	219	219	219	219	219
R2	0.63	0.36	0.33	0.31	0.50
Country FE	Y	Y	Y	Y	Y

(Clustered SE) by Dist; cond. on Infrastructure, Epicenter proximity, Population.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

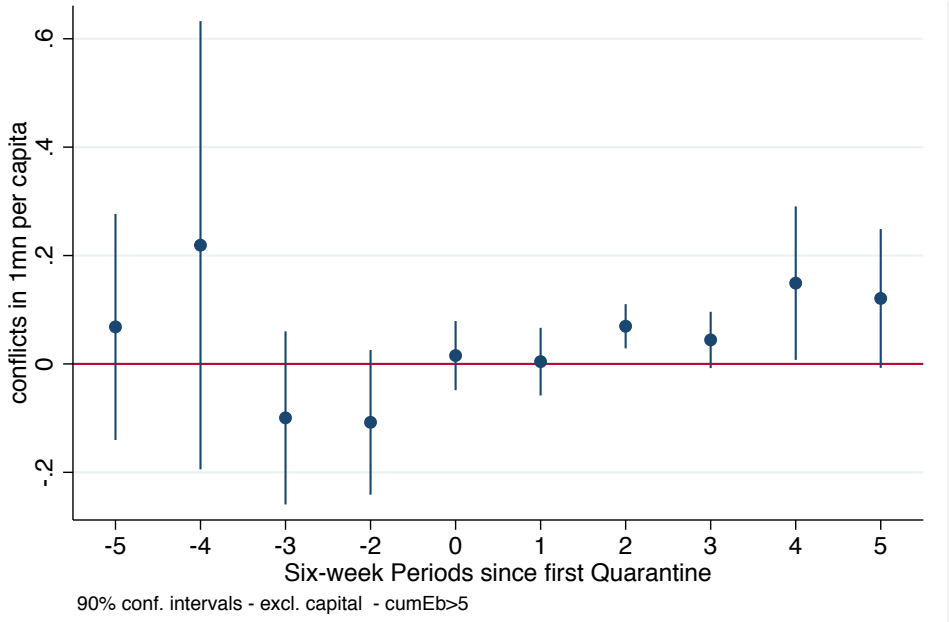
Notes: All variables are pre-determined, measured before the start of the outbreak. Outcomes: military district-quarantine, Ebola treatment units (ETUs), Laboratories, Community care centers (CCCs) $\in \{0, 1\}$.

Figure A.12: Difference-in-Difference in Quarantines per cap.



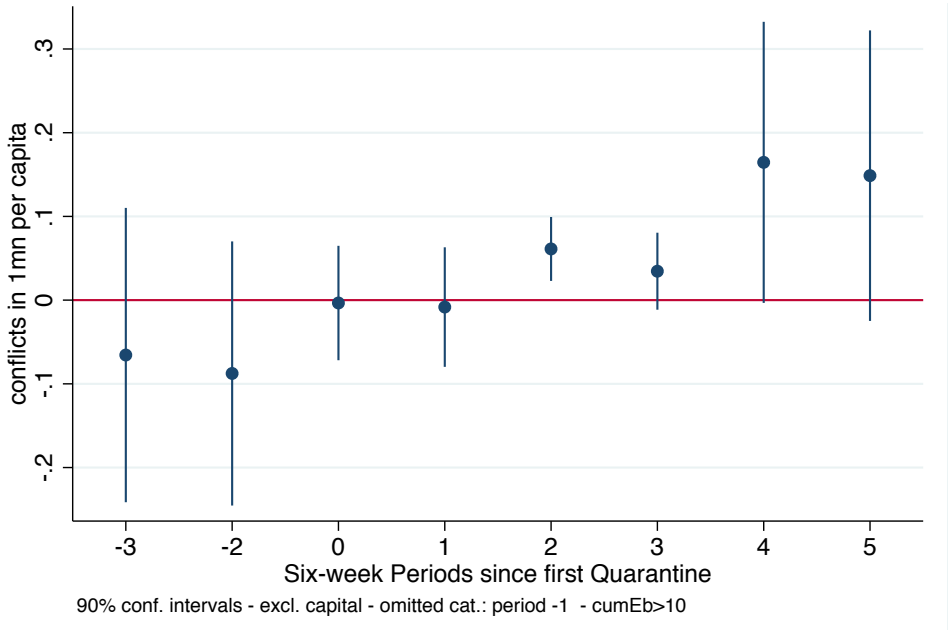
Notes: The figure shows the change in conflict incidence over time due to an additional quarantine in 100'000 per capita. Regression of conflict in 1mn per capita on 6-week time dummies interacted with an ever/never quarantine dummy divided by population per 100'000. Time 0 is in July 2014, which is the first time a quarantine was ever established.

Figure A.13: Event study before/after actual Quarantine - cond. on 5 Ebola cases



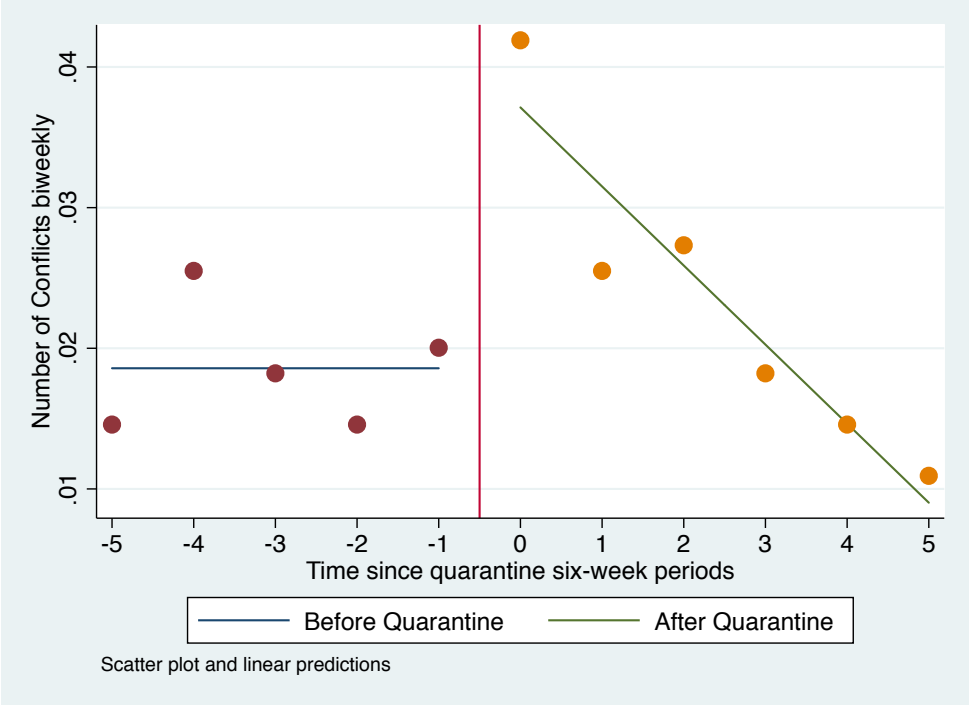
Notes: Change in conflict incidence after the imposition of a quarantine (population-weighted). Regression of conflict in 1mn per capita on 6-week time dummies divided by population per 100'000, only for ever quarantined locations. Time 0 is the date of the establishment of a quarantine.

Figure A.14: Event study before/after actual Quarantine - cond. on 10 Ebola cases



Notes: Change in conflict incidence after the imposition of a quarantine (population-weighted). Regression of conflict in 1mn per capita on 6-week time dummies divided by population per 100'000, only for ever quarantined locations. Time 0 is the date of the establishment of a quarantine.

Figure A.15: Scatter plot and linear prediction before/after Quarantine



Notes: Conflict incidence for ever-quarantined locations, before and after the Quarantine.

Table A.23: Military district-quarantines - OLS results (restricted to ever quarantined locations)

Outcome: conflict(t)	Pre-Quar		Pre/Post-Quar			
	(1)	(2)	(3)	(4)	(5)	(6)
WithinQuar			0.371*	0.446**		
			(0.1960)	(0.1918)		
PostQuar					0.436	0.550*
					(0.3054)	(0.2886)
PreQuar × Trend	-0.00560	-0.00696*				
	(0.0038)	(0.0035)				
ebola(t-1)		0.00938***		0.00897**		0.00937***
		(0.0033)		(0.0035)		(0.0033)
ebolaCum(t-2)		-0.00150**		-0.00174**		-0.00151**
		(0.0006)		(0.0007)		(0.0007)
N	1348	1233	1348	1233	1348	1233
R2	0.193	0.209	0.190	0.205	0.192	0.209
Time FE	Y	Y	Y	Y	Y	Y
Chiefd FE	Y	Y	Y	Y	Y	Y
cumEbola>20	Y	Y	Y	Y	Y	Y

(Clustered SE) by Chiefd

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. $ebolaCum$ is the cumulative number of cases in 100'000 per capita. $WithinQuar$ is a dummy variable taking value 1 if the location is currently under a military quarantine and 0 otherwise. $PostQuar$ takes value 1 if the location has already been quarantined once and 0 otherwise.

Table A.24: Public Goods: Heterogeneous effects with Health Centers - Full Specification

	(1) conflict(t)	(2) conflict(t)	(3) conflict(t)	(4) conflict(t)	(5) conflict(t)
ebola(t-1)	0.0220***	0.00214	0.0195**	0.0301	0.0140
	(0.0071)	(0.0039)	(0.0079)	(0.0192)	(0.0098)
ebola(t-1) × PostEmerg	-0.0177***	-0.00343	-0.0157***	-0.0309	-0.0172*
	(0.0059)	(0.0039)	(0.0056)	(0.0194)	(0.0102)
ebola(t-1) × NearETU^{end}		0.0368***			0.0376***
		(0.0052)			(0.0093)
ebola(t-1) × PostEmerg × NearETU^{end}		-0.0209***			-0.0226**
		(0.0055)			(0.0087)
NearETU^{end} × PostEmerg		0.196**			0.209**
		(0.0895)			(0.0945)
ebola(t-1) × NearLab^{end}			0.0270		0.00133
			(0.0360)		(0.0369)
ebola(t-1) × PostEmerg × NearLab^{end}			-0.0188		0.00789
			(0.0378)		(0.0386)
NearLab^{end} × PostEmerg			0.0171		-0.0438
			(0.0695)		(0.0681)
ebola(t-1) × NearCCC^{end}				-0.0102	-0.0154
				(0.0211)	(0.0099)
ebola(t-1) × PostEmerg × NearCCC^{end}				0.0178	0.0175*
				(0.0209)	(0.0101)
NearCCC^{end} × PostEmerg				0.0467	0.0540
				(0.0502)	(0.0536)
N	36480	33516	33516	36480	33516
R2	0.010	0.013	0.012	0.010	0.014

(Clustered SE) by Dist; Excl. capital; only Epidemic period; Time FE, Chiefd FE, Reg × Month FE

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. $ebolaCum$ is the cumulative number of cases in 100'000 per capita. $PostEmerg$ is a post-treatment dummy taking value 1 after September 2014, when a great amount of emergency assistance is released. $NearETU_i^{end}$ is the normalized inverse distance to the closest Ebola treatment unit (ETU) ever available to a given location. $NearLab_i^{end}$ is the normalized inverse distance to the closest Laboratory for rapid testing of the Virus ever available to a given location. $NearCCC_i^{end}$ is the normalized inverse distance to the closest Community Care Center (CCC) ever available to a given location.

Table A.25: Public Goods: Heterogeneous effects with Health Centers - cond. on cumulative Ebola

	(1) conflict(t)	(2) conflict(t)	(3) conflict(t)	(4) conflict(t)	(5) conflict(t)
ebola(t-1)	0.0221***	0.00225	0.0196**	0.0302	0.0139
	(0.0071)	(0.0039)	(0.0079)	(0.0192)	(0.0098)
ebola(t-1) × PostEmerg	-0.0200***	-0.00460	-0.0181***	-0.0317	-0.0176*
	(0.0066)	(0.0039)	(0.0066)	(0.0194)	(0.0102)
ebola(t-1) × NearETU^{end}		0.0368***			0.0375***
		(0.0052)			(0.0093)
ebola(t-1) × PostEmerg × NearETU^{end}		-0.0229***			-0.0241***
		(0.0059)			(0.0090)
ebola(t-1) × NearLab^{end}			0.0270		0.00152
			(0.0360)		(0.0369)
ebola(t-1) × PostEmerg × NearLab^{end}			-0.0184		0.00786
			(0.0377)		(0.0387)
ebola(t-1) × NearCCC^{end}				-0.0101	-0.0153
				(0.0211)	(0.0099)
ebola(t-1) × PostEmerg × NearCCC^{end}				0.0157	0.0163
				(0.0213)	(0.0102)
N	36160	33222	33222	36160	33222
R2	0.010	0.014	0.012	0.010	0.014

(Clustered SE) by Dist; Excl. capital; only Epidemic period; Time FE, Chiefd FE

Cond. on cumEbola. Omitted: ETU*PostEmerg, Lab*PostEmerg, CCC*PostEmerg

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. $ebolaCum$ is the cumulative number of cases in 100'000 per capita. $PostEmerg$ is a post-treatment dummy taking value 1 after September 2014, when a great amount of emergency assistance is released. $NearETU_i^{end}$ is the normalized inverse distance to the closest Ebola treatment unit (ETU) ever available to a given location. $NearLab_i^{end}$ is the normalized inverse distance to the closest Laboratory for rapid testing of the Virus ever available to a given location. $NearCCC_i^{end}$ is the normalized inverse distance to the closest Community Care Center (CCC) ever available to a given location.

Table A.26: Public Goods: Heterogeneous effects with Health Centers - interaction with High Ebola

	(1) conflict(t)	(2) conflict(t)	(3) conflict(t)	(4) conflict(t)	(5) conflict(t)
ebola(t-1)	0.114	0.102	0.118	0.118	0.107
	(0.1146)	(0.1184)	(0.1205)	(0.1143)	(0.1175)
ebola(t-1) × PostEmerg	-0.126	-0.120	-0.131	-0.132	-0.127
	(0.1126)	(0.1168)	(0.1183)	(0.1122)	(0.1157)
ebola(t-1) × I(HighEbola)	-0.0923	-0.100	-0.0988	-0.0882	-0.0939
	(0.1144)	(0.1187)	(0.1205)	(0.1135)	(0.1169)
ebola(t-1) × PostEmerg × I(HighEbola)	0.108	0.117	0.115	0.102	0.110
	(0.1125)	(0.1171)	(0.1182)	(0.1115)	(0.1150)
ebola(t-1) × NearETU^{end}		0.0369***			0.0375***
		(0.0053)			(0.0093)
ebola(t-1) × PostEmerg × NearETU^{end}		-0.0188***			-0.0203**
		(0.0057)			(0.0091)
ebola(t-1) × NearLab^{end}			0.0272		0.00228
			(0.0360)		(0.0368)
ebola(t-1) × PostEmerg × NearLab^{end}			-0.0186		0.00635
			(0.0374)		(0.0382)
ebola(t-1) × NearCCC^{end}				-0.00986	-0.0146
				(0.0209)	(0.0095)
ebola(t-1) × PostEmerg × NearCCC^{end}				0.0180	0.0171*
				(0.0202)	(0.0095)
N	36480	33516	33516	36480	33516
R2	0.010	0.013	0.012	0.010	0.013

(Clustered SE) by Dist; Excl. capital; only Epidemic period; Time FE, Chiefd FE

Omitted: ETU*PostEmerg, Lab*PostEmerg, CCC*PostEmerg High*PostEmerg

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. $ebolaCum$ is the cumulative number of cases in 100'000 per capita. $PostEmerg$ is a post-treatment dummy taking value 1 after September 2014, when a great amount of emergency assistance is released. $NearETU_i^{end}$ is the normalized inverse distance to the closest Ebola treatment unit (ETU) ever available to a given location. $NearLab_i^{end}$ is the normalized inverse distance to the closest Laboratory for rapid testing of the Virus ever available to a given location. $NearCCC_i^{end}$ is the normalized inverse distance to the closest Community Care Center (CCC) ever available to a given location.

Table A.27: Is Total Conflict predictive of the amount of Public Goods? (correlations)

	CCC		ETU		Lab	
	(1) Nr.CCCs	(2) NearCCC	(3) Nr.ETUs	(4) NearETU	(5) Nr.Labs	(6) NearLab
$Confl^{Epidemic}$	23.8*	23.8*	-1.8	-6.12	4.15	1.76
	(12.4)	(12.4)	(9.44)	(8.54)	(7.19)	(7.73)
Mean	0.31	0.11	0.11	0.09	0.10	0.08
N	572	572	572	540	572	540
R2	0.38	0.38	0.31	0.33	0.14	0.14
Region FE	Y	Y	Y	Y	Y	Y

(Clustered SE) by Dist; Controls: Ebola Total, popdens, NearEpic

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: Outcomes: number or inverse distance to Ebola treatment units (ETUs), Laboratories, Community care centers (CCCs).

Table A.28: Economic mechanism?

	(1) conflict(t)	(2) conflict(t)	(3) conflict(t)	(4) conflict(t)	(5) conflict(t)
ebola(t-1)	0.0186*	0.0208***	0.0191*	0.0218***	
	(0.0102)	(0.0054)	(0.0096)	(0.0053)	
PricePalmOil(t-1)		0.0000159		0.0000153	0.00000581
		(0.0000)		(0.0000)	(0.0000)
PriceImpRice(t-1)			-0.000143*	-0.000132*	-0.000113
			(0.0001)	(0.0001)	(0.0001)
Observations	7714	7272	7309	7272	7272
R^2	0.067	0.068	0.068	0.068	0.068

(Clustered SE) by Dist, with Time FE, Chiefd FE

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita in the own region. *ebola* is the number Ebola cases in 100'000 per capita. $ebolaCum$ is the cumulative number of Ebola infections over time. Price of palm oil and price of imported rice is are prices measured at monthly level and collected by (Glennerster et al., 2013) during the epidemic.

Table A.29: Baseline correlates of civil violence

Outcome: conflict(quarter)	Pre-Epidemic	Epidemic
	(1)	(2)
ebola Total per cap.	0.000	0.006***
	(0.000)	(0.001)
Strongly Relig	-0.280	-0.046
	(0.270)	(0.176)
Trad Relig	-0.007	0.003
	(0.012)	(0.010)
Ethnic Salience	-0.136	0.107
	(0.233)	(0.196)
Ethnic Fractionaliz.	1.586	-2.291
	(2.096)	(1.594)
Ethnic Polariz.	-1.342	1.546
	(1.695)	(1.291)
Trust Leaders	0.466	0.157
	(0.509)	(0.481)
Trust People	0.060	0.022
	(0.060)	(0.042)
Trust Local Instit.	-0.691*	-0.173
	(0.359)	(0.304)
Trust Army	-0.144	-0.360
	(0.364)	(0.358)
Mean	0.48	0.73
N	1752	1971
R2	0.03	0.05
Country FE	Y	Y
Time FE	Y	Y

(Clustered SE) by Dist; Controls: ProxEpic, ProxEpic2, Population, Population Density

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: $conflict$ are the number of conflicts in one million per capita over a quarter of a year in the pre-epidemic and in the post-epidemic period. $ebolaTotalpercap.$ is the total cumulative number of Ebola cases in 100'000 per capita measured post epidemic. The covariates are Afrobarometer data measured pre-epidemic and aggregated at our unit of observation (other variables), i.e. chiefdom, district and sub-prefecture for Sierra Leone, Liberia and Guinea, respectively. Trust in people is a summary index statistic grouping trust in neighbors, in other citizens, family members. Incumbent and opposition are summary index statistics grouping trust and votes for the either (including them separately leads to similar results).

Table A.30: Correlates of civil violence - Political preferences, War and Infrastructure

Outcome: conflict(t)	(1)	(2)	(3)	(4)
ebola(t)	0.0145**	0.0158***	0.0167***	0.0169*
	(0.0055)	(0.0057)	(0.0057)	(0.0086)
ebola(t-1) × Incumbent	-0.0000177			
	(0.0019)			
ebola(t-1) × Opposition	-0.000663			
	(0.0026)			
ebola(t-1) × Civil War		-0.145		
		(0.3363)		
ebola(t-1) × Infrastructure^{SumIndex}			0.000189	
			(0.0003)	
ebola(t-1) × Electricity				0.0383***
				(0.0129)
ebola(t-1) × Piped Water				-0.00641
				(0.0088)
ebola(t-1) × Health Centers				-0.0187
				(0.0284)
ebola(t-1) × Markets				-0.0221*
				(0.0118)
ebola(t-1) × Paved Roads				0.826***
				(0.0237)
Mean	0.0968	0.0968	0.0968	0.0968
N	23370	65208	24852	24852
R2	0.0530	0.0392	0.0503	0.0529
Time FE	Y	Y	Y	Y
Chiefd FE	Y	Y	Y	Y

(Clustered SE) by Dist; Time, Chiefd; Control: ebola × DistEpic, ebola × DistEpic^2; Excl. capital

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Notes: t are two-week periods. $conflict$ are the number of conflicts in one million per capita. *ebola* is the number Ebola cases in 100'000 per capita. The covariates are Afrobarometer data measured pre-epidemic and aggregated at our unit of observation (other variables), i.e. chiefdom, district and sub-prefecture for Sierra Leone, Liberia and Guinea, respectively. $Infrastructure$ is a summary index statistic grouping access to roads, water, electricity, hospitals. $CivilWar$ is the sum of conflict events in each location during the wars in Liberia and Sierra Leone, starting with the first available newspaper reports from the ACLED dataset (1997-2003).

Table A.31: Correlates of civil violence - Table of p-values and q-values

	p-values and q-values				
Outcome: conflict(t)	(1)	(2)	(3)	(4)	(5)
ebola(t)	.015	.018	.001	5.18e-08	.541
ebola(t-1) × Strongly Relig.	**.059**	**.049**			**.048**
	.164	.149			.149
	.142	.132			.132
ebola(t-1) × Tradit. Relig		.235			.381
		.433			.599
		.355			.519
ebola(t-1) × Ethnic Fractional.			**.028**		.155
			.117		.342
			.101		.261
ebola(t-1) × Ethnic Polariz.			**.037**		**.004**
			.137		**.029**
			.121		**.028**
ebola(t-1) × Ethnic Salience			.364		.335
			.599		.582
			.519		.504
ebola(t-1) × Trust Leaders				**.010**	**.003**
				.050	**.029**
				.042	**.028**
ebola(t-1) × Trust Local Instit.				**.001**	.117
				.012	.277
				.011	.202
ebola(t-1) × Trust President				.408	.229
				.613	.433
				.519	.355
ebola(t-1) × Trust Opposition				.802	.559
				.828	.703
				.761	.621
ebola(t-1) × Trust Army				**.006**	.745
				.037	.828
				.033	.761
ebola(t-1) × Trust People				.784	.212
				.828	.433
				.761	.355

First row: standard p-values.

Second row: q-values introduced by Benjamini and Hochberg (1995).

Third row: sharpened two-stage q-values introduced by Benjamini, Krieger, and Yekutieli (2006).

Notes: This is a table of p-values and q-values corresponding to Table 10. q-values are p-values that are adjusted for the number of multiple hypoheses being tested. We adjust them considering all hypotheses tested in Tables 10 and A.30, following Anderson (2008).

Table A.32: Correlates of civil violence - Political preferences, War and Infrastructure - Table of p-values and q-values

	p-values and q-values			
Outcome: conflict(t)	(1)	(2)	(3)	(4)
ebola(t)	.010	.008	.004	.053
ebola(t-1) × Incumbent	.992			
	.993			
	.823			
ebola(t-1) × Opposition	.797			
	.828			
	.761			
ebola(t-1) × Civil War		.666		
		.787		
		.751		
ebola(t-1) × Infrastructure^{SumIndex}			.574	
			.703	
			.621	
ebola(t-1) × Electricity				**.004**
				.029
				.028
ebola(t-1) × Piped Water				.469
				.674
				.582
ebola(t-1) × Health Centers				.511
				.703
				.621
ebola(t-1) × Markets				**.065**
				.167
				.145
ebola(t-1) × Paved Roads				**4.16e-41**
				.001
				.001

First row: standard p-values.

Second row: q-values introduced by Benjamini and Hochberg (1995).

Third row: sharpened two-stage q-values introduced by Benjamini, Krieger, and Yekutieli (2006).

Notes: This is a table of p-values and q-values corresponding to Table A.30. q-values are p-values that are adjusted for the number of multiple hypoheses being tested. We adjust them considering all hypotheses tested in Tables 10 and A.30, following Anderson (2008).

B Supplementary Appendix

Table B.1: Conflict events reported in Newspapers (1)

Ebola-related violence during the Ebola outbreak in Western Africa

- "At least 21 members of a government outreach team were injured when residents of Wome near Nzerekore attacked them with sticks and stones, thinking they were coming to bring Ebola to the village. 8 of them were killed." 8 reported fatalities, Womey, Guinea, *Agence France Presse*, September 2017.
- "An ambulance carrying suspected Ebola patients crashed into a ditch in the northwestern district of Port Loko after a mob pelted it with stones. No injuries reported." *Port Loko, Sierra Leone, Oct 2014, Agence France Presse*
- "Hundreds of youth violently prevented the installation of an Ebola treatment center, setting fires and breaking furniture." Kissidougou, Guinea, *Agence France Presse*, Dec 2014
- "Residents attacked a group of three police officers and their driver who stopped on their way to a funeral, claiming the victims were spreading Ebola and had killed a local resident whom one of the victims had given a sedative. The mob used machetes." Kindia, Guinea, *Agence France Presse*, Jan 2015
- "Thousands of protesters marched on the main Ebola hospital in Kenema and threatened to burn it down and remove the patients after a rumour spread about "cannabalistic rituals" occurring there; police fired tear gas to disperse the crowd." Kenema, Sierra Leone, *Reuters*, July 2014
- "Heavy rioting took place in the West Point neighbourhood between residents and police in response to the imposition of a curfew and quarantine." Montserrado, Liberia, *FrontPageAfrica* August 2014
- "Protesters barricaded one of the main entrances of the ELWA hospital to protest against the establishment of an Ebola Center in the hospitals compound." Montserrado, Liberia, *The Inquirer*, July 2014
- "An angry crowd confronted Red Cross workers regarding the burial of an Ebola victim. The police tried unsuccessfully to calm the situation as the rioters burned a Red Cross vehicle.", Conakry, Guinea, *Xinhua News*, December 2014

Table B.2: Conflict events reported in Newspapers (2)

Non-Ebola-related violence during the Ebola outbreak in Western Africa

- "Security forces in Labe killed a protester and wounded four others, witnesses said Thursday, as opposition supporters clashed with police at anti-government rallies in its largest towns and cities." 1 reported fatality, Labe, Guinea, *Agence France Presse*, April 2015
- "Twenty people were arrested and two police officers injured during a riot at the Plam Oil Production project in Mattru Jong, after stakeholders incited people against a government takeover of the project." Bonthe, Sierra Leone, *Concord Times* May 2014
- "A motorcyclist was killed by a soldier of the Armed Forces of Liberia following a scuffle on Tuesday in Thinker Village Community in Paynesville. Meanwhile, police have arrested the soldier and are investigating the event." 1 reported fatality. Montserrado, Liberia, *Front Page Africa*, April 2015
- "An unknown number of people are feared injured or dead following alleged clashes between members of the traditional Poro or Sande Societies in Grand Bassa County, mainly in Kpokon. Several houses were burnt down in the clashes." 10 fatalities reported." Grand Bassa, Liberia, *Front Page Africa*, April 2016

Table B.3: Conflict events reported in Newspapers (3)

Civil violence in other epidemic outbreaks throughout Africa

- "Cholera treatment centre attacked due to misinformation and suspicion among locals that it was spreading the disease", Ancuabe, Mozambique, *All Africa*, 2009.
- "A Frelimo branch secretary was seized by an angry mob and killed, over accusations that party leaders were spreading cholera", Maputo, Mozambique, *Agencia de Informacao de Mocambique*, 2013.
- "A Frelimo official and community leader in Mecufi, was buried alive, up to his neck, and then killed by an angry mob over accusations that party leaders were spreading cholera in Macomia district", *Agencia de Informacao de Mocambique*, 2013.
- "Dodoth warriors kill UPDF soldier who had been suffering from malaria", Kalapata, Uganda, *All Africa*, 2009.
- "Two men where alledgedly killed by their sons after the sons accused their fathers of being 'witches', following the deaths of a sister and brother due to childbirth and malaria respectively", Buba, DRC, *Radio Okapi*, 2016.
- "500 AIDS activists marched by a police station to protest police brutality", Johannesburg, South Africa, *Reuters News*, 2003.

Table B.4: Newspapers pre and post Epidemic - Guinea

Newspapers reporting on conflict in Guinea	Number of Articles		
	pre-Ebola	post-Ebola	Total
Agence France Presse	29	35	64
Aminata	24	23	47
Associated Press	12	6	18
Guineenews	3	7	10
Media Foundation for West Africa	2	1	3
Radio France Internationale	11	4	15
Reuters	2	1	3
Xinhua	10	17	27
Africa News	1	0	1
Afrik.com	12	0	12
AfriquInfos	1	0	1
All Africa	5	0	5
AngolaPress	9	0	9
Committee to Protect Journalists	1	0	1
Daily Record	1	0	1
Guinee 7	2	0	2
Human Rights Watch (Washington, DC)	1	0	1
IHS Global Insight Daily Analysis	1	0	1
Jeune Afrique	4	0	4
Kankan Radio	1	0	1
Le Point	2	0	2
Lejourguinee.com	3	0	3
Relief Web	2	0	2
Reuters Africa	1	0	1
Slate Afrique	1	0	1
Sunday Mail	1	0	1
Xinau General News	3	0	3
Mali Actu	1	0	1
AlertNet (London)	0	1	1
Amnesty International	0	1	1
Daily Independent (Lagos)	0	1	1
Deutsche Welle	0	2	2
France 24	0	2	2
International Freedom of Expression Exchange Clearing House	0	1	1
PBS News Hour	0	1	1
Syndigate Media	0	1	1
Video News	0	1	1
Voice of America (Washington, DC)	0	1	1
Total	146	106	252

Table B.5: Newspapers pre and post Epidemic - Sierra Leone

Newspapers reporting on conflict in Sierra Leone	Number of Articles		
	pre-Ebola	post-Ebola	Total
Associated Press International	1	1	2
Awareness Times	5	2	7
Concord Times	6	14	20
APANEWS	1	0	1
Agence de Presse Africaine	1	0	1
Cocoricko	2	0	2
Public Agenda	1	0	1
Think Africa Press	2	0	2
Agence France Presse	0	6	6
Al Jazeera - English	0	1	1
Aminata	0	1	1
Awoko.org	0	12	12
BBC News	0	1	1
CBC News	0	1	1
Media Foundation for West Africa	0	2	2
Pan African News Agency	0	4	4
Reuters	0	4	4
Star Africa	0	1	1
The Mercury (South Africa)	0	1	1
Vice News	0	1	1
Voice of America (Washington, DC)	0	1	1
Total	19	53	72

Table B.6: Newspapers pre and post Epidemic - Liberia

Newspapers reporting on conflict in Liberia	Number of Articles		
	pre-Ebola	post-Ebola	Total
Agence France Presse	4	5	9
Associated Press	1	2	3
Front Page Africa	6	2	8
Heritage	5	18	23
New Dawn Liberia	5	1	6
New Democrat	14	1	15
The Analyst	7	2	9
The Inquirer	4	21	25
The NEWS	6	13	19
The New Dawn	15	55	70
The New Republic Liberia	5	14	19
All Africa	2	0	2
Liberian News	2	0	2
Liberian Times	3	0	3
Reuters	1	0	1
The Front Page Africa	1	0	1
The Informer	4	0	4
The Liberian Times	4	0	4
The New Zealand Herald	2	0	2
Voice of America	1	0	1
Associated Press International	0	1	1
Daily Observer	0	1	1
Foreign Policy	0	1	1
FrontPageAfrica	0	49	49
GNN Liberia	0	1	1
International Business Times	0	2	2
International Freedom of Expression Exchange Clearing House	0	2	2
Leadership (Abuja)	0	1	1
Liberia News Agency	0	6	6
Liberian Observer	0	18	18
PBS News Hour	0	1	1
Radio France Internationale	0	2	2
Reporters Sans Frontieres (RSF)	0	1	1
Syndigate Media	0	4	4
The Mercury (South Africa)	0	1	1
Total	92	225	317

Figure B.1: Raw data - Weekly Ebola cases and Conflict incidence for each prefecture in Guinea (1)

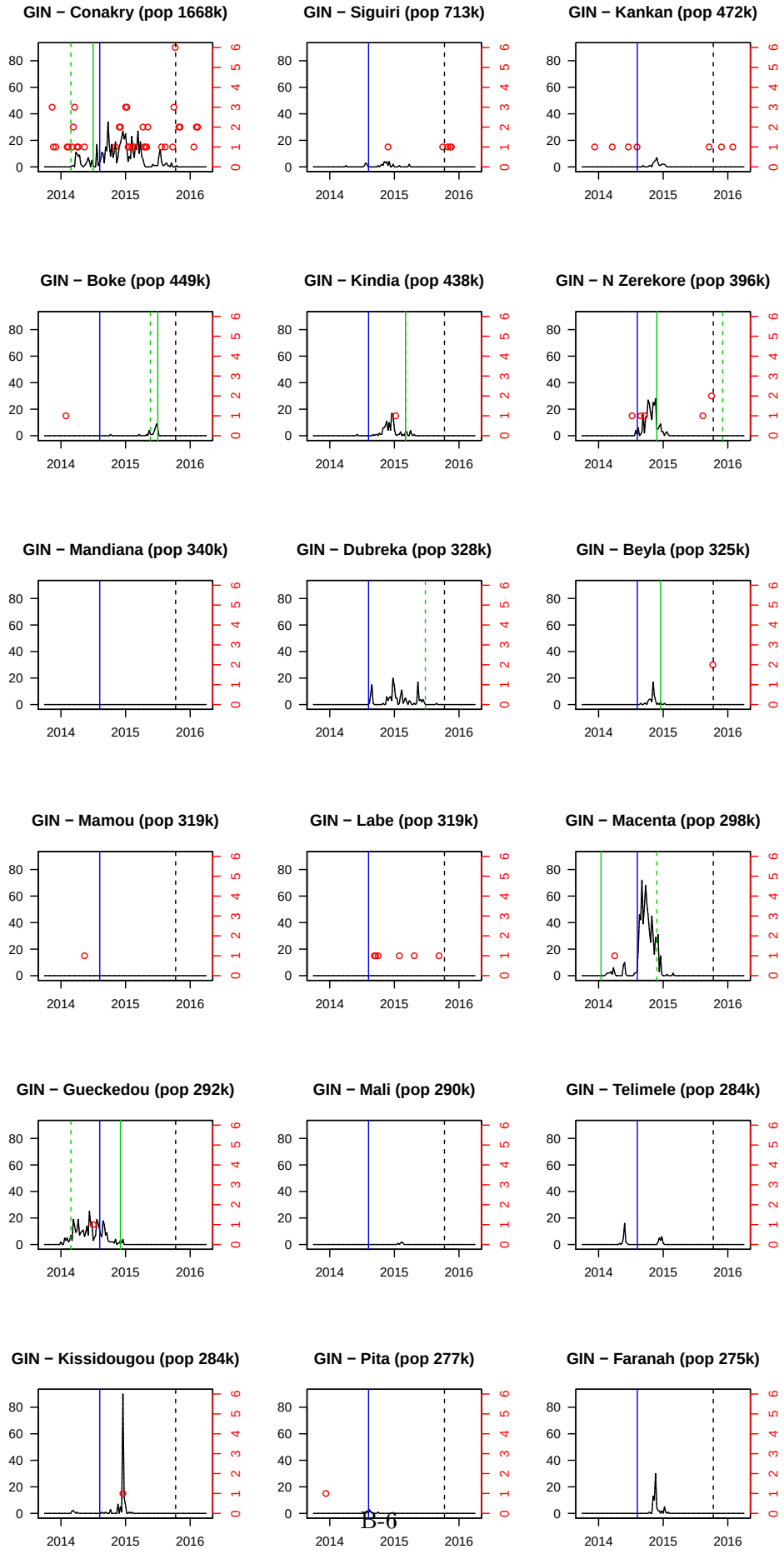


Figure B.2: Raw data - Weekly Ebola cases and Conflict incidence for each prefecture in Guinea (2)

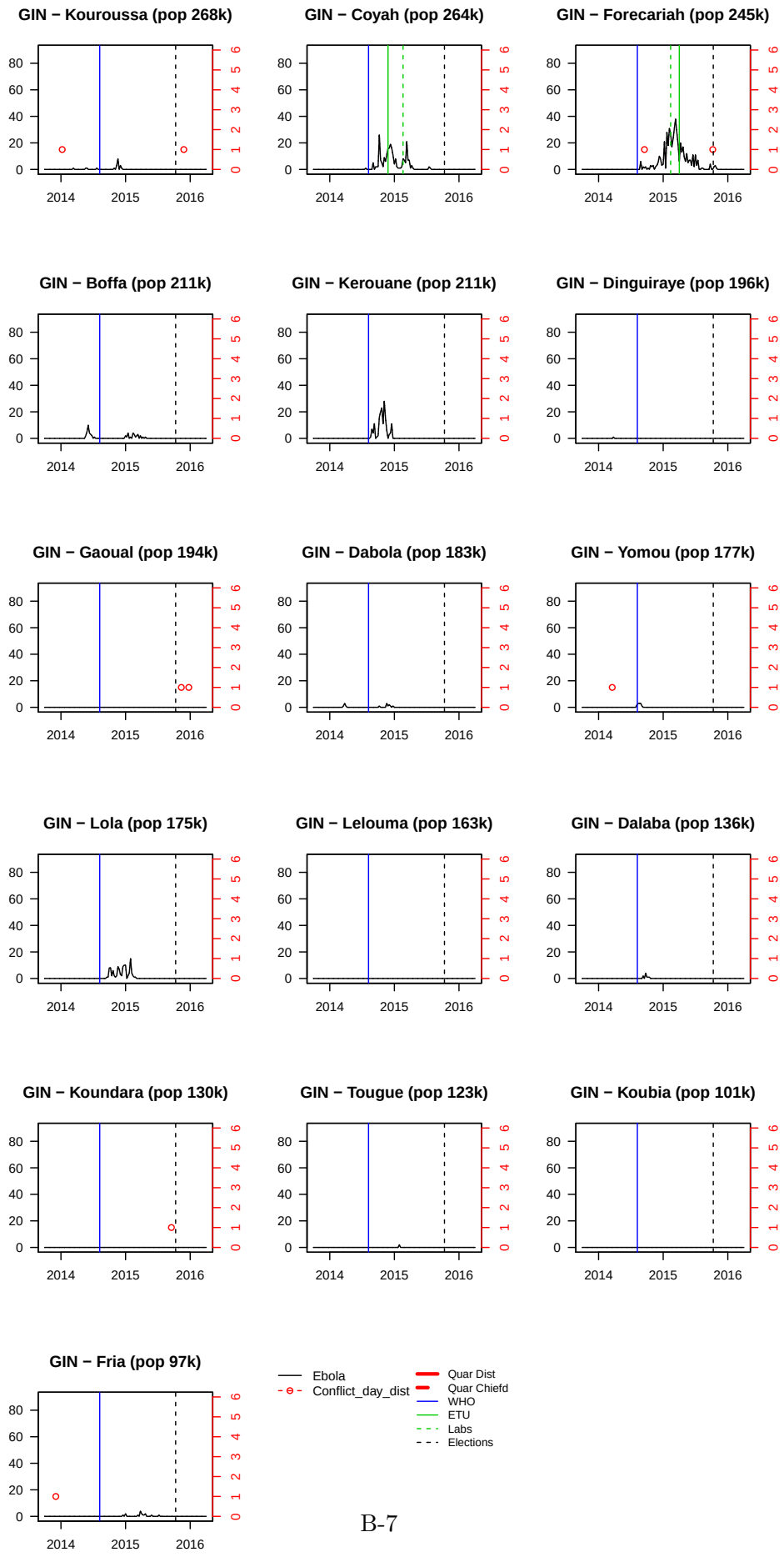


Figure B.3: Raw data - Weekly Ebola cases and Conflict incidence for each county in Liberia

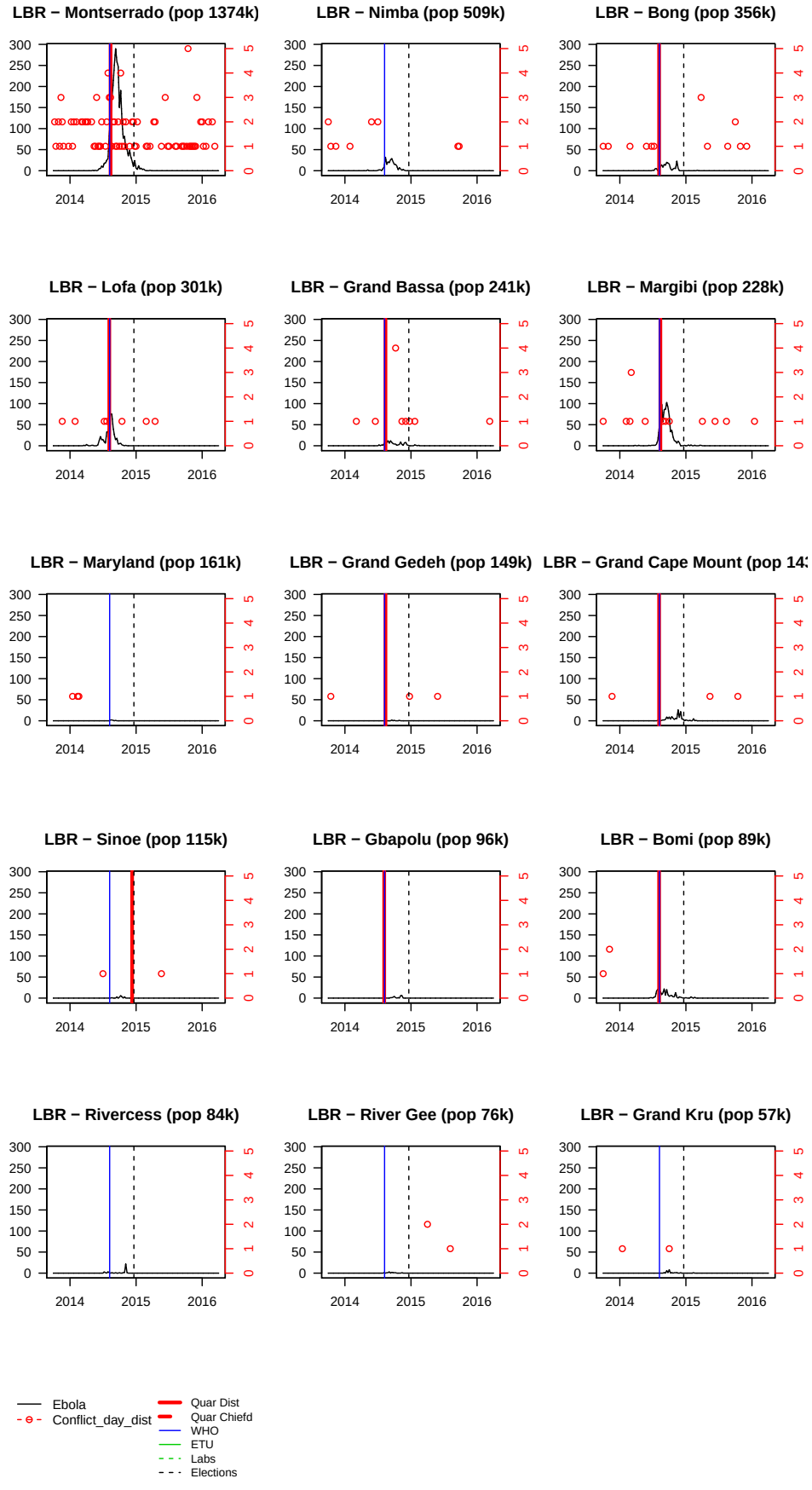


Figure B.4: Raw data - Weekly Ebola cases and Conflict incidence for each district in Sierra Leone