# Ebola and State Legitimacy*

Matthias Flückiger† Markus Ludwig‡ Ali Sina Önder§

**Abstract**

We exploit the recent West African Ebola epidemic as an event that necessitated the provision of a common-interest public good, Ebola control measures, to empirically investigate the effect of public good provision on state legitimacy. Our regression results show that state legitimacy, measured by trust in central authorities and willingness to pay taxes, increased disproportionately in districts that experienced a greater exposure to Ebola. We argue, supported by results from SMS-message-based surveys, that one potentially important channel underlying this finding is a greater valuation of Ebola control measures in regions with intense Ebola transmission. Evidence further indicates that the effects of Ebola exposure are more pronounced in regions where the governments responded relatively robustly to the epidemic. To address concerns related to the possibility that the spread and intensity of the epidemic are influenced by local differences in state legitimacy, we rely on a 2SLS-IV approach for identification. Observed Ebola case numbers are instrumented with simulation-derived predicted numbers. Variation in timing and intensity of the simulated EVD epidemic is restricted to aspects that are plausibly exogenous with respect to changes in legitimacy.

JEL CLASSIFICATION: D7, I15, H12, H51, C36
KEYWORDS: State Legitimacy, Ebola, West Africa

*We are grateful for valuable comments received from the editor, two anonymous referees, Arcangelo Dimico, Hartmut Egger, Erik Hornung, and David Stadelmann as well as conference and seminar participants at the 2018 Royal Economic Society conference in Brighton, the Irish Economic Association Annual Conference 2018 in Dublin, the 2018 Workshop on Geodata and Economics in Braunschweig, and Bayreuth University.

†Queen's University Belfast. Email: matthias.flueckiger@qub.ac.uk
‡University of Bayreuth. Email: markus.ludwig@uni-bayreuth.de
§University of Bayreuth. Email: ali-sina.oender@uni-bayreuth.de

# 1 Introduction

State capacity, the state's ability to implement policies, is an important determinant of economic development (Besley and Persson, 2009; Acemoglu et al., 2015). In much of the developing world, states struggle to raise taxes, provide public goods and administer their territories effectively. Lack of legitimacy is potentially an important factor explaining these low levels of state capacity (e.g., North, 1981, p.53; Englebert, 2002, p.92). Without legitimacy, governments cannot rely on citizens to voluntarily comply with centrally mandated policies, making implementation of policies costly, the provision of public goods inefficient and capacity building difficult (e.g., Gilley, 2009, p.147f.; Levi, 2006).[1] Recent theoretical research (Besley and Persson, 2009; Gennaioli and Voth, 2015; Alesina et al., 2017) suggests that the provision of common-interest public goods—typically provoked by external threats such as wars—increased state capacity in historical times. It is unclear, however, whether the provision of common-interest public goods increases state legitimacy and capacity in the modern era. This paper contributes to filling this gap.

We investigate how infectious disease control, a particular type of common-interest public good, influences state legitimacy.[2] For our empirical analysis, we focus on the recent outbreak of the Ebola virus disease (EVD) in West Africa from 2013 to 2016 and the resulting provision of Ebola control measures in the three most affected countries: Guinea, Liberia, and Sierra Leone. The dimension of the West African EVD epidemic was unprecedented, both in terms of geographic spread and intensity. Over the course of the epidemic, more than 28,000 Ebola cases were reported (WHO, 2016b). In addition to direct health effects, the epidemic also disrupted economic and social interactions (Overseas Security Advisory Council, 2015; Bowles et al., 2016). Governments, and under their auspices international organizations, reacted by developing control measures. The implementation of, and compliance with, these public health policies was instrumental in eradicating the disease (e.g., WHO Ebola Response Team, 2016; Blair et al., 2017). Arguably, the effect of the epidemic on state legitimacy depends on people's perception of these policies. In regions where the risk of contracting Ebola is greater, valuation and perception of government-led control efforts is likely higher. Furthermore, there is clear evidence from the political sci-

[1] Legitimacy and trust in government is especially important in the context of public health interventions, where acceptance of, and compliance with, policies is crucial for success (e.g., Alsan and Wanamaker, forthcoming; Blair et al., 2017).

[2] Since transmission occurs via human interaction, infectious diseases represent a negative externality. It therefore lies in the interest of all members of an interconnected group, e.g., a nation, to contain the spread of infectious diseases.

ence literature that the effect of natural disasters on political support crucially depends on the robustness of the government's response, where support increases with effectiveness of the response (e.g., Bechtel and Hainmueller, 2011; Gasper and Reeves, 2011). Similarly, we expect that perception of the state improves with increased quantity and quality of government response.

To empirically assess the validity of these hypotheses, we use pre- and post-epidemic individual-level survey data from the Afrobarometer. The panel-type nature of the data allows us to employ a difference-in-difference estimation strategy in which we can account for time-invariant location-specific characteristics, such structural differences in levels of trust.

Concerns related to measurement error in Ebola case numbers as well as endogeneity between Ebola exposure and legitimacy, however, remain. Endogeneity issues arise because exposure to Ebola as well as the effectiveness of policies potentially depend on state legitimacy (e.g., Blair et al., 2017). Anecdotal evidence indicates that where legitimacy was low, compliance with control measures was initially weak, leading to more intense transmission. To obviate these issues, we rely on a linear 2SLS approach for identification, where observed Ebola prevalence is instrumented with simulated prevalence. The simulations are based on an epidemiological mathematical model in which the outbreak is represented as a network of local epidemics that are interconnected across districts. The model incorporates solely predetermined (and time-invariant) population numbers, Euclidean distances as well as generic parameters of the Ebola disease. Differences in state legitimacy or perception of the state that reportedly influenced spread and intensity of the West African epidemic (Manguvo and Mafuvadze, 2015) do not generate variation in the model. The simulation-derived prevalence thus allows us to identify variation in the observed Ebola prevalence that is plausibly exogenous with respect to local differences in state legitimacy and other socioeconomic characteristics.

Employing our linear 2SLS-IV regressions approach, we find that state legitimacy— proxied by trust in central government (parliament and president) and police—increased disproportionately in regions with higher exposure to the epidemic. An increase in average Ebola prevalence of one case per 100,000 people raises post-epidemic trust in all three government entities by around 0.15 standard deviations compared to the pre-epidemic era. The effect on trust in central state is also reflected in a relative increase in the willingness to pay taxes. To investigate the plausibility of differential valuation of control measures as a potential channel underlying these effects, we conducted a retrospective mobile-phone-based

survey in Liberia. The data show that valuation of Ebola-control-related state interventions is greater among individuals who resided in high Ebola risk regions. Furthermore, we find suggestive evidence that the epidemic's effects on state legitimacy are particularly pronounced in regions that experienced a relatively large influx of relief-effort related resources as well as in areas where implied quality of Ebola control (measured by the difference between simulated and observed prevalence) was comparatively higher. These heterogeneities support the interpretation that our results are reflecting changes in perceptions induced by the governments' response to the epidemic rather than by the exposure to the disease itself. In a final step, we show that the Ebola-epidemic-induced effects on trust are also reflected in voting behaviour. Support for the presidential candidate of the incumbent party increased in areas that saw an intense Ebola transmission relative to regions with low transmission intensities.

An important assumption underlying our empirical strategy is that the simulated diffusion process specifically captures the progression of the Ebola epidemic and influences state legitimacy only via realised Ebola transmission intensity. Falsification tests provide support for the plausibility of these assumptions. We further conduct a number of robustness checks to document the stability of our results. These include the use of alternative measures of Ebola exposure, varying the set of control variables and employing different approaches to compute standard errors.

Overall, our results show that a one-time provision of a common-interest public good—in our case triggered by a devastating epidemic—can increase state legitimacy, an otherwise slowly evolving determinant of economic development, within a short period of time. An implication of this finding is that the supply of public goods could constitute an instrument with which policy makers in developing countries can increase state legitimacy and thereby overcome historically rooted capacity constraints. However, given the short period of time that has passed since the end of the epidemic as well as the transitory nature of the public good, our estimates should only be interpreted as short-run effects. It remains to be seen whether these Ebola-induced attitudinal changes persist. If so, there is hope that the West African Ebola epidemic may—in contrast to the devastating effects on health and economic activity observed in the short run (Bowles et al., 2016)—have positive effects on determinants of long-run growth.

The remainder of this paper is organised as follows: In the next section, we discuss how our study relates and contributes to the literature. In Section 3, we provide background information on the West African Ebola epidemic. We then outline the empirical strategy

in Section 4 before presenting the data and summary statistics in Section 5. The results of the regression analysis are discussed in Section 6. Finally, Section 7 concludes.

# 2 Related Literature

Our work directly relates to the political science literature (mentioned in the introduction) that identifies state legitimacy as a fundamental determinant of state building and economic development (e.g., Migdal, 1988; Weber, 1984; North, 1981; Englebert, 2002) and analyses factors that influence legitimacy (Hutchison and Johnson, 2011; Gilley, 2009). Closely connected to our analysis is further the branch of this literature that investigates the relationship between public good provision, state legitimacy and capacity. The mostly qualitative results indicate that the efficient provision of public goods raises state legitimacy (e.g., Rotberg, 2003; Easton, 1965, p.278; Fukuyama, 2015). Our findings empirically validate the qualitative evidence.

Highly relevant for our paper are further studies that investigate the origin of state capacity and state building. This literature primarily focuses on the analysis of historical roots of capacity building in a general context (e.g., Besley and Persson, 2009, 2010; Gennaioli and Voth, 2015; Alesina et al., 2017). Therein, the provision of common interest public goods is identified as an important factor that facilitates capacity building. A number of recent studies, however, specifically investigate the historical causes of weak state capacity in Africa (e.g., Gennaioli and Rainer, 2007; Nunn, 2008; Nunn and Wantchekon, 2011; Michalopoulos and Papaioannou, 2013). In contrast to our work, the results of these studies are silent about potential factors that induce changes in state capacity in the modern era.

Another body of literature our paper contributes to analyses how the government's response to natural disasters affects political accountability and citizens' perception of the state. Overwhelmingly, the studies find that the electorate rewards incumbents for efficient post-disaster management. Typically, the increase in vote share is interpreted as the consequence of effective disaster relief providing a strong signal of quality and capacity of the government (Healy and Malhotra, 2010; Bechtel and Hainmueller, 2011; Gasper and Reeves, 2011; Cole et al., 2012). In a recent paper, Gallego (2015) proposes aid-induced availability of resources for buying votes as an alternative mechanism underlying the post-disaster increase in support for incumbents. In addition to changing voting behaviour, Fair et al. (2017) show that natural disasters can increase political engagement (turnout and political literacy) and thereby strengthen the democratisation process in developing countries. We

complement this literature by analysing the effects of health epidemics—and the ensuing response of governments—on citizens' attitudes towards the state and support for political parties in power.

Also linked to our work are studies that analyse the association between state capacity and level of development (e.g., Acemoglu et al., 2015; Dell et al., 2017; Dincecco and Katz, 2016). These papers document the crucial importance of state capacity for economic prosperity. In a recent study, Dell and Querubin (2018) document that regions exposed to more intensive bombings during the Vietnam War experienced a decrease in state capacity and legitimacy.

Our paper also builds on, and contributes to, the literature on disease and development. Various channels have been proposed through which adverse disease environments influence economic development. Prominently discussed is the relative importance of direct effects on contemporaneous health (e.g., Gallup and Sachs, 2001; Sachs and Malaney, 2002; Bleakley, 2007) versus indirect effects working through the disease environment's influence on the quality of institutions (e.g., Acemoglu et al., 2001, 2003). Our results suggest the existence of a further channel that links infectious diseases to development: Successful government-led disease control may not only improve public health but also increase state legitimacy and thereby capacity.

Finally, the results of our study also directly speak to the line of recent research concerned with assessing the socio-economic effects of the West African Ebola epidemic. A number of studies document its devastating impact on the economies in the short run (Thomas et al., 2015; Davis, 2015; Bowles et al., 2016). We complement these studies by analysing the epidemic's effect on fundamental factors of growth, rather than looking at direct measures of economic prosperity. Changes in these fundamentals may take a long time to materialise in observable macroeconomic effects.

# 3 Background

Ebola virus disease, first identified in 1976, causes a highly contagious infectious disease that is associated with a case-fatality rate of 69 to 88 percent (Van Kerkhove et al., 2015). Starting point of outbreaks is a transmission event from an unknown animal reservoir into the human population. The virus then spreads among humans via direct contact with bodily fluids of infected individuals (Gire et al., 2014; Rewar and Mirdha, 2014). Absent

a medical cure,[3] preventive measures, such as avoiding physical contact and practising careful hygiene, as well as isolation of infected people (alive and dead) are the sole defenses against the spread of Ebola. Compliance with these infection-control measures are therefore central to successful containment and eradication of the disease (WHO Ebola Response Team, 2016).

In the 41 years since discovery, 24 Ebola outbreaks have been reported. The West African epidemic (2013–2016) stands out as the most intensive and geographically widespread event. In contrast to previous episodes, the outbreak was not restricted to isolated rural areas, but also spread into densely populated regions. Over the course of the epidemic, a total of 28,616 cases were reported, of which 11,310 resulted in death (WHO, 2016b). The overwhelming majority—more than 99.9%—of cases occurred in Guinea, Liberia and Sierra Leone. In addition to direct health effects, the epidemic had an extremely disruptive impact on economic activity and, more generally, social interactions within these three countries (e.g., Overseas Security Advisory Council, 2015; Bowles et al., 2016).

Figure 1, panel (a), depicts the timeline of the epidemic. The first case occurred in Guinea's Guéckédou prefecture in late December 2013. From there, the virus spread to neighbouring Liberia and Sierra Leone. Prevalence rates were relatively low until June 2014, when case numbers dramatically increased. On 7 August 2014, the WHO declared the epidemic a 'Public Health Emergency of International Concern'. Panel (b) in Figure 1 illustrates that the intensification of transmission was not a locally restricted phenomenon. The fraction of districts[4] for which Ebola cases were reported (grey line) also rose substantially starting in June.

Reacting to the intensification of the epidemic, all three countries established a national Ebola task force or committee in charge of coordinating the implementation of control measures. Chairing these groups was the president of the respective country (DuBois et al., 2015; Marston et al., 2017). The government-led control strategies—supported by national and international partners—were initially ineffective in containing the disease. Among the main factors contributing to the sharp increase in case numbers were the lack of resources, such as skilled health care workers[5] and miscommunication (Chan, 2014; WHO, 2014).

[3]At the time of writing, a medical cure or approved vaccine for Ebola does not exist. A clinical vaccination trial conducted during the final phase of the West African epidemic produced promising results (Henao-Restrepo et al., 2017). The vaccine has since been approved for experimental use during the most recent Ebola outbreak in the Democratic Republic of the Congo in May 2017 (Maxmen, 2017).

[4]See Section 5 for a definition of districts.

[5]A report by the WHO (2015), for example, documents that infected health care workers made up 16% of the total Ebola cases during the initial phases of the epidemic. Case fatality rates among health

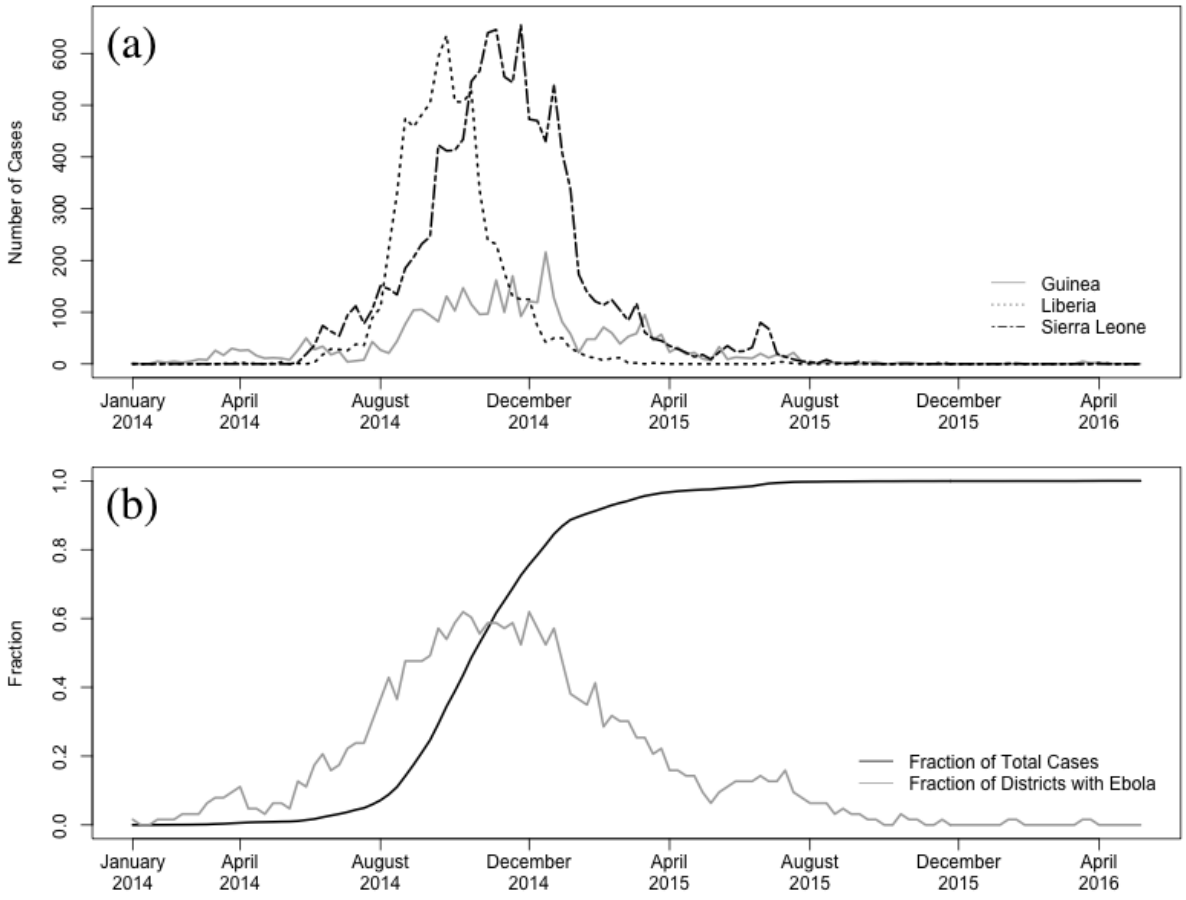


Figure 1: Panel (a) Figure depicts the number of Ebola cases during the epidemic (December 2013–April 2016). Panel (b) shows the cumulative distribution function of total EVD cases as well as the share of total districts in which Ebola was observed in a given week. Data: WHO situation summary, 11 May 2016.

Equally important, low legitimacy of, and trust in, governments and health authorities meant that control measures were not complied with or actively opposed, rendering them ineffective (e.g., Manguvo and Mafuvadze, 2015; World Health Organization, 2015; Blair et al., 2017). However, with increased awareness of the epidemic's dimension as well as the involvement of local traditional leaders and communities, attitudes and behaviours changed in later stages of the epidemic. Resistance largely abated and disease control measures gained legitimacy and were adopted (Mark, 2014; Neal, 2015; Africa APPG, 2016). These attitudinal and behavioural changes—manifested as compliance with control measures—are viewed as central factors that contributed to the reduction in transmission intensities observed between October 2014 and January 2015 (Carrión Martín et al., 2016;

care workers were initially even higher than among non-health care workers (World Health Organization, 2014a).

Tsai et al., 2015). Government-led containment of the epidemic was further facilitated by the increased availability of resources after the intensification of international response in September 2014.[6] On 29 March 2016, the WHO terminated the Public Health Emergency of International Concern status of the outbreak and the epidemic was declared officially over on 9 June 2016. However, Figure 1, panel (b), shows that the vast majority, 85% of all cases, had already occurred in 2014.

First qualitative evidence from post-epidemic surveys indicates that the majority of population judged the national government to have been effective in controlling the epidemic (RIWI, 2015; Armah-Attoh and Okuru, 2016). This suggests that the epidemic—more precisely the ensuing public health interventions—may have improved legitimacy of the state. The remainder of the paper is concerned with empirically assessing the existence of this effect.

# 4 Empirical Strategy

Using simple OLS to estimate the effect of the Ebola epidemic on state legitimacy will likely produce biased coefficients. As discussed in the introduction, reverse causation and measurement error, in particular, are potential sources of bias. To obviate these problems, we employ a 2SLS approach and instrument observed Ebola prevalence with simulated prevalence.[7,8] In a first step, we regress reported EVD prevalence on simulated prevalence. The regression is given by:

$$E_{i,d,c,t} = \theta\, S_{i,d,c,t} + \Omega\, \mathbf{X}_{i,d,c,t} + \alpha_{d,c} + \tau_{c,t} + \psi_{i,d,c,t}. \tag{1}$$

The dependent variable, Ebola prevalence ($E_{i,d,c,t}$), reflects the average number of Ebola cases per 100,000 people in individual $i$'s district of residence $d$ (located in country $c$) in year $t$. Simulated prevalence is represented by $S_{i,d,c,t}$. The vector $\mathbf{X}_{i,d,c,t}$ contains individual-level controls, such as age, sex and ethnicity. In all regressions we allow these characteristics

[6] On 19 September 2014, the UN Security Council created the United Nations Mission for Ebola Emergency Response (UNMEER) with the "objective of scaling up the response on the ground and establishing unity of purpose among responders in support of the nationally led efforts". `http://ebolaresponse.un.org/un-mission-ebola-emergency-response-unmeer`.

[7] The simulations are described in detail in Section 5.2.

[8] A similar identification strategy, in which the effect of a regional shock on individual-level outcomes is estimated using an instrumental variable approach, is employed in Rohner et al. (2013), Autor et al. (2014) and Lowes and Montero (2018).

to have different effects in the pre- and post epidemic era by interacting them with year dummies. District-specific fixed effects are symbolised by $\alpha_{d,c}$, country-time-fixed effects by $\tau_{c,t}$, and the idiosyncratic error term by $\psi_{i,d,c,t}$. All individual-level regressions are weighted using sample weights provided by Afrobarometer; the standard errors are clustered at the district level.

In the second step, we use the predicted values derived from regression Eq.(1) to quantify the effect of Ebola prevalence on state legitimacy. These predictions only reflect the part of the variation in observed prevalence that is due to disease-specific and time-invariant characteristics, which themselves are exogenous with respect to the actual spread of the West African epidemic. The second-stage regression takes the following form:

$$y_{i,d,c,t} = \beta \, \widehat{E}_{i,d,c,t} + \Phi \, \mathbf{X}_{i,d,c,t} + \mu_{d,c} + \gamma_{c,t} + \varepsilon_{d,c,t}, \tag{2}$$

where $y_{i,d,c,t}$ represents the outcome for individual $i$ residing in district $d$ and country $c$ in year $t$. $\widehat{E}_{i,d,c,t}$ symbolises predicted Ebola prevalence obtained from the first-stage regression Eq.(1); the vector $\mathbf{X}_{i,d,c,t}$ represents the individual-level controls. The inclusion of district-fixed effects ($\mu_{d,c}$) and country-time-fixed effects ($\gamma_{c,t}$) implies that—in analogy to a standard difference-in-difference approach—we exploit only within-district variation in our empirical analysis. That is, we abstract from district-specific time-invariant level differences such as differences in ethnic composition, level of economic activity, population composition or attitudes towards the state.

The validity of our identification strategy hinges upon two crucial assumptions: (a) The model-derived predictions have to be correlated with actual prevalence, and (b), the predictions must not be correlated with the error term. The plausibility of these assumptions are discussed in Sections 5.2 and 6.

# 5 Data, Simulations, and Descriptive Analysis

## 5.1 Data

To analyse the effect of Ebola exposure on state legitimacy, we draw on pre-and post-epidemic individual-level survey data from the Afrobarometer. The pre-epidemic surveys (round 5) were conducted between June 2012 and April 2013, the post-epidemic surveys

(round 6) between March and June 2015.[9] We combine these repeated cross sections to analyse the effect of the epidemic on state legitimacy by looking at changes in trust in parliament, president, and police.[10] Trust in these entities are seen as close proxies for state legitimacy (Newton, 2007; Weatherford, 1992; Hutchison and Johnson, 2011). As an additional proxy for state legitimacy, we use willingness to pay taxes. All outcomes represent ordinal variables with values ranging from zero to three, and in the case of willingness to pay taxes from zero to four. To facilitate interpretation, we standardise outcome variables to a mean of zero and a standard deviation of one. In total, our individual–year dataset encompasses 2 periods and 6,201 individuals.

Our measure for Ebola exposure is based on subnationally stratified weekly numbers of new Ebola cases which are published in the WHO Ebola Situation Reports. For Guinea and Sierra Leone, data are available at the second subnational administrative level, referred to as prefectures and districts, respectively. EDV cases for Liberia are reported at the first subnational administrative level (counties). The number of territorial units in Guinea, Liberia and Sierra Leone are 34, 15 and 14 respectively.[11] For simplicity, we will collectively refer to these units as 'districts'. For these administrative regions, weekly information on new Ebola cases are available for the period between 30 December 2013 and 11 May 2016.[12] For each district and week, we compute the prevalence of Ebola, defined as the number of Ebola cases per 100,000 people. The (time-invariant) district-level population numbers used in the calculations stem from Gridded Population of the World, v4. Based on the weekly prevalence rates, we define our measure for Ebola exposure as the average Ebola prevalence between the start of the epidemic and the beginning of the Afrobarometer survey fieldwork in the district in which a given individual resides.[13,14] It is well documented that the Ebola case data, especially the district-stratified data, are prone to measurement and reporting error (e.g., WHO, 2014, Gignoux et al., 2015, or Scarpino et al., 2015). However, by instrumenting reported cases with simulated numbers, we mitigate issues related to mismeasurement.

[9] Even though the WHO declared the epidemic officially over only on 9 June 2016, 96% of all cases occurred before round 6 of the Afrobarometer surveys were conducted.

[10] Afrobarometer questions used in our analysis are listed in Table A.1.

[11] The average size of the administrative units is similar across all countries: 7,196km$^2$ (Guinea), 6,395km$^2$ (Liberia), and 5,186km$^2$ (Sierra Leone).

[12] EVD case data are available at `http://apps.who.int/gho/data/node.ebola-sitrep`

[13] Formally, the measure is defined as: $\frac{1}{\text{Total Weeks}} \sum \text{Ebola prevalence in district}_d$, where $d$ is the district in which individual $i$ resides.

[14] Fieldwork in Guinea started on 16 March 2015, in Liberia on 6 May 2015, and in Sierra Leone on 22 May 2015.

## 5.2 Simulations

We simulate the spatiotemporal spread of the Ebola epidemic using a modified version of the simulation model developed in Backer and Wallinga (2016).[15] Therein, the outbreak is represented as a network of local epidemics that are interconnected across districts through a gravity model. Inputs into the model are disease-specific parameters as well as gravity parameters. In contrast to Backer and Wallinga (2016), we do not feed the model with parameter values calibrated to the West African Ebola outbreak, but use estimates derived from other Ebola outbreaks (all values listed in Table 1). Therefore, variation in the disease generation process is restricted to aspects that are unrelated to the spatiotemporal spread of the West African epidemic. This property is crucial in the context of our analysis as local socio-economic conditions can influence path and intensity of the epidemic. State legitimacy, for example, reportedly influenced compliance with control measures. This, in turn, affected extent and intensity of the epidemic (Manguvo and Mafuvadze, 2015). The use of parameter values fitted to the West African epidemic as inputs in the simulations would therefore result in the simulations being endogenous with respect to our outcome variables.

Local intensity of Ebola transmission is crucially determined by the effective reproduction number, i.e. the expected number of individuals that are infected by a single infected person. Local disease intensity is further influenced by the time period—referred to as serial interval—between the onset of symptoms with the primary patient and the onset of symptoms with the secondary patient. Combining these two determinants, the number of Ebola cases generated in district $d$ in week $t$ can be written as:

$$\Lambda_d(t) = R_d(t) \sum_{\tau=1}^{T} \omega(\tau) I_d(t-\tau). \tag{3}$$

The number of locally generated new Ebola cases in week $t$, $\Lambda_d(t)$, depends on the (simulated) number of cases $I_d$ in the $\tau$ weeks preceding current week $t$. The function $\omega(\tau)$ represents the serial interval distribution and determines the likelihood of onset within $\tau$ weeks from infection. We assume that the time between infection and onset, i.e., the serial interval, takes a maximum of 6 weeks, i.e., $T = 6$ (cf. Backer and Wallinga (2016)). Values for the mean and standard deviation of $\omega(\cdot)$ are taken from Maganga et al. (2014) who

[15] Description and code available at `http://journals.plos.org/ploscompbiol/article?id=10.1371/journal.pcbi.1005210#sec008`.

base their estimates on the Ebola outbreak in Democratic Republic of Congo in 2014.

The reproduction number, $R(t)$, is a key parameter in disease progression models. For the purposes of our study, we set $R(t) = 2.347$. This corresponds to the average of $R(t)$ estimates obtained in the non-West African-specific studies reviewed in Van Kerkhove et al. (2015).[16,17] A reproduction number above unity implies that the entire population will eventually be infected with the Ebola virus. To contain contagion within our model, we allow $R(t)$ to fall below one, and therefore the epidemic to die out, if cumulative case numbers reach a threshold. Specifically, we set $R(t)$ to 0.5 once the threshold has been reached (Backer and Wallinga, 2016). The threshold is defined as a random share $\tilde{s}$ of the district's total population and is therefore unrelated to the timing and sequence of the implementation of actual control measures. The share is drawn from a uniform distribution with support $[s/2, 2s]$. Based on data from Maganga et al. (2014), we set $s = 0.0007$.[18] Formally, the threshold-dependent reproduction number is given by:

$$R_d(t) = \begin{cases} 2.347 & \text{if } t \leq t_d^b \\ 0.5, & t > t_d^b, \end{cases} \tag{4}$$

where $t_d^b = \min_t \{t : I_d(t) > I_d^b\}$ and $I_d^b$ is the randomly drawn threshold value.

The total number of cases in district $d$ and week $t$ is not only determined by local disease dynamics, as described in Eq.(3), but also by the number of Ebola cases imported from other districts. To model the migration process, we assume that a constant fraction $\phi$ of infected persons leaves a given district $d$ in each week. These cases then scatter across the remaining districts ($j \neq d$) according to the bilateral connectivity matrix $m$. Specifically, the probability with which an infected individual who leaves district $d$ migrates into district

[16] Van Kerkhove et al. (2015) conduct a systematic review of epidemiological parameters from Ebola outbreaks. For non-West African epidemics, the review shows that $R(t)$ estimates range between 1.34 and 4.71. The range of estimates for the West African epidemic is even wider; values lie between 1.26 and 9. However, Figure B.1 illustrates that the estimates for $R(t)$ are concentrated around a value of 2, for both the West African epidemic and other epidemics. All parameter estimates of the studies reviewed in Van Kerkhove et al. (2015) are listed in Table S2 in the Data Records Section of Van Kerkhove et al. (2015).

[17] Note that values of $R(t)$ during the West African epidemic are likely to have varied across different countries (e.g., WHO Ebola Response Team, 2014). However, incorporating such differences would potentially introduce endogeneity into our simulation model as differences in transmission intensities (partly) reflect differences in state legitimacy (see Section 3).

[18] For comparison, the overall prevalence rate for the West African epidemic was 0.0012.

$j$, $m_{d,j}$, is given by the gravity equation :

$$m_{d,j} = \frac{pop_j^{\gamma^{pop}} dist_{d,j}^{\gamma^{dist}}}{\sum_{j \neq d} pop_j^{\gamma^{pop}} dist_{d,j}^{\gamma^{dist}}}. \tag{5}$$

Connectivity increases with population of the district of destination ($pop_j$) and decreases with bilateral distance ($dist_{d,j}$). Both variables, population numbers and Euclidean distances, are time-invariant and predetermined. The gravity parameters $\gamma^{dist}$ and $\gamma^{pop}$ are taken from Wesolowski et al. (2015), who base their mobility estimates on Kenyan mobile phone data.[19] The value of $\phi$ is also based on these gravity estimates. It is defined as the average share of population that moves across districts in a week.[20]

Combining Eqs. (3) and (5), the number of Ebola cases in district $d$ and week $t$ can be written as the fraction of locally generated cases that does not leave the district plus the cases imported from other districts:

$$I_d(t) = (1 - \phi)\ \Lambda_d(t) + \sum_{j \neq d} \phi\, m_{j,d}\, \Lambda_j(t). \tag{6}$$

The model described in Eq.(6) represents a generic spatiotemporal model of Ebola transmission. The only aspect that makes it West-African specific is the population dispersion matrix $m$, the population numbers used in generating the (random) threshold for the change in $R_d(t)$ as well as the choice of starting point. However, with exception of the starting point, these are time-invariant features and therefore exogenous with respect to the spatiotemporal spread of the epidemic. Omitting the starting point in the regression analysis leaves the results unchanged.

To construct our instrument, we first run the simulation model in Eq.(6) one thousand times and take the mode of simulated cases for each district and week. Starting point for each simulation is Guéckédou, where patient zero was observed in December 2013. In a second step, we then determine simulated prevalence rates by dividing simulated case numbers by population. As a final step, we compute the instrument as average

[19] Using mobile phone data implies that the gravity parameters capture short-run mobility, i.e., the type of movement we focus on. Census-based estimates, on the other hand, reflect the effects of distance and population concentration on more permanent movements (Wesolowski et al., 2015).

[20] More specifically, we compute the share of total population that travels to other districts in any given week based on the gravity parameters of Wesolowski et al. (2015). We then define $\phi$ as the average of these 'mobility shares' across all districts.

Table 1: Simulation Inputs

| Input | Description | Value | Source/Basis |
|---|---|---|---|
| | | *Time-invariant district-level characteristics* | |
| $pop_j$ | population of districts | true value | Gridded Population of the World, v4 |
| $dist_{d,j}$ | bilateral Euclidean distance | true value | Computed using spatial analysis software. |
| | | *Parameters local disease progression* $\Lambda$ | |
| $R_H(t)$ | reproduction number before threshold | 2.347 | Van Kerkhove et al. (2015) |
| $R_L(t)$ | reproduction number after threshold | 0.5 | Backer and Wallinga (2016) |
| $I_d^b$ | threshold $R$, defined as $I_d^b = \tilde{s} \times pop_d$ | $\tilde{s} \sim U\,(s/2, 2s)$, with $s = 0.0007$ | Maganga et al. (2014) |
| $\omega\,(\cdot)$ | serial interval distribution | mean=16.1, sd=4.4, T=6 | Maganga et al. (2014) |
| | | *Parameters inter-district connectivity* $m$ | |
| $\phi$ | migration fraction | 0.043 | Wesolowski et al. (2015) |
| $\gamma^{dist}$ | gravity parameter distance | –2.05 | Wesolowski et al. (2015) |
| $\gamma^{pop}$ | gravity parameter population | 1.22 | Wesolowski et al. (2015) |

predicted Ebola prevalence in an individual's district of residence between the start of the epidemic and the beginning of the survey fieldwork. In the remainder of this paper, we use this simulation-based instrument to quantify the effect of the Ebola epidemic on state legitimacy.

## 5.3 Descriptive Analysis

Table 2 reports summary statistics for the key variables. On average, districts reported 303 Ebola cases during the epidemic. This corresponds to a mean prevalence of 0.856 cases per 100,000 people over the two survey years (of which the first one was Ebola free). Figure B.2 depicts the density of observed average prevalence along with the density function of simulated average prevalence. While both densities exhibit a similar shape, with prevalence concentrated around the same value, the (right) tail of observed prevalence is longer. This difference could arise, for example, because our simulations explicitly abstract from socioeconomic aspects that could have intensified the spread of the disease.

Figure 2 below visualises the regional variation in average Ebola exposure exploited in our analysis. Panel (a) depicts the average observed prevalence rates (classified into quintiles separately for each country); panel (b) represents the spatial variation in simulation-derived predicted Ebola prevalence. Starting with the next section, we formally assess how these differences in exposure influenced state legitimacy.

A general worry, discussed in more detail in Section 6, is that variation in regional Ebola exposure are correlated with other, Ebola unrelated, district-specific differences. In Table

Table 2: Descriptive Statistics Key Variables

| **Variable** | **Mean** | **Std. Dev.** | **Min.** | **Max.** | **Obs.** |
|---|---|---|---|---|---|
| Ebola prevalence | 0.856 | 1.470 | 0 | 5.939 | 6201 |
| Simulated prevalence | 0.157 | 0.248 | 0 | 2.280 | 6201 |
| Trust in parliament (SD) | 0 | 1 | -1.350 | 1.403 | 6201 |
| Trust in president (SD) | 0 | 1 | -1.494 | 1.079 | 6201 |
| Trust in police (SD) | 0 | 1 | -1.045 | 1.642 | 6201 |
| Willingness to pay taxes (SD) | 0 | 1 | -0.507 | 3.689 | 6201 |

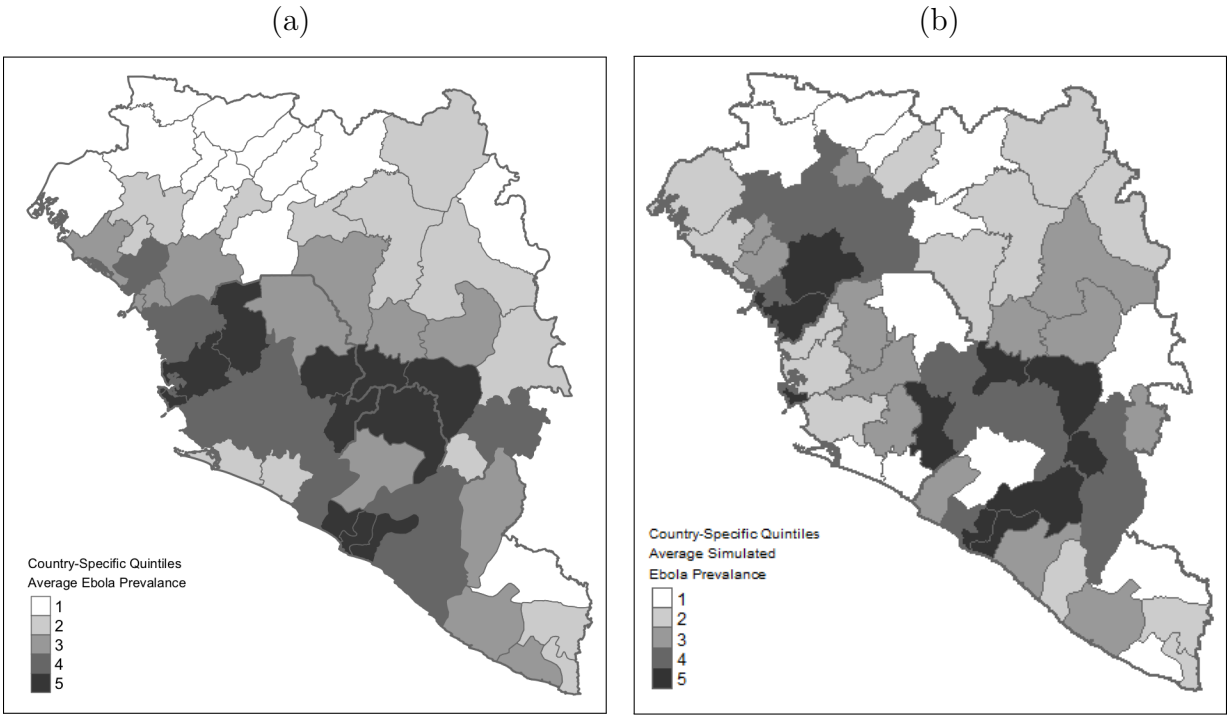


Figure 2: Panel (a) depicts the country-sepcific quintiles of average Ebola prevalence per 100,000 people. Panel (b) depicts the country-sepcific quintiles of average simulated Ebola prevalence per 100,000 people.

3 we therefore investigate the extent to which simulation-derived predicted prevalence is correlated with various time-invariant and time-varying district-level characteristics, once country-specific dummies are accounted for. We start with the two key drivers in our spatial diffusion process. These are population numbers as well as network connectivity to Guéckédou (i.e., the starting point of the epidemic).[21] Unsurprisingly, these two aspects are strongly correlated with predicted Ebola prevalence. The gravity-type nature of our diffusion model implies that both, a larger population and greater connectivity to Guéckédou

[21]For a given district, the network connectivity to Guéckédou is computed as the direct (bilateral) connectivity to Guéckédou (given by Eq.(5)) plus the indirect connectivity via all other districts.

results in more (simulated) Ebola cases being imported from other districts. Raw distances, on the other hand, are not statistically significantly correlated with predicted Ebola exposure. The same is true for the pre-epidemic provision of public goods, which is proxied roughly by the district-level averaged probability of being located in walking distance to a school, health clinic, and police station, respectively.[22]

To investigate if predicted prevalence is correlated with the level or change in income, we use, in the absence of better data, a number of crude proxies. The value of crop production per capita (IIASA/FAO, 2012) and the presence of minerals (such as diamonds or gold, Tollefsen et al. (2012)) are used as proxy for difference in the regional level of income. For neither of these variables do we find a statistically significant correlation with our simulations. This is also true when we look at the change in average rainfall, temperature and drought exposure between the pre- and post epidemic survey years.[23] These variables are commonly used as a proxy for income shocks in economies dominated by the agricultural sector (e.g., Burke et al., 2015; Marshall et al., n.d.; Harari and La Ferrara, Forthcoming).[24] Overall, Table 3 shows that the degree of correlation between simulated Ebola prevalence and district-level characteristics is limited. The only two aspects that are statistically significantly correlated with simulated prevalence are population numbers and network distance to Guéckédou. In the next section, we discuss in detail to what extent this result poses a threat to our identification strategy.

# 6 Results

In this section, we first establish that state legitimacy disproportionately increased among individuals who resided in areas with high Ebola transmission intensities. We then provide support for the validity and stability of this finding. In the second part, we discuss potential mechanisms underlying our baseline results.

[22]The proxies for pre-epidemic public good provision are computed as the district-level averages of the responses to the following Afrobarometer (wave 5) questions: 'Are the following facilities present in the primary sampling unit/enumeration area, or within easy walking distance: School?' 'Are the following facilities present in the primary sampling unit/enumeration area, or within easy walking distance: Police station?' 'Are the following facilities present in the primary sampling unit/enumeration area, or within easy walking distance: Health clinic?' For each of the questions, the answer can be either yes or no.

[23]Data on temperature and rainfall are drawn from the CRU TS v4.01 dataset provided by the Climate Research Unit, University of East Anglia. The drought index is based on data taken from the SPEI Global Drought Monitor.

[24]More than 70% of the total labour force is employed in the agricultural sector in all three countries.

Table 3: Correlations Simulated Prevalence and District-Level Characteristics

| | Coefficient | Standard Error | Obs. |
|---|---|---|---|
| *Model Inputs* | | | |
| Population (SD) | 0.076*** | 0.026 | 61 |
| Network Distance to Guéckédou (SD) | 0.070** | 0.027 | 61 |
| *Spatial Characteristics* | | | |
| Distance to Guéckédou (SD) | -0.097 | 0.062 | 61 |
| Distance to capital (SD) | -0.019 | 0.034 | 61 |
| Distance to national border (SD) | -0.018 | 0.034 | 61 |
| Longitude (SD) | 0.011 | 0.042 | 61 |
| Latitude (SD) | -0.161 | 0.097 | 61 |
| *Public Goods 2013* | | | |
| School nearby (SD) | 0.000 | 0.022 | 61 |
| Health clinic nearby (SD) | -0.021 | 0.029 | 61 |
| Police station nearby (SD) | 0.061 | 0.043 | 61 |
| *Economic Characteristics* | | | |
| Value crop production per capita (SD) | -0.048 | 0.029 | 61 |
| Presence of minerals (SD) | 0.084 | 0.051 | 61 |
| $\Delta$ rainfall (SD) | 0.135 | 0.105 | 61 |
| $\Delta$ temperature (SD) | -0.015 | 0.028 | 61 |
| $\Delta$ drought index (SD) | -0.009 | 0.025 | 61 |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Table depicts the results of district-level regressions of predicted prevalence on district-level characteristics and country-fixed effects. White-Huber standard errors are reported.

## 6.1 Ebola Exposure and State Legitimacy

We start our analysis by employing our 2SLS approach to examine whether greater exposure to the Ebola epidemic differentially increased legitimacy of the state, as proxied by trust in various government agencies.[25] The result presented in column (1) of Table 4 documents that trust in parliament increased with EVD transmission intensity. One additional Ebola case per 100,000 people raises trust by 0.165 standard deviations. This implies that a one-standard deviation increase in Ebola exposure raises trust by economically meaningful 0.24 standard deviations. The point estimate is significant at the 99 percent confidence level and the first-stage Kleibergen-Paap F-statistic for the excluded instrument of 14.95 indicates that the probability of a bias due to weak instruments is low. Trust in president (column (2)) and police (column (3))—an agency involved in the im-

[25]The corresponding reduced-form estimates are reported in Table C.1

plementation and maintenance of control measures—also increase. Coefficient sizes as well as levels of statistical significance are similar compared to the results presented in column (1). The Ebola-induced effects on legitimacy are also reflected in a higher willingness to pay taxes, or as phrased in the Afrobarometer survey, a lower inclination to refuse to pay taxes. The point estimate reported in column (4) implies that an additional Ebola case per 100,000 people reduces the propensity to refuse to pay taxes by 0.19 standard deviations.

Table 4: Ebola Exposure and State Legitimacy

| | Trust in Parliament | Trust in President | Trust in Police | Refusal to Pay Taxes |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Ebola prevalence | 0.165*** | 0.172*** | 0.136*** | -0.191** |
| | (0.060) | (0.051) | (0.050) | (0.085) |
| | First stage regression: Ebola prevalence | | | |
| Simulated Ebola prevalence | 2.851*** | 2.851*** | 2.851*** | 2.851*** |
| | (0.737) | (0.737) | (0.737) | (0.737) |
| Country×year FE | yes | yes | yes | yes |
| District FE | yes | yes | yes | yes |
| Individual-level controls×year FE | yes | yes | yes | yes |
| Obs. | 6,201 | 6,201 | 6,201 | 6,201 |
| F-test excl. IV | 14.95 | 14.95 | 14.95 | 14.95 |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are clustered at the district level. The F-test represents the first-stage Kleibergen-Paap F-statistic for the excluded instrument.

Taken together, the results of Table 4 show that state legitimacy increased with exposure to the Ebola epidemic. They do not, however, provide any insights into the underlying channels. As outlined in the introduction, potential mechanisms are differences in government response as well as perception thereof. Before investigating the plausibility of these channels, we first discuss concerns related to our identification strategy.

*Threats to Identification and Robustness*

The identifying assumption underlying our analysis is that simulation-derived predicted prevalence influences state legitimacy only via its effect on realised Ebola prevalence. Since (time-invariant) population numbers and network connectivity are inputs in our disease generation process, one immediate concern is that these factors impact state legitimacy directly, i.e., not only indirectly via their influence on the Ebola diffusion process. If this is the case, our estimates could pick up these Ebola-unrelated effects. However, due to the inclusion of district fixed effects in all our regressions, this would pose a threat to our identification strategy only if these input factors influenced state legitimacy via an

Ebola-unrelated channel and the magnitude of this effect changed between the pre-and post epidemic period. Similarly, for time-invariant (pre-epidemic) characteristics, such as the availability of public goods or economic structure of a district,to bias our results, they would have to be correlated with simulated Ebola exposure and additionally exhibit time-varying effects.[26] While the limited extend of correlation between simulated Ebola prevalence (Table 3) indicates that the existence of such effects are unlikely, we formally address these concerns. To this end, we augment our regression setup to include all variables listed in Table 3, where time-invariant characteristics are interacted with time period fixed effects to generate temporal variation. Table E.1 documents that coefficient estimates remain stable, albeit somewhat less precisely estimated.

Another worry related to the violation of the exclusion restriction is that simulated prevalence influences state legitimacy through channels other than actual observed Ebola prevalence. This could be the case, for example, if health authorities used simulation-based forecasts to inform their decision making. Simulations could then influence state legitimacy not through actual, but prevented Ebola exposure. However, the existence of such exposure-unrelated simulation-induced effects are unlikely due to the lack of realtime data. Refined subnationally disaggregated epidemiological data, a prerequisite for forecasting the diffusion processes within countries, only became publicly available after November 2014, i.e., after the major surge in case numbers had already subsided (Chowell et al., 2017). Furthermore, limited coordination and sharing among (primarily academic) groups that worked on developing forecasting models as well as lags in dissemination meant that spatiotemporal simulation models stratified at the subnational level became only available late in the epidemic and were of limited use to policy makers (Chretien et al., 2015; Chowell et al., 2017).[27,28] We provide more formal evidence that simulation-based interventions do not bias our results by showing that we obtain very similar estimates if we consider only Ebola cases (observed as well as predicted) that occurred before November 2014 in the construction of our exposure measures (Table E.2).[29] Additionally, we implement the pro-

[26] A conceivable example for such a time-varying effect is that the valuation of existing public goods changes between the pre- and post epidemic period.

[27] Additional (suggestive) evidence for simulations not playing a role in the coordination of relief efforts within countries is the fact that the predictive power of our instrument is strong. If resources had been (effectively) allocated based on simulations, one would expect simulated prevalence to convey only limited information regarding the actual path and intensity of the epidemic.

[28] The first subnational spatiotemporal EVD simulation models were published in early 2015 (Chretien et al., 2015).

[29] This results is not surprising as the correlation between overall prevalence rate (computed as the average prevalence rate between the start of the epidemic and the start of the Afrobaromter surveys) and

cedure developed in Conley et al. (2012) to gauge how sensitive our estimates are to small violations of the exclusion restriction. The results in Table E.3 suggest that the effects of the epidemic persist even when we allow the direct effect of the instrument, i.e., the violation of the exclusion restriction, to be up to 50% of the magintude of the (instrumented) effect of prevalence.

A further potential concern is that our simulation model does not specifically capture the spatiotemporal diffusion process of the EVD epidemic, but other, Ebola-unrelated spatial diffusion processes, such as the dispersion of information. To mitigate this concern, we conduct two falsification tests. First, we simulate the epidemic one thousand times, each time initiating the simulation at a randomly drawn starting point rather than the true one. As documented in columns (1)–(4) of Table 5, the resulting simulations have no predictive power regarding actual transmission intensity. The first-stage F-statistic is 1.06. This strongly suggests that our results, more specifically the predictive power of the simulations, are unlikely to have arisen due to chance. It also implies that if our results were, in fact, driven by an Ebola-unrelated process, this process would have to (a) progress across space and time in the same manner as our Ebola simulation model and (b) start at the same location. To document that the existence of such a process is highly unlikely, we conduct a second falsification test. Therein, we initiate the simulations at the true starting point (i.e., Guéckédou) and analyse how the epidemic is predicted to spread across the three neighbouring countries Ivory Coast, Mali and Senegal. While in direct proximity to the epicentre of the West African Ebola outbreak, these countries did not see widespread transmission. In total, only nine cases were reported for the three nations (WHO, 2016a).[30] If our model specifically captures the Ebola diffusion process, simulated prevalence should not exert an effect on state legitimacy due to the lack of transmission. To test whether this is the case, we compile pre-and post-epidemic individual-level survey data for the three neighbouring countries in analogy to the procedure described in Section 5.[31] We then investigate if simulated prevalence influences state legitimacy using regression setup Eq.(1). The resulting reduced-form estimates, shown in Table 5, columns (5)–(8), reassuringly produce null results throughout. Coefficients are statistically non-significant

pre-November prevalence rate (computed as the average between the start of the epidemic and November 2014) is above 0.85, for both observed and simulated prevalence. We also obtain similar results if we only include observations/simulations prior mid September 2014, i.e., prior to the large influx of international resources.

[30]For Ivory Coast, no cases were reported. Mali reported eight cases while one case occurred in Senegal (WHO, 2016a).

[31]Simulations and data are described in Appendix D.

and close to zero.

Table 5: Falsification Exercises

| | Random Starting Point | | | | Reduced-Form Regressions Neighbouring Countries | | | |
|---|---|---|---|---|---|---|---|---|
| | Trust in Parliament | Trust in President | Trust Police | Refusal to Pay Taxes | Trust in Parliament | Trust in President | Trust in Police | Refusal to Pay Taxes |
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
| Ebola prevalence | -0.189 | -0.310 | 0.056 | -0.162 | | | | |
| | (0.241) | (0.418) | (0.215) | (0.498) | | | | |
| | First stage regression: Ebola prevalence | | | | | | | |
| Simulated Ebola prevalence | 0.191 | 0.191 | 0.191 | 0.191 | -0.056 | -0.063 | -0.012 | 0.083 |
| | (0.186) | (0.186) | (0.186) | (0.186) | (0.083) | (0.126) | (0.099) | (0.068) |
| Country×year FE | yes | yes | yes | yes | yes | yes | yes | yes |
| District FE | yes | yes | yes | yes | yes | yes | yes | yes |
| Indiv. controls×year FE | yes | yes | yes | yes | yes | yes | yes | yes |
| Obs. | 6,201 | 6,201 | 6,201 | 6,201 | 6,589 | 6,589 | 6,589 | 6,589 |
| F-test excl. IV | 1.060 | 1.060 | 1.060 | 1.060 | | | | |
| Region | Ebola | Ebola | Ebola | Ebola | Neighbour | Neighbour | Neighbour | Neighbour |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are clustered at the district level. The F-test represents the first-stage Kleibergen-Paap F-statistic for the excluded instrument.

In Appendix E we conduct a number of robustness checks to further document the stability of our estimates. To demonstrate that our results are not driven by outliers, we re-define the Ebola exposure measure (as well as the instrument) as the logarithmised prevalence (plus one) and rerun the regressions of Table 4. The results remain qualitatively unaltered (Table E.4). Similarly, point estimates remain stable when dropping one region at a time (Figures E.1–E.4).[32] To address concerns related to the possibility that unaccounted spatial correlation in the error terms influences statistical inference, we implement two alternative standard error computing procedures, both based on the approach developed in Conley (1999). In the first case, we take the common approach and allow for spatial autocorrelation across districts that declines linearly in geographic distance up to a cut-off of 150 kilometers.[33] In the second case, we adapt the Conley (1999) approach to the spatial structure of the network used in our simulations. Specifically, we use the bilateral connectivity between two districts—defined in Eq.(5)—as weighting kernel (cf. Acemoglu et al., 2015). With both approaches, we obtain very similar standard errors compared to the ones presented in the main part (Tables E.6–E.7). While these results suggest that inference is not biased due to unaccounted spatial correlation in the error terms, they are uninformative about the extent of spatial spillovers in Ebola prevalence itself.

[32]This also implies that our results are robust to omitting Guéckédou prefecture, i.e., the (endogenous) starting point of our simulations.

[33]This corresponds to the average distance to the second-neighbour district. The results are stable to varying the cut-off.

High Ebola transmission intensity in nearby districts could influence people's perception of the government either directly—through spatial spillovers of Ebola cases—or through Ebola-induced changes of state legitimacy in neighbouring regions, i.e., through spatial spillovers of state legitimacy. To investigate this possibility, we compute the (distance weighted) average simulated Ebola prevalence of the first and second neighbours for each district.[34] We then include this spatial lag as an additional regressor, in the first-stage, reduced-form and the IV setup. The point estimate of the spatial lag in the first-stage regression is, albeit statistically insignificant, positive and economically non-negligible (Table E.8, column (1)). This is unsurprising as both, the simulated and the actual epidemic diffuse across space, implying that the (simulated) transmission intensity in the nearby district have (some) predictive power regarding observed prevalence in the own district. However, this result does not suggests the presence of spatial spillovers of Ebola-prevalence-related effects. Conditional on simulated Ebola prevalence of the 'home district', the spatial lag does not influence state legitimacy.[35] The coefficient of the spatial lag is non-significant and close to zero in both the reduced-form and the IV regressions (Table E.8 columns (2)–(4) and Table E.9), while the size of the direct (i.e., district specific) estimates are unaffected. Together the results suggests that spatial spillovers do not play a prominent role in explaining the findings of Table 4.

In a final robustness check, we show that our results are not specific to the choice of parameter values. Our 2SLS estimates remain stable if we allow $R(t)$ to fluctuate within the range of 1.56 to 2.51 (Tables E.10–E.11).[36]

## 6.2 Potential Mechanisms

As outlined in the introduction, we argue that the positive effects of Ebola exposure on state legitimacy are due to changes in people's perception induced by the government's response to the epidemic, rather than the direct result of the disease itself. Potential mechanisms underlying such response-induced changes in perception are differential valuation or effectiveness of control measures. Below, we investigate the plausibility of these

[34] This is in keeping with the approach used in computing the Conley (1999) standard errors that correct for spatial correlation. Changing the cut-off produces very similar results.

[35] The spatial lag even remains statistically non-significant if we do not control for the exposure of the own district.

[36] These values represents the top and bottom quintile of all $R(t)$ estimates reviewed in Van Kerkhove et al. (2015).

channels.[37] It is important to note that the subsequent analysis should be interpreted as suggestive evidence. Lack of detailed information on location, type and effectiveness of control measures as well as people's perceptions thereof prevent us from cleanly disentangling (the relative importance of) individual channels.

*Valuation*

A potentially important mechanism linking Ebola exposure to state legitimacy is differential valuation of control measures. As illustrated in Figure 3, we argue that perceived risk of contracting Ebola was higher in regions with more intense Ebola transmission, leading to greater valuation of control measures. To empirically investigate the plausibility of this

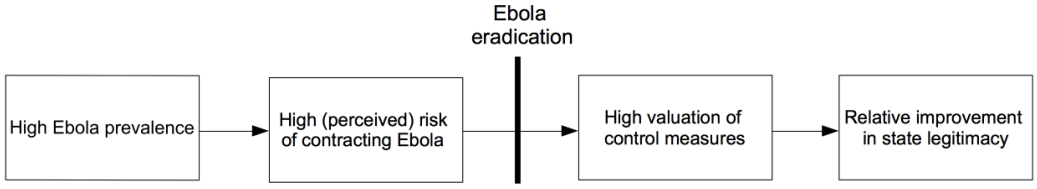


Figure 3: Potential Mechanism: Valuation of Control Measures.

mechanism, we conducted a (cross-sectional) retrospective SMS-message-based survey in Liberia using the GeoPoll mobile surveying platform. For Liberia, but not for Guinea and Sierra Leone, this platform allows for subnationally representative sampling in terms of age and sex. In our surveys, we asked individuals about their views on risk of contracting Ebola as well as valuation and necessity of control measures. Furthermore, we elicited information on age, sex, education as well as the district (county) the respondent was living in during the epidemic.[38] In total, 512 individuals submitted their answers. After dropping incomplete responses, the sample is reduced to 403 observations.[39] For the empirical analysis, we match each GeoPoll survey respondent to the average Ebola prevalence of her/his county of residence during the epidemic. The summary statistics of

[37]Due to the lack of detailed data on implementing agency, success of control measures and people's perception of these policies, we cannot gauge the extent to which the subsequent results are the reflection of citizens (falsely) attributing the success of Ebola eradication to the government's actions (rather than international organizations). However, accounts indicate that, while resources and know-how were to a large part provided by international organisations, governments were heavily involved in the coordination and implementation of the Ebola control measures (DuBois et al., 2015; Marston et al., 2017).

[38]Questions are listed at the bottom of Table 6. Survey was conducted in October 2017.

[39]Observations are primarily dropped because we could not infer the individual's place of residence at the time of the epidemic. The results do not change if we use the full sample and assume that the county of residence did not change for individuals we have no information on district of residence during the epidemic.

the resulting dataset are presented in Table F.1.[40] In the following cross-sectional regressions, we control for age, sex, education, and urban residency fixed effects.[41] The results are presented in Table 6. The first column shows that simulation-based Ebola prevalence

Table 6: Ebola Exposure and Valuation: Retrospective Mobile Phone Survey (Liberia)

| | Perceived Ebola Risk (SD)[a] | Valuation Control Measures (SD)[b] | Success Control Measures (SD) [b] | Willingness to Finance Public Goods (SD)[d] |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Simulated Ebola prevalence | 1.234*** (0.466) | | | |
| Perceived Ebola risk | | 0.332*** (0.104) | 0.287* (0.171) | 0.752*** (0.276) |
| | | First stage regression: Perceived Ebola Risk (SD) | | |
| Simulated Ebola prevalence | | 2.234** (0.513) | 2.234** (0.513) | 2.234** (0.513) |
| Individual-level controls | yes | yes | yes | yes |
| Obs. | 403 | 403 | 403 | 403 |
| F-test excl. IV | | 5.787 | 5.787 | 5.787 |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Individual-level controls include age fixed effects, education fixed effects, urban status and gender.

[a] 'Perceived Ebola Measures (SD)' is the answer to the question: How high would you personally say was the risk of contracting Ebola in your region during the epidemic? Very low, low, high, very high.

[b] 'Valuation Control Measures (SD)' is the answer to the question: How high is your valuation of the Ebola containment measures implemented by the central government? Very low, low, high, very high.

[c] 'Success Control Measures (SD)' is the answer to the question: Have the Ebola control measures implemented by the government reduced your risk of contracting Ebola? Strongly disagree, disagree, agree, strongly agree.

[d] 'Willingness to Finance Public Goods (SD)' is the answer to the question: Would you be willing to pay more taxes to finance public goods (such as Ebola containment measures) because of your experience during the epidemic? Yes, no.

statistically significantly increases the perceived risk of contracting Ebola, an ordinal variable ranging from very low (1) to very high (4). This finding provides additional support for the Ebola-specificity of our instrument. The result in column (2) illustrates that the perceived Ebola risk—instrumented with simulated prevalence—increases the valuation of Ebola containment measures.[42] Similarly, Ebola control measures provided by the central government are judged to have been more important in reducing transmission in regions where perceived Ebola risk was greater (column (3)). Finally, column (4) documents that, as a result of the personal experience during the epidemic, the willingness to pay taxes ear-

[40] Compared to the Afrobarometer data, the age and sex distribution are very similar (see Table F.1). However, the GeoPoll data over-samples urban residents and highly educated individuals.

[41] The second-stage regression takes the following form: $y_{i,d} = \alpha + \beta \widehat{E}_{i,d} + \Phi \, \mathbf{X}_{i,d} + \varepsilon_{i,d}$, where $y_{i,d}$ is the outcome variable of individual $i$ living in district $d$, $\widehat{E}_{i,d}$ is the (predicted) prevalence in district $d$, and vector $\mathbf{X}_{i,d}$ includes the individual-level controls.

[42] We get similar results if we replace perceived Ebola risk with Ebola prevalence.

marked for financing public goods is higher for individuals that saw the risk of contracting Ebola as being high. Overall, the results of Table 6 provide support for differential valuation of control measures being a potentially important mechanism underlying the observed relative increase in state legitimacy in regions where the Ebola transmission intensity was high.

*Heterogeneities*

In addition to raising the valuation for a given level of public good provision, state legitimacy could also have increased due to more public resources being directed towards districts with higher Ebola exposure. Studies based on field work from India, for example, document that the local provision of public goods and services by the government increases state visibility and legitimacy (Corbridge et al., 2005). To investigate whether variation in the quantity of Ebola relief efforts underlies our findings, we first establish that more resources were allocated to districts that experienced intense transmission. As a proxy for resources, we use the number of Ebola treatment units, Ebola test laboratories and Ebola community care centres in a given district.[43] Table 7 shows that all three measures of response intensity increase with EVD exposure.

In a second step, we investigate if this exposure-induced increased provision of resources can help explain our main results. To this end, we divide the districts into two groups, according to whether the amount of resources received lay above or below the country-specific median.[44] We then investigate the existence of heterogeneities by estimating separate slope coefficients for these two groups using our main individual-level Afrobarometer dataset.[45,46] As depicted in columns (1) and (4) of Table 8, the resulting estimates exhibit the expected pattern. The estimates of Ebola exposure on trust in parliament and president are larger

[43]Ebola treatment units (ETUs) were usually large facilities equipped to isolate patients and provide clinical care. Community care centres, were established to bring 'disease prevention and control capabilities to the community-level to complement larger and more centralized ETUs.' (UNICEF, 2016).

[44]The classification is based on the total number of Ebola facilities, i.e., the sum of Ebola control centers, Ebola test laboratories and Ebola community care centres.

[45]We have opted to estimate separate slope coefficients when analysing heterogeneities rather than introducing a simple interaction term. Econometrically, the two approaches are equivalent. However, differences are more clearly (directly) illustrated when estimating separate slope coefficients. Formally, the second-stage regression is given by: $y_{i,d,c,t} = \beta_H \, I_d \times \widehat{E}_{i,d,c,t} + \beta_L \, (1 - I_d) \times \widehat{E}_{i,d,c,t} + \Phi \, \mathbf{X}_{i,d,c,t} + \mu_{d,c} + \gamma_{c,t} + \varepsilon_{i,d,c,t}$, where $I_d$ represents an indicator variable that takes the value one if the district belongs to the group that received above-median resources and zero otherwise. The set of controls is analogous the one included in previous regressions. We flexibly allow these controls to exert a different effect in the two years as well as across the two groups by interacting them with year-group dummies.

[46]The results of the first-stage regressions are show in Tables G.1–G.2.

for the group of districts that received relatively more resources.[47]

Table 7: Ebola Exposure and Ebola Interventions: Cross-Section Analysis

| | Ebola Treatment Units (SD)[a] | Ebola Laboratories (SD)[a] | Ebola Community Care Centres (SD)[a] |
|---|---|---|---|
| | (1) | (2) | (3) |
| Ebola Prevalence | 0.381*** | 0.656*** | 0.533*** |
| | (0.125) | (0.083) | (0.131) |
| | First stage regression: Ebola prevalence | | |
| Simulated Ebola prevalence | 1.670** | 1.670** | 1.670** |
| | (0.672) | (0.672) | (0.672) |
| Country FE | yes | yes | yes |
| Obs. | 61 | 61 | 61 |
| F-test excl. IV | 6.170 | 6.170 | 6.170 |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

[a] 'Ebola Treatment Units (SD)' represents the number of Ebola treatment units (z-score). 'Ebola Laboratories (SD)' represents the number of Ebola laboratories (z-score). 'Ebola Community Care Centres (SD)' represents the number of Ebola community care centres (z-score). Regressions run at the district level; White-Huber standard errors are reported. We control for country fixed effects. Data sources: `https://data.humdata.org/ebola` and `https://data.hdx.rwlabs.org/dataset/ebola-treatment-centers`.

In addition to resource-allocation-related heterogeneities, a natural expectation is that the effect of the epidemic on trust in government depends on the effectiveness of relief efforts (cf., Gasper and Reeves, 2011; Cole et al., 2012). Absent data on effectiveness of control measures, we use the difference between predicted and observed Ebola prevalence as a measure of implied effectiveness. We then again classify districts into two groups, depending on whether implied effectiveness is above or below the country-specific median. The estimates for the separate slope coefficients are reported in columns (2) and (5) of Table 8. Aligning with expectations, we find that the effect of Ebola exposure is greater in regions where the epidemic was (relatively) less virulent than predicted by our simulation model.

So far, Table 8 provides suggestive evidence that differences in quantity and quality of relief efforts are potentially important in explaining why greater Ebola exposure increased state legitimacy. In a final step, we move from analysing heterogeneities in government response to investigating heterogeneities along the political dimension. Specifically, we test whether the effect of the epidemic varies with representation in government. In areas where people are excluded from the political decision making process (i.e., in politically alienated regions), the ability to eradicate a highly infectious disease may have provided a relatively stronger signal of government quality. Furthermore, the epidemic required

[47] For brevity, we only report results from regressions in which trust in parliament and president are used as dependent variables.

the central government to involve and coordinate with local leaders and communities in their eradication efforts (see Section 3). This increase in cooperation plausibly increased

Table 8: Heterogeneities

| | Trust in Parliament | | | Trust in President | | |
|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| High × | 0.215*** | 0.524*** | 0.102 | 0.172** | 0.505** | 0.088 |
| Ebola prevalence | (0.080) | (0.141) | (0.066) | (0.069) | (0.211) | (0.055) |
| Low × | 0.048 | 0.095 | 0.375** | 0.083 | 0.065 | 0.489** |
| Ebola prevalence | (0.071) | (0.135) | (0.164) | (0.071) | (0.095) | (0.243) |
| Country×year FE | yes | yes | yes | yes | yes | yes |
| District FE | yes | yes | yes | yes | yes | yes |
| Individual controls×year FE | yes | yes | yes | yes | yes | yes |
| Obs. | 6,201 | 6,201 | 6,201 | 6,201 | 6,201 | 6,201 |
| F-test excl. IVs | 5.76,19.55 | 39.57,3.14 | 34.34,20.22 | 5.76,19.55 | 39.57,3.14 | 34.34,20.22 |
| P-value equality (one-sided) | 0.060 | 0.011 | 0.080 | 0.184 | 0.024 | 0.053 |
| Heterogeneity | # Ebola facilities | Difference predicted observed prevalence | Representation in government | # Ebola facilities | Difference predicted observed prevalence | Representation in government |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are clustered at the district level. The F-test represents the first-stage Kleibergen-Paap F-statistic for the excluded instruments. P-value equality represents the p-value for the one-sided test of equality of coefficients between High and Low group. In columns (1), (2), (4) and (5), we test the hypothesis High¿Low; in columns (3) and (6), we test the hypothesis Low¿High. First-stage regressions shown in Tables G.1 and G.2.

trust in central government, especially in politically alienated regions (Acaps, 2015; Carter et al., 2017). To test for differential effects along the political dimension, we classify districts according to their political representation in government. Based on data from the Ethnic Power Relations (EPR) Dataset (Wimmer et al., 2009), we define districts as having no political influence if the majority of the population is of an ethnicity that is not represented in government as junior or senior partner. Separately estimating effects for these two groups unveils that the Ebola-related effects on trust are particularly strong in regions which are not represented in government (Table 8, columns (3) and (6)). This indicates that the provision of public goods is especially effective in fostering trust when individuals are politically excluded.

*From Perception to Accountability*

As a final step in our study, we turn to analysing Ebola-related effect on presidential election outcomes. Compared to our previous analysis, where we assessed the effects on trust in government entities, there are two important differences. First, rather than looking at changes in legitimacy, we are now focussing on changes in behaviour. Second, the subject of our analysis is no longer the state (as proxied by parliament, president and police), but the political party of the incumbent president. While maybe subtle, these differences are important. The Ebola-related relative improvement in state legitimacy documented above

will only be reflected in increased vote shares if people (partly) attribute the successful eradication to the actions of the president and her/his political party. While not necessary, the fact that the presidents were directly involved in the crisis management in all three countries (see Section 3) suggests that this may have been the case.

To empirically asses the effects of the epidemic on voting outcomes, we collect district-level information on the share of total votes received by the presidential candidate of the incumbent party in the first round of the presidential elections before and after the Ebola epidemic, respectively.[48,49] Using this district-level panel data, we then investigate whether a greater exposure to the epidemic lead to a differential change in the support for the candidate of the incumbent party. The regression approach employed is analogous to the one introduced in Section 4, the sole difference being that we operate at the district rather than the individual level.[50]

A caveat pertains to the interpretation of the subsequent results. Because we do not possess information on voter turnout or characteristics of voters, we cannot separately analyse the effects of the epidemic on mobilisation and persuasion of voters. The following results therefore capture the overall effects which include both the change in voter turnout as well as the shifts in voting behaviour among people who voted in pre-and post epidemic elections.

The result depicted in column (1) of Table 9 suggests that one additional Ebola case per 100,000 people increases the vote share of the candidate of the incumbent party by 0.207 standard deviations. This corresponds to an increase of 5.8 percentage points, indicating that the effect is economically non-negligible.[51,52] To support the interpretation that this result is induced by differences in the response to the epidemic (or perception thereof) rather than variation in exposure to the disease itself, we investigate the existence of

[48]The first-round elections took place in June 2010 and October 2015 (Guinea), October 2011 and October 2017 (Liberia), and November 2012 and March 2018 (Sierra Leone).

[49]We use the vote share for the presidential candidate of the incumbent party (rather than the incumbent president her-/himself) as the dependent variable because the incumbent presidents were constitutionally ineligible to run for another term in Liberia and Sierra Leone. Our focus on presidential rather than parliamentary elections is motivated by the fact that the election outcomes represent a measure for the support in ruling persons (and parties) that is uniform across time and space. This is not the case, for example, for parliamentary or local elections, where the party in power can vary across districts.

[50]Specifically, the second-stage regression takes the following form: $v_{d,c,t} = \beta \widehat{E}_{d,c,t} + \mu_{d,c} + \gamma_{c,t} + \varepsilon_{d,c,t}$, where $v_{d,c,t}$ is the vote share for the presidential candidate of the incumbent party in district $d$ of country $c$ in year $t$, $\widehat{E}_{d,c,t}$ is predicted Ebola prevalence, $\mu_{d,c}$ represents district fixed effects, and $\gamma_{c,t}$ are country-year dummies.

[51]The sample mean of the vote share for the presidential candidate of the incumbent party is 38%.

[52]This result is robust to dropping one country at a time.

Table 9: Ebola Exposure and Vote Share Presidential Candidate

| | Vote Share Presidential Candidate of Incumbent Party | | | |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Ebola prevalence | 0.207*** | | | |
| | (0.064) | | | |
| High × | | 0.216*** | 0.286** | 0.154* |
| Ebola prevalence | | (0.073) | (0.119) | (0.083) |
| Low × | | -0.007 | 0.139 | 0.272*** |
| Ebola prevalence | | (0.128) | (0.094) | (0.102) |
| District FE | yes | yes | yes | yes |
| Country-year FE | yes | yes | yes | yes |
| Obs. | 122 | 122 | 122 | 122 |
| F-test excl. IV(s) | 6.33 | 3.53, 3,42 | 308.71, 16.27 | 14.50, 66.89 |
| P-value equality (one-sided) | | 0.061 | 0.168 | 0.179 |
| Heterogeneity | | # Ebola facilities | Difference predicted observed prevalence | Representation in government |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Regressions run at the district-year level. Standard errors are clustered at the district level. The F-test represents the first-stage Kleibergen-Paap F-statistic for the excluded instrument(s). P-value equality represents the p-value for the one-sided test of equality of coefficients between High and Low group. In columns (2) and (3), we test the hypothesis High¿Low; in column (4), we test the hypothesis Low¿High. First-stage regressions shown in Tables G.3–G.4. Data sources: Guinea: `http://www.ceniguinee.org`, Liberia: `http://www.necliberia.org`, and Sierra Leone `https://electiondata.io`.

heterogeneities in analogy to Table 8. The results, shown in columns (2)–(4) of Table 9, exhibit the familiar pattern. An Ebola-induced differential increase in the vote share is only observable in districts that experienced an above-median influx of response-related resources. Similarly, the effect of the epidemic is more pronounced in regions where the epidemic was (relatively) less intense than predicted by our simulation model (column (3)). Together, these results suggest that the success of control measures was (partly) attributed to the party of the incumbent president, leading to a relative increase in support in areas where the quantity and implied quality of control efforts was relatively high.[53] These findings parallel results from studies which show that vote shares of incumbents increase when the government's response to natural disasters is robust (e.g., Bechtel and Hainmueller, 2011; Gasper and Reeves, 2011). The result of column (4) further shows that the effect of the epidemic was stronger, albeit statistically insignificantly so, in politically alienated regions. A possible interpretation of this finding is that the signal of quality of the incumbent party was relatively stronger in areas that are excluded from the political decision making process. Taken together, the results of this last part indicate that the

[53]Note that it lies outside the scope of this study to analyse to what extent the Ebola epidemic was exploited for strategic electoral purposes, e.g., by postponing elections or by disproportionately allocating resources to swing districts.

electorate holds the party in power accountable for its action during health emergencies.

# 7 Conclusion

Our study documents that the provision of a common-interest public good—in our case triggered by the need to react to a devastating epidemic—increases state legitimacy, an otherwise slowly evolving determinant of economic development, within a short period of time. This suggests that governments can use one-time, large scale, provisions of common-interest public goods as an instrument to increase legitimacy and thereby overcome historically rooted capacity constraints. The case of the West African Ebola epidemic further suggests that, by supporting government policies, international organizations can play an important role in promoting state legitimacy. When interpreting our results, it is important to keep in mind that we focus on a very specific effect of the West African Ebola epidemic. While Ebola-induced changes in state legitimacy have positive implications for economic development, we cannot draw any conclusions regarding the overall welfare effects of the epidemic. In the short run, the effects of the epidemic have certainly been devastating.

# References


Acaps (2015) 'Ebola in West Africa.' Thematic Note – 24 April 2015

Acemoglu, Daron, Camilo García-Jimeno, and James A. Robinson (2015) 'State capacity and economic development: A network approach.' *American Economic Review* 105(8), 2364–2409

Acemoglu, Daron, Simon Johnson, and James A. Robinson (2001) 'The colonial origins of comparative development: An empirical investigation.' *American Economic Review* 91(5), 1369–1401

Acemoglu, Daron, Simon Johnson, and James Robinson (2003) 'Disease and development in historical perspective.' *Journal of the European Economic Association* 1(2-3), 397–405

Africa APPG (2016) 'Lessons from Ebola affected communities.' Technical Report, Royal African Society

Alesina, Alberto, Bryony Reich, and Alessandro Riboni (2017) 'Nation-building, nationalism and wars.' Working Paper 23435, National Bureau of Economic Research, May

Alsan, Marcella, and Marianne Wanamaker (forthcoming) 'Tuskegee and the health of black men.' *The Quarterly Journal of Economics*

Armah-Attoh, Daniel, and Mina Okuru (2016) 'Liberians on Ebola: Foreign aid most effective, but government performed well, is now better prepared.' Afrobarometer Dispatch 116, Afrobarometer

Autor, David H., David Dorn, Gordon H. Hanson, and Jae Song (2014) 'Trade adjustment: Worker-level evidence.' *The Quarterly Journal of Economics* 129(4), 1799–1860

Backer, Jantien A., and Jacco Wallinga (2016) 'Spatiotemporal analysis of the 2014 Ebola epidemic in West Africa.' *PLOS Computational Biology*

Bechtel, Michael M., and Jens Hainmueller (2011) 'How lasting is voter gratitude? an analysis of the short- and long-term electoral returns to beneficial policy.' *American Journal of Political Science* 55(4), 852–868

Besley, Timothy, and Torsten Persson (2009) 'The origins of state capacity: Property rights, taxation, and politics.' *American Economic Review* 99(4), 1218–1244

_ (2010) 'State capacity, conflict, and development.' *Econometrica* 78(1), 1–34

Blair, Robert A., Benjamin S. Morse, and Lily L. Tsai (2017) 'Public health and public trust: Survey evidence from the Ebola Virus Disease epidemic in Liberia.' *Social Science and Medicine* 172, 89 – 97

Bleakley, Hoyt (2007) 'Disease and development: Evidence from hookworm eradication in the American South.' *The Quarterly Journal of Economics* 122(1), 73–117

Bowles, Jeremy, Jonas Hjort, Timothy Melvin, and Eric Werker (2016) 'Ebola, jobs and economic activity in Liberia.' *Journal of Epidemiology & Community Health* 70(3), 271–277

Burke, Marshall, Solomon Hsiang, and Edward Miguel (2015) 'Global non-linear effect of temperature on economic production.' *Nature* 527, 235–239

Carrión Martín, A.I., T. Derrough, P. Honomou, N. Kolie, B. Diallo, M. Koné, G. Rodier, C. Kpoghomou, and J.M. Jansà (2016) 'Social and cultural factors behind community resistance during an Ebola outbreak in a village of the Guinean Forest region, February 2015: a field experience.' *International Health* 8(3), 227–229

Carter, Simone E., Marion O'Reilly, Jack Frith-Powell, Alpha Umar Kargbo, Daniel Byrne, and Eva Niederberger (2017) 'Treatment seeking and Ebola community care centers in Sierra Leone: A qualitative study.' *Journal of Health Communication* 22(sup1), 66–71. PMID: 28854139

Center for International Earth Science Information Network -CIESIN (2016) 'Gridded population of the World, version 4 (gpwv4): Population count'

Chan, Margaret (2014) 'Ebola Virus Disease in West Africa - no early end to the outbreak.' *New England Journal of Medicine* 371(13), 1183–1185. PMID: 25140856

Chowell, Gerardo, Cécile Viboud, Lone Simonsen, Stefano Merler, and Alessandro Vespignani (2017) 'Perspectives on model forecasts of the 2014-2015 Ebola epidemic in West Africa: lessons and the way forward.' *BMC Medicine* 15(1), 42

Chretien, Jean-Paul, Steven Riley, and Dylan B George (2015) 'Mathematical modeling of the West Africa Ebola epidemic.' *eLife* 4, e09186

Cole, Shawn, Andrew Healy, and Eric Werker (2012) 'Do voters demand responsive governments? Evidence from Indian disaster relief.' *Journal of Development Economics* 97(2), 167 – 181

Conley, T. G. (1999) 'GMM estimation with cross sectional dependence.' *Journal of Econometrics* 92(1), 1–45

Conley, Timothy G., Christian B. Hansen, and Peter E. Rossi (2012) 'Plausibly exogenous.' *The Review of Economics and Statistics* 94(1), 260–272

Corbridge, Stuart, Glyn Williams, and Manoj Srivastava (2005) *Seeing the State: Governance and Governmentality in India* (Cambridge University Press)

Davis, Peter (2015) 'Ebola in Sierra Leone: Economic impact & recovery.' Technical Report, Department for International Development, London

Dell, Melissa, and Pablo Querubin (2018) 'Nation building through foreign intervention: Evidence from discontinuities in military strategies.' *Quarterly Journal of Economics* 133(2), 701–764

Dell, Melissa, Nathan Lane, and Pablo Querubin (2017) 'The historical state, local collective action, and economic development in Vietnam.' Technical Report

Dincecco, Mark, and Gabriel Katz (2016) 'State capacity and long-run economic performance.' *Economic Journal* 126(590), 189–218

DuBois, Marc, Caitlin Wak, Scarlett Sturridge, and Christina Bennett (2015) 'The Ebola response in West Africa.' Technical Report, Overseas Development Institute

Easton, David (1965) *Systems Analysis of Political Life Hardcover* (John Wiley & Sons Ltd)

Englebert, Pierre (2002) *State Legitimacy and Development in Africa* (Lynne Rienner Publishers)

Fair, C. Christine, Patrick Kuhn, and Neil A. Malhotra and (2017) 'Natural disasters and political engagement: Evidence from the 2010-11 Pakistani floods.' Stanford University Graduate School of Business Research Paper 17-42, Stanford University Graduate School of Business

Fukuyama, Francis (2015) 'Why is democracy performing so poorly?' *Journal of Democracy* 26, 11–20

Gallego, Jorge (2015) 'Natural disasters and clientelism: the case of floods and landslides in Colombia.' Documentos de Trabajo 012537, Universidad del Rosario, February

Gallup, John Luke, and Jeffrey D. Sachs (2001) 'The economic burden of malaria.' *The American Journal of Tropical Medicine and Hygiene* 64(1 suppl.), 85–96

Gasper, John T., and Andrew Reeves (2011) 'Make it rain? Retrospection and the attentive electorate in the context of natural disasters.' *American Journal of Political Science* 55(2), 340–355

Gennaioli, Nicola, and Hans-Joachim Voth (2015) 'State capacity and military conflict.' *The Review of Economic Studies* 82(4), 1409–1448

Gennaioli, Nicola, and Ilia Rainer (2007) 'The modern impact of precolonial centralization in africa.' *Journal of Economic Growth* 12(3), 185–234

Gignoux, Etienne, Rachel Idowu, Luke Bawo, Lindis Hurum, Armand Sprecher, Mathieu Bastard, and Klaudia Porten (2015) 'Use of capture–recapture to estimate underreporting of Ebola Virus Disease, Montserrado county, Liberia.' *Emerging Infectious Diseases* 21(12), 2266–2267

Gilley, Bruce (2009) *The Right to Rule: How States Win and Lose Legitimacy* (Columbia University Press)

Gire, Stephen K., Augustine Goba, Kristian G. [...] Andersen, and Pardis C. Sabeti (2014) 'Genomic surveillance elucidates Ebola virus origin and transmission during the 2014 outbreak.' *Science* 345(6202), 1369–1372

Harari, Mariaflavia, and Eliana La Ferrara (Forthcoming) 'Conflict, climate and cells: A disaggregated analysis.' *Review of Economics and Statistics*

Healy, Andrew, and Neil Malhotra (2010) 'Random events, economic losses, and retrospective voting: Implications for democratic competence.' *Quarterly Journal of Political Science* 5(2), 193–208

Henao-Restrepo, Ana Maria, AntonCamacho, and Marie-Paule Kieny (2017) 'Efficacy and effectiveness of an rVSV-vectored vaccine in preventing Ebola virus disease: Final results

from the Guinea ring vaccination, open-label, cluster-randomised trial (Ebola ca suffit!).' *The Lancet* 389(10068), 505–518

Hutchison, Marc L, and Kristin Johnson (2011) 'Capacity to trust? institutional capacity, conflict, and political trust in Africa, 2000–2005.' *Journal of Peace Research* 48(6), 737–752

IIASA/FAO (2012) 'Global agro-ecological zones (gaez v3.0).' Laxenburg, Austria and FAO, Rome, Italy

Levi, Margaret (2006) 'Why we need a new theory of government.' *Perspectives on Politics* 4(1), 5–19

Lowes, Sara, and Eduardo Montero (2018) 'The legacy of colonial medicine in Central Africa.' Technical Report

Maganga, Gaël D., Jimmy Kapetshi, Nicolas [...] Berthet, and Eric M. Leroy (2014) 'Ebola Virus Disease in the Democratic Republic of Congo.' *New England Journal of Medicine* 371(22), 2083–2091. PMID: 25317743

Manguvo, Angellar, and Benford Mafuvadze (2015) 'The impact of traditional and religious practices on the spread of Ebola in West Africa: time for a strategic shift.' *The Pan African Medical Journal* 22(Suppl 1), 9

Mark, Monica (2014) 'Ebola crisis: how health workers on West African frontline are paying with their lives.' *The Guardian*

Marshall, Burke, Gong Erick, and Jones Kelly 'Income shocks and hiv in africa.' *The Economic Journal* 125(585), 1157–1189

Marston, Barbara J., Amanda van Steelandt E. Kainne Dokubo, Lise Martel, Desmond Williams, Sara Hersey, Amara Jambai, Sakoba Keita, Tolbert G. Nyenswah, , and John T. Redd (2017) 'Ebola response impact on public health programs, West Africa, 2014-2017.' *Emerging Infectious Diseases*

Maxmen, Amy (2017) 'Ebola vaccine approved for use in ongoing outbreak.' *Nature News 30 May 2017*

Michalopoulos, Stelios, and Elias Papaioannou (2013) 'Pre-colonial ethnic institutions and contemporary African development.' *Econometrica* 81(1), 113–15

Migdal, Joel S. (1988) *Strong Societies and Weak States: State-society Relations and State Capabilities in the Third World* (Princeton University Press)

Neal, Anthony (2015) 'Keeping the faith.' Technical Report, CAFOD

Newton, Kenneth (2007) *Social and Political Trust* (Oxford University Press)

North, Douglass C. (1981) *Structure and Change in Economic History* (WW Norton & Company)

Nunn, Nathan (2008) 'The long-term effects of Africa's slave trades.' *The Quarterly Journal of Economics* 123(1), 139–176

Nunn, Nathan, and Leonard Wantchekon (2011) 'The slave trade and the origins of mistrust in Africa.' *American Economic Review* 101(7), 3221–3252

Overseas Security Advisory Council (2015) 'Impact of Ebola in Sierra Leone.' In 'Sierra Leone 2015 Crime and Safety Report'

Rewar, Suresh, and Dashrath Mirdha (2014) 'Transmission of Ebola Virus Disease: An overview.' *Annals of Global Health* 80(6), 444 – 451. Tropical Medicine in the Era of Global Connectivity

RIWI (2015) 'Perceptions of Ebola in Nigeria, Liberia, and Sierra Leone.' Technical Report, RIWI Corp.

Rohner, Dominic, Mathias Thoenig, and Fabrizio Zilibotti (2013) 'Seeds of distrust: conflict in uganda.' *Journal of Economic Growth* 18(3), 217–252

Rotberg, Robert (2003) 'The failure and collapse of nation-states: Breakdown, prevention, and repair.' In 'When States Fail: Causes and Consequences' (Princeton University Press)

Sachs, Jeffrey, and Pia Malaney (2002) 'The economic and social burden of malaria.' *Nature* 415, 680–685

Scarpino, Samuel V., Atila Iamarino, Chad [...] Wells, and Jeffrey P. Townsend (2015) 'Epidemiological and viral genomic sequence analysis of the 2014 Ebola outbreak reveals clustered transmission.' *Clinical Infectious Diseases* 60(7), 1079

Thomas, Mark Roland, Gregory Smith, Francisco H. G. Ferreira, David Evans, Maryla Maliszewska, Marcio Cruz, Kristen Himelein, and Mead Over (2015) 'The economic impact of Ebola on sub-Saharan Africa: updated estimates for 2015.' Technical Report, World Bank Group

Tollefsen, Andreas Forø, Håvard Strand, and Halvard Buhaug (2012) 'Prio-grid: A unified spatial data structure.' *Journal of Peace Research* 49(2), 363–374

Tsai, Lily, Robert Blair, and Benjamin Morse (2015) 'Patterns of trust and compliance in the fight against Ebola: Results from a population-based survey of Monrovia, Liberia.' Economic impacts of Ebola bulletin three, International Growth Centre

UNICEF (2016) 'Ebola community care centers: Lessons learned from unicef's 2014-2015

experience in sierra leone.' 3 UN Plaza, New York, NY 10017

Van Kerkhove, Maria D., Ana I. Bento, Harriet L. Mills, Neil M. Ferguson, and Christl A. Donnelly (2015) 'A review of epidemiological parameters from Ebola outbreaks to inform early public health decision-making.' *Scientific data* 2, 150019 EP –

Weatherford, M. Stephen (1992) 'Measuring political legitimacy.' 86(1), 149–166

Weber, Max (1984) 'Legitimacy, politics and the state.' In *Legitimacy and the State,* ed. William Connolly (New York University Press)

Wesolowski, Amy, Wendy Prudhomme O'Meara, Nathan Eagle, Andrew J Tatem, and Caroline O Buckee (2015) 'Evaluating spatial interaction models for regional mobility in sub-Saharan Africa.' *PLoS computational biology* 11(7), e1004267

WHO (2014) 'Barriers to rapid containment of the Ebola outbreak.' In 'Ebola situation assessment' (World Health Organization)

WHO (2015) 'Health worker Ebola infections in Guinea, Liberia and Sierra Leone.' *World Health Organization*

WHO Ebola Response Team (2014) 'Ebola Virus Disease in West Africa - the first 9 months of the epidemic and forward projections.' *New England Journal of Medicine* 371(16), 1481–1495

_ (2016) 'After Ebola in West Africa - unpredictable risks, preventable epidemics.' *New England Journal of Medicine* 375(6), 587–596

Wimmer, Andreas, Lars-Erik Cederman, and Brian Min (2009) 'Ethnic politics and armed conflict: A configurational analysis of a new global data set.' *American Sociological Review* 74(2), 316–337

World Health Organization (2014a) 'Barriers to rapid containment of the Ebola outbreak.' *Ebola Response Roadmap Situation Report*

_ (2014b) 'Ebola situation assessment 14 August 2014'

_ (2015) 'Factors that contributed to undetected spread of the Ebola virus and impeded rapid containment.' *One year into the Ebola epidemic, January 2015*

_ (2016a) 'Situation Report Ebola Virus Disease 10 June 2016'

_ (2016b) 'Situation Report Ebola Virus Disease 30 March 2016'

# Appendices

## A Afrobarometer Questions

Table A.1: Parameter Values Simulations

| Variable Name | Afrobarometer Question |
|---|---|
| Trust in Parliament | How much do you trust each of the following, or haven't you heard enough about them to say: The President? |
| Trust in President | How much do you trust each of the following, or haven't you heard enough about them to say: Parliament? |
| Trust in Police | How much do you trust each of the following, or haven't you heard enough about them to say: The Police? |
| Refusal to Pay Taxes | Here is a list of actions that people sometimes take as citizens when they are dissatisfied with government performance. For each of these, please tell me whether you, personally, have done any of these things during the past year. If not, would you do this if you had the chance: Refused to pay a tax or fee to government. |

Notes: Possible responses are ordinal. Values between 0 (low) and 3 (high).

# B Probability Density Functions

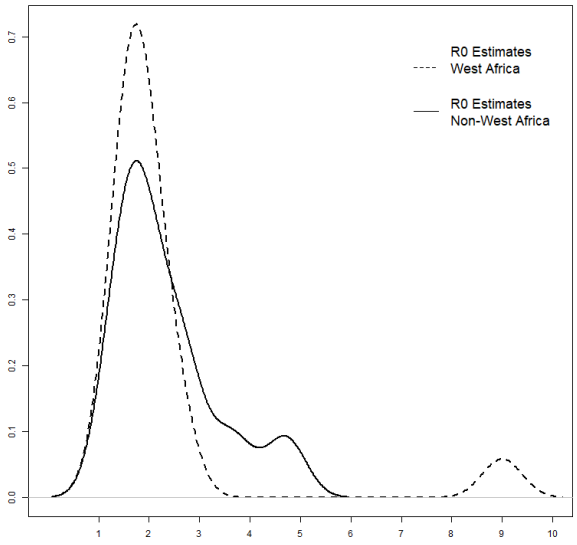


Figure B.1: Figure depicts the Kernel density of $R(t)$ estimates reviewed in Van Kerkhove et al. (2015) for two subsets of studies: studies that base estimates on data of the West African Ebola epidemic (2013–2016) and studies that rely on non-West African data for estimation.

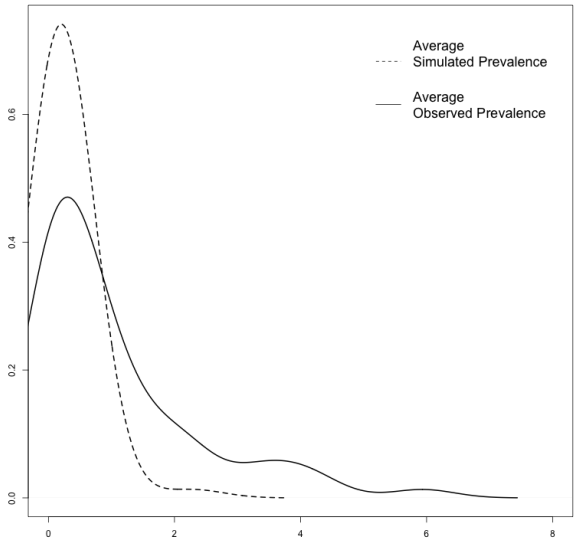


Figure B.2: Figure depicts the Kernel densities of predicted and observed average prevalence rates.

# C Reduced-Form Regressions

Table C.1: Simulated Ebola Exposure and State Legitimacy

| | Trust in Parliament | Trust in President | Trust in Police | Refusal to Pay Taxes |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Simulated | 0.472*** | 0.489*** | 0.388** | -0.543*** |
| Ebola prevalence | (0.169) | (0.167) | (0.160) | (0.169) |
| Country×Year FE | yes | yes | yes | yes |
| District FE | yes | yes | yes | yes |
| Individual-level controls×year FE | yes | yes | yes | yes |
| Obs. | 6,201 | 6,201 | 6,201 | 6,201 |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are clustered at the district level.

# D Data Construction Neighbouring Countries

The individual-level dataset for the three countries neighbouring the epicentre of the Ebola epidemic—Ivory Coast, Mali and Senegal—is constructed in analogy to our main dataset. Pre-epidemic information is drawn from Afrobarometer survey round 5, post-epidemic

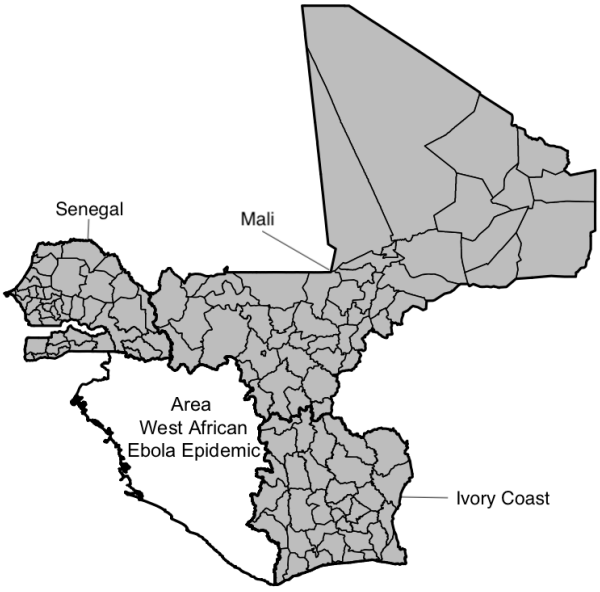


Figure D.1: Figure depicts subnational units (shaded grey) in Ivory Coast, Mali and Senegal for which prevalence rates are predicted using simulation model Eq.(6).

data from round 6. Simulated Ebola prevalence is generated using the simulation model described in Section 5.2 (Eq.(6)). We start the simulations in Guéckédou and then analyse how the epidemic is predicted to spread across the districts (defined as the second subnational administrative level) of the three neighbouring countries at a weekly interval. The geographic scope of the dataset along with the units for which Ebola exposure is predicted is shown in Figure D.1.

Following the procedure outlined in Section 5, we run these simulations one thousand times and compute the mode for each district and week. Simulated Ebola exposure is then computed as the average predicted Ebola prevalence between the start of the epidemic and the beginning of the survey fieldwork in the district in which the individual resides.[54] To investigate whether simulated prevalence influences state legitimacy, we run the following

[54]Fieldwork in Ivory Coast started on 25 August 2014, in Mali on 1 December 2014, and in Senegal on 22 November 2014.

reduced-form regressions:

$$y_{i,d,c,t} = \theta\, S_{i,d,c,t} + \Omega\, \mathbf{X}_{i,d,c,t} + \alpha_{d,c} + \tau_{c,t} + \psi_{i,d,c,t}, \tag{D.1}$$

where $y_{i,d,c,t}$ represents the outcome for individual $i$ residing in district $d$ and country $c$ in year $t$. Predicted Ebola prevalence ($S_{i,d,c,t}$), reflects the average simulated number of Ebola cases per 100,000 people in individual $i$'s district of residence $d$ (located in country $c$) in year $t$. The vector $\mathbf{X}_{i,d,c,t}$ contains individual-level controls. In all regressions we allow these characteristics to have different effects in the pre- and post epidemic era by interacting them with a year-specific dummy. District-specific fixed effects are symbolised by $\alpha_{d,c}$, country-time-fixed effects by $\tau_{c,t}$, and the idiosyncratic error term by $\psi_{i,d,c,t}$. The regression results are depicted in Table 5, columns (5)–(8).

# E Robustness

Table E.1: Robustness Inclusion of District-Level Characteristics

| | Trust in Parliament | Trust in President | Trust in Police | Refusal to Pay Taxes |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Ebola prevalence | 0.143** | 0.142** | 0.153** | -0.153** |
| | (0.073) | (0.065) | (0.076) | (0.067) |
| | First stage regression: Ebola prevalence | | | |
| Simulated Ebola prevalence | 2.813*** | 2.813*** | 2.813*** | 2.813*** |
| | (0.682) | (0.682) | (0.693) | (0.682) |
| Country×year FE | yes | yes | yes | yes |
| District FE | yes | yes | yes | yes |
| Individual-level controls×year FE | yes | yes | yes | yes |
| Time-invariant district-level characteristics×year FE | yes | yes | yes | yes |
| Time-varying district-level characteristics | yes | yes | yes | yes |
| Obs. | 6,201 | 6,201 | 6,201 | 6,201 |
| F-test excl. IV | 17.02 | 17.02 | 17.02 | 17.02 |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are clustered at the district level. The F-test represents the first-stage Kleibergen-Paap F-statistic for the excluded instrument.

Table E.2: (Simulated) Ebola prevalence computed using time-period December 2013–October 2014.

| | Trust in Parliament | Trust in President | Trust in Police | Refusal to Pay Taxes |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Ebola prevalence | 0.119*** | 0.123*** | 0.098*** | -0.131** |
| | (0.039) | (0.035) | (0.035) | (0.060) |
| | First stage regression: Ebola prevalence | | | |
| Simulated Ebola prevalence | 2.855*** | 2.855*** | 2.855*** | 2.855*** |
| | (0.695) | (0.695) | (0.695) | (0.695) |
| Country×year FE | yes | yes | yes | yes |
| District FE | yes | yes | yes | yes |
| Individual-level controls×year FE | yes | yes | yes | yes |
| Obs. | 6,201 | 6,201 | 6,201 | 6,201 |
| F-test excl. IV | 16.86 | 16.86 | 16.86 | 16.86 |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are clustered at the district level. The F-test represents the first-stage Kleibergen-Paap F-statistic for the excluded instrument.

Table E.3: Plausibly Exogenous IVs: Union of Confidence Intervals

| | | Trust in Parliament | Trust in President | Trust in Police | Refusal to Pay Taxes |
|---|---|---|---|---|---|
| | | (1) | (2) | (3) | (4) |
| Point estimates main IV regressions (Table 4) | | 0.174 | 0.138 | 0.114 | -0.219 |
| $\sigma = 0.00$ | 95CI Lower Bound | 0.049 | 0.071 | 0.039 | -0.358 |
| | 95CI Upper Bound | 0.282 | 0.272 | 0.233 | -0.023 |
| $\sigma = 0.05$ | 95CI Lower Bound | 0.04 | 0.065 | 0.035 | -0.367 |
| | 95CI Upper Bound | 0.289 | 0.279 | 0.238 | -0.019 |
| $\sigma = 0.15$ | 95CI Lower Bound | 0.034 | 0.054 | 0.025 | -0.386 |
| | 95CI Upper Bound | 0.303 | 0.293 | 0.249 | -0.010 |
| $\sigma = 0.25$ | 95CI Lower Bound | 0.024 | 0.043 | 0.016 | -0.404 |
| | 95CI Upper Bound | 0.317 | 0.307 | 0.259 | -0.001 |
| Obs. | | 6,201 | 6,201 | 6,201 | 6,201 |

Note: The table reports the point estimates of Table 4 along with the corresponding confidence intervals computed based on the 'Union of Confidence Intervals' (UI) method proposed by Conley et al. (2012). The UI method relaxes the assumption that the instrument (i.e., in our case simulated Ebola prevalence) is strictly exogenous and allows for a direct influence, $\gamma$, of the instrument on the outcome variables. The UI methods requires a pre-specified support of $\gamma$. Following Conley et al. (2012), we define the support as $[-2|\beta|\delta, 2|\beta|\delta]$ where $\beta$ are our IV estimates resulting from our baseline specification in Table 4 and $\delta$ varies within the range $[0, 0.25]$. Standard errors are clustered at the district level.

Table E.4: Exposure measure defined as log prevalence + 1

| | Trust in Parliament | Trust in President | Trust in Police | Refusal to Pay Taxes |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Log Ebola prevalence +1 | 0.344** | 0.399*** | 0.327*** | -0.417** |
| | (0.146) | (0.130) | (0.120) | (0.195) |
| | First stage regression: Log (Ebola prevalence +1) | | | |
| Log simulated Ebola prevalence + 1 | 1.896*** | 1.896*** | 1.896*** | 1.896*** |
| | (0.295) | (0.295) | (0.295) | (0.295) |
| Country×Year FE | yes | yes | yes | yes |
| District FE | yes | yes | yes | yes |
| Individual-level controls×year FE | yes | yes | yes | yes |
| Obs. | 6,201 | 6,201 | 6,201 | 6,201 |
| F-test excl. IV | 41.44 | 41.44 | 41.44 | 41.44 |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are clustered at the district level. The F-test represents the first-stage Kleibergen-Paap F-statistic for the excluded instrument.

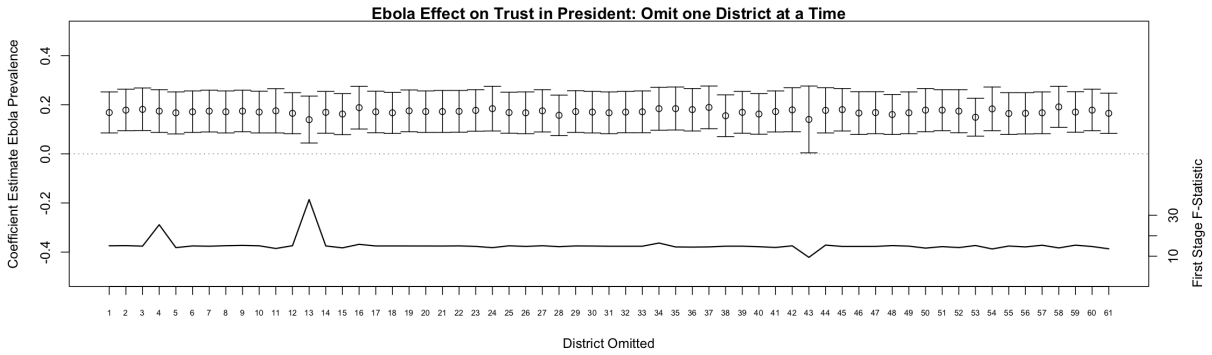


Figure E.1: Dropping one district at a time: president (district IDs listed in Table E.5). The circles represent the point estimates, the bars indicate the 90 percent confidence intervals, where the standard errors are clustered at the district level.

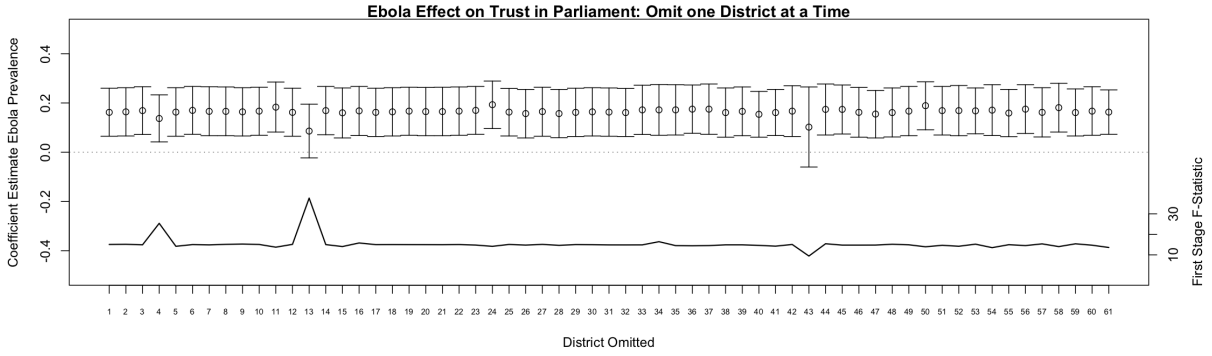


Figure E.2: Dropping one district at a time: parliament (district IDs listed in Table E.5). The circles represent the point estimates, the bars indicate the 90 percent confidence intervals, where the standard errors are clustered at the district level.

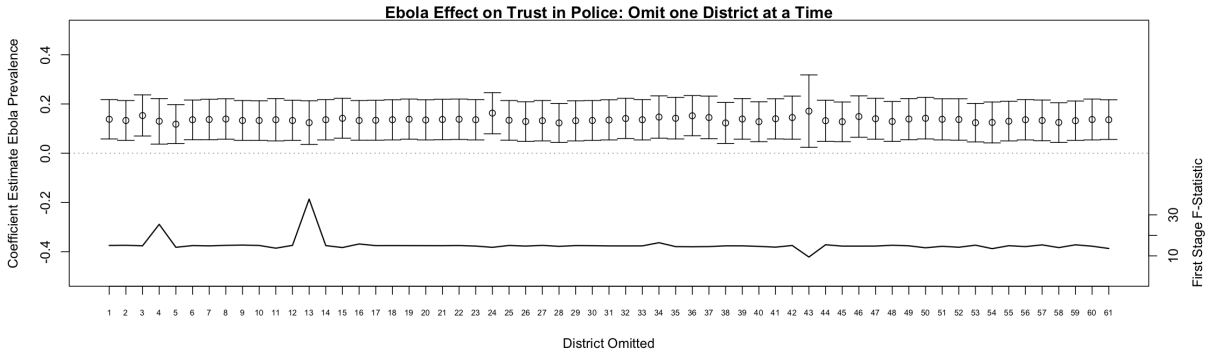


Figure E.3: Dropping one district at a time: police (district IDs listed in Table E.5). The circles represent the point estimates, the bars indicate the 90 percent confidence intervals, where the standard errors are clustered at the district level.

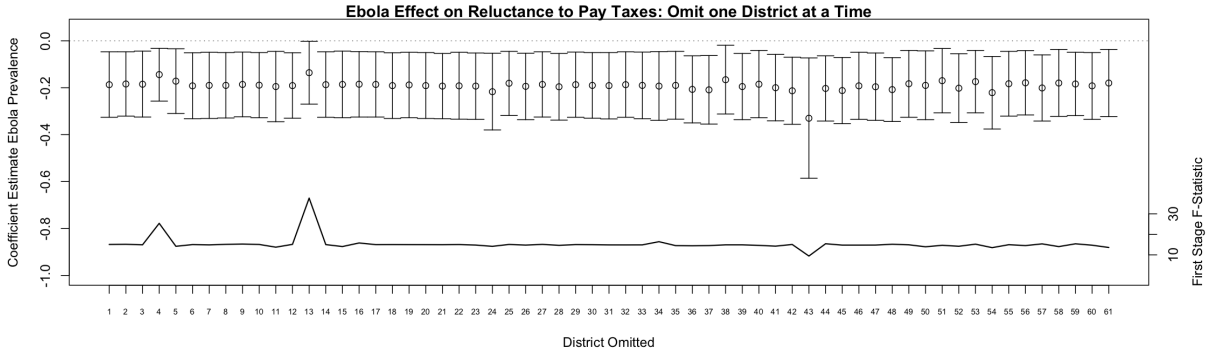


Figure E.4: Dropping one district at a time: refusal to pay taxes (district IDs listed in Table E.5). The circles represent the point estimates, the bars indicate the 90 percent confidence intervals, where the standard errors are clustered at the district level.

Table E.5: District IDs

| ID | District | Country | ID | District | Country |
|---|---|---|---|---|---|
| 1 | Beyla | GIN | 32 | Yamou | GIN |
| 2 | Boffa | GIN | 33 | Bomi | LBR |
| 3 | Boké | GIN | 34 | Bong | LBR |
| 4 | Conakry | GIN | 35 | Gbapolu | LBR |
| 5 | Coyah | GIN | 36 | Grand Cape Mount | LBR |
| 6 | Dabola | GIN | 37 | Grand Bassa | LBR |
| 7 | Dalaba | GIN | 38 | Grand Gedeh | LBR |
| 8 | Dinguiraye | GIN | 39 | Grand Kru | LBR |
| 9 | Dubréka | GIN | 40 | Lofa | LBR |
| 10 | Faranah | GIN | 41 | Margibi | LBR |
| 11 | Forécariah | GIN | 42 | Maryland | LBR |
| 12 | Gaoual | GIN | 43 | Montserrado | LBR |
| 13 | Guéckédou | GIN | 44 | Nimba | LBR |
| 14 | Kankan | GIN | 45 | River Cess | LBR |
| 15 | Kindia | GIN | 46 | River Gee | LBR |
| 16 | Kissidougou | GIN | 47 | Sinoe | LBR |
| 17 | Koubia | GIN | 48 | Bo | SLE |
| 18 | Koundara | GIN | 49 | Bombali | SLE |
| 19 | Kouroussa | GIN | 50 | Bonthe | SLE |
| 20 | Kérouané | GIN | 51 | Kailahun | SLE |
| 21 | Labé | GIN | 52 | Kambia | SLE |
| 22 | Lola | GIN | 53 | Kenema | SLE |
| 23 | Lélouma | GIN | 54 | Koinadugu | SLE |
| 24 | Macenta | GIN | 55 | Kono | SLE |
| 25 | Mali | GIN | 56 | Moyamba | SLE |
| 26 | Mamou | GIN | 57 | Port Loko | SLE |
| 27 | Nzérékoré | GIN | 58 | Pujehun | SLE |
| 28 | Pita | GIN | 59 | Tonkolili | SLE |
| 29 | Siguiri | GIN | 60 | Western Rural | SLE |
| 30 | Tougué | GIN | 61 | Western Urban | SLE |
| 31 | Télimélé | GIN | | | |

Table E.6: Conley (1999) Standard Error Clustering Approach: Euclidean Distance Weighting Kernel

| | Trust in Parliament | Trust in President | Trust in Police | Refusal to Pay Taxes |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Ebola prevalence | 0.165*** | 0.172*** | 0.136*** | -0.191** |
| | (0.057) | (0.053) | (0.052) | (0.093) |
| | First stage regression: Ebola prevalence | | | |
| Simulated Ebola prevalence | 2.851*** | 2.851*** | 2.851*** | 2.851*** |
| | (0.722) | (0.722) | (0.722) | (0.722) |
| Country×year FE | yes | yes | yes | yes |
| District FE | yes | yes | yes | yes |
| Individual-level controls×year FE | yes | yes | yes | yes |
| Obs. | 6,201 | 6,201 | 6,201 | 6,201 |
| F-test excl. IV | 15.55 | 15.55 | 15.55 | 15.55 |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are computed using the approach proposed by Conley (1999). The Euclidean distance weighting kernel decays linearly (cutoff: 150 kilometers). The F-test represents the first-stage Kleibergen-Paap F-statistic for the excluded instrument.

Table E.7: Conley (1999) Standard Error Clustering Approach: Network Connectivity weighted kernel

| | Trust in Parliament | Trust in President | Trust in Police | Refusal to Pay Taxes |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Ebola prevalence | 0.165*** | 0.172*** | 0.136*** | -0.191** |
| | (0.057) | (0.051) | (0.050) | (0.089) |
| | First stage regression: Ebola prevalence | | | |
| Simulated Ebola prevalence | 2.851*** | 2.851*** | 2.851*** | 2.851*** |
| | (0.702) | (0.702) | (0.702) | (0.702) |
| Country×year FE | yes | yes | yes | yes |
| District FE | yes | yes | yes | yes |
| Individual-level controls×year FE | yes | yes | yes | yes |
| Obs. | 6,201 | 6,201 | 6,201 | 6,201 |
| F-test excl. IV | 16.34 | 16.34 | 16.34 | 16.34 |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are computed using the approach proposed by Conley (1999) adapted to the bilateral connectivity within our simulation network ($m_{i,j}$ in Eq. (5)). The F-test represents the first-stage Kleibergen-Paap F-statistic for the excluded instrument.

Table E.8: Including spatial lags: First-stage and reduced-form estimates

| | First-Stage Regression | Reduced-Form Regressions | | | |
|---|---|---|---|---|---|
| | Observed Ebola Prevalence | Trust in Parliament | Trust in President | Trust in Police | Refusal to Pay Taxes |
| | (1) | (2) | (3) | (4) | (5) |
| Simulated | 0.666*** | 0.119*** | 0.119*** | 0.092*** | -0.123*** |
| Ebola prevalence$_d$ (SD) | (0.180) | (0.036) | (0.033) | (0.031) | (0.033) |
| $\mathbf{W}$ × simulated | 0.325 | -0.018 | 0.017 | 0.034 | -0.093 |
| Ebola prevalence$_{j\neq d}$ (SD) | (0.239) | (0.080) | (0.090) | (0.082) | (0.111) |
| Country×Year FE | yes | yes | yes | yes | yes |
| District FE | yes | yes | yes | yes | yes |
| Individual-level controls×year FE | yes | yes | yes | yes | yes |
| Obs. | 6,201 | 6,201 | 6,201 | 6,201 | 6,201 |

Notes: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are computed using the approach proposed by Conley (1999). The Euclidean distance weighting kernel decays linearly (cut-off: 150 kilometres).

Table reports results of the following regressions:

$$y_{i,d,c,t} = \theta\, S_{d,c,t} + \Phi \sum_{j \neq d} W_{d,j} \times S_{j,c,t} + \Omega\, \mathbf{X}_{i,d,c,t} + \alpha_{d,c} + \tau_{c,t} + \psi_{i,d,c,t}.$$

$y_{i,d,c,t}$ represents the outcome for individual $i$ residing in district $d$ and country $c$ in year $t$. Simulated prevalence for district $d$ is represented by $S_{d,c,t}$. The summation term captures the distance-weighted average simulated prevalence in the first and second neighbour district ($j \neq d$). The matrix $\mathbf{W}$ represents a weighting matrix, where weights are defined as the inverse bilateral distance between district $d$ and $j$. For ease of comparability, both simulated prevalence and (spatially) lagged simulated prevalence are standardised to a mean of zero and a standard deviation of one.

Table E.9: Including spatial lags: 2SLS-IV regressions

| | Trust in Parliament | Trust in President | Trust in Police | Refusal to Pay Taxes |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Ebola | 0.169*** | 0.168*** | 0.130*** | -0.173** |
| prevalence | (0.056) | (0.052) | (0.053) | (0.077) |
| $\mathbf{W}$ × simulated | -0.018 | -0.017 | -0.033 | -0.093 |
| Ebola prevalence$_{j\neq d}$ (SD) | (0.095) | (0.091) | (0.081) | (0.122) |
| | First stage regression: Ebola prevalence | | | |
| Simulated | 0.666*** | 0.666*** | 0.666*** | 0.666*** |
| Ebola prevalence$_d$ (SD) | (0.180) | (0.180) | (0.180) | (0.180) |
| $\mathbf{W}$ × simulated | 0.325 | 0.325 | 0.325 | 0.325 |
| Ebola prevalence$_{j\neq d}$ (SD) | (0.239) | (0.239) | (0.239) | (0.239) |
| Country×year FE | yes | yes | yes | yes |
| District FE | yes | yes | yes | yes |
| Individual-level controls×year FE | yes | yes | yes | yes |
| Obs. | 6,201 | 6,201 | 6,201 | 6,201 |
| F-test excl. IV | 13.70 | 13.70 | 13.70 | 13.70 |

Notes: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are computed using the approach proposed by Conley (1999). The Euclidean distance weighting kernel decays linearly (cut-off: 150 kilometres).

Table reports results of the following (second-stage) regressions:

$$y_{i,d,c,t} = \beta\, \widehat{E}_{i,d,c,t} + \Phi \sum_{j \neq d} W_{d,j} \times S_{j,c,t} + \theta, \mathbf{X}_{i,d,c,t} + \mu_{d,c} + \gamma_{c,t} + \varepsilon_{d,c,t}.$$

$y_{i,d,c,t}$ represents the outcome for individual $i$ residing in district $d$ and country $c$ in year $t$. The term $\widehat{E}_{i,d,c,t}$ symbolises predicted Ebola prevalence obtained from the first stage regression (see Eq.(1)). The summation term captures the distance-weighted average of simulated prevalence in the first and second neighbour district ($j \neq d$). The matrix $\mathbf{W}$ represents a weighting matrix, where weights are defined as the inverse bilateral distance between district $d$ and $j$. For ease of comparability, both simulated prevalence and (spatially) lagged simulated prevalence are standardised to a mean of zero and a standard deviation of one.

Table E.10: Varying $R_0$ ($R_0 = 1.56$): Ebola Exposure and State Legitimacy

| | Trust in Parliament | Trust in President | Trust in Police | Refusal to Pay Taxes |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Ebola prevalence | 0.227** | 0.215** | 0.157* | -0.280** |
| | (0.091) | (0.090) | (0.083) | (0.121) |
| | First stage regression: Ebola prevalence | | | |
| Simulated Ebola prevalence | 8.347*** | 8.347*** | 8.347*** | 8.347*** |
| | (3.222) | (3.222) | (3.222) | (3.222) |
| Country×year FE | yes | yes | yes | yes |
| District FE | yes | yes | yes | yes |
| Individual-level controls×year FE | yes | yes | yes | yes |
| Obs. | 6,201 | 6,201 | 6,201 | 6,201 |
| F-test excl. IV | 6.709 | 6.709 | 6.709 | 6.709 |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are clustered at the district level. The F-test represents the first-stage Kleibergen-Paap F-statistic for the excluded instrument.

Table E.11: Varying $R_0$ ($R_0$=2.51): Ebola Exposure and State Legitimacy

| | Trust in Parliament | Trust in President | Trust in Police | Refusal to Pay Taxes |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Ebola prevalence | 0.143*** | 0.147*** | 0.106** | -0.152** |
| | (0.053) | (0.048) | (0.049) | (0.077) |
| | First stage regression: Ebola prevalence | | | |
| Simulated Ebola prevalence | 3.191*** | 3.191*** | 3.191*** | 3.191*** |
| | (0.627) | (0.627) | (0.627) | (0.627) |
| Country×year FE | yes | yes | yes | yes |
| District FE | yes | yes | yes | yes |
| Individual-level controls×year FE | yes | yes | yes | yes |
| Obs. | 6,201 | 6,201 | 6,201 | 6,201 |
| F-test excl. IV | 25.93 | 25.93 | 25.93 | 25.93 |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are clustered at the district level. The F-test represents the first-stage Kleibergen-Paap F-statistic for the excluded instrument.

# F Summary Statistics GeoPoll Survey Data

Table F.1: Descriptive Statistics SMS-Based Survey

| **Variable** | **Mean** | **Std. Dev.** | **Min.** | **Max.** | **Obs.** |
|---|---|---|---|---|---|
| | Geopoll - Mobile Phone Survey data (Liberia) | | | | |
| Perceived Ebola Risk (SD) | 0 | 1 | -1.928 | 1.030 | 403 |
| Valuation Control Measures (SD) | 0 | 1 | -2.915 | 0.958 | 403 |
| Success Control Measures (SD) | 0 | 1 | -3.430 | 0.858 | 403 |
| Willingness to Finance Public Goods (SD) | 0 | 1 | -2.160 | 0.461 | 403 |
| Age | 32.503 | 9.181 | 17 | 87 | 403 |
| Gender | 0.429 | 0.496 | 0 | 1 | 403 |
| Education | 2.141 | 0.8149 | 0 | 3 | 403 |
| Urban | 0.905 | 0 .292 | 0 | 1 | 403 |
| | Control Variables Afrobarometer (Liberia) | | | | |
| Age | 35.262 | 11.463 | 18 | 99 | 2,095 |
| Gender | 0.501 | 0.500 | 0 | 1 | 2,095 |
| Education | 1.548 | 1.004 | 0 | 3 | 2,095 |
| Urban | 0.488 | 0.500 | 0 | 1 | 2,095 |

Note: Gender is an indicator variable that takes value one if the individual is female, and zero otherwise. Education measures the level of formal schooling: 0: no formal education; 1: primary education; 2: secondary education; 3: post-secondary education. Urban is an indicator variable that takes value one if the individual lives in a city or town, and zero otherwise.

# G First-Stage Regressions Heterogeneities

Table G.1: First-Stage Regressions High Groups

| | Dependent Variable: High Group × Ebola Prevalence | | | | | |
|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| High × simulated Ebola prevalence | 2.290*** | 1.069*** | 4.859*** | 2.290*** | 1.069*** | 4.859*** |
| | (0.715) | (0.125) | (0.794) | (0.715) | (0.125) | (0.794) |
| Low × simulated Ebola prevalence | -0.089 | 0.025 | 0.036 | -0.089 | 0.025 | 0.036 |
| | (0.754) | (0.026) | (0.088) | (0.754) | (0.026) | (0.088) |
| Country×year FE | yes | yes | yes | yes | yes | yes |
| District FE | yes | yes | yes | yes | yes | yes |
| Individual controls×year FE | yes | yes | yes | yes | yes | yes |
| Obs. | 6,201 | 6,201 | 6,201 | 6,201 | 6,201 | 6,201 |
| Heterogeneity | # Ebola facilities | Difference predicted observed prevalence | Representation in government | # Ebola facilities | Difference predicted observed prevalence | Representation in government |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are clustered at the district level.

Table G.2: First-Stage Regressions Low Groups

| | Dependent Variable: Low Group × Ebola Prevalence | | | | | |
|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| High × simulated Ebola prevalence | -0.010 | 0.051 | 0.003 | -0.010 | 0.051 | 0.003 |
| | (0.034) | (0.084) | (0.013) | (0.034) | (0.084) | (0.013) |
| Low × simulated Ebola prevalence | 8.960*** | 2.915** | 1.099*** | 8.960*** | 2.915** | 1.099*** |
| | (1.530) | (1.171) | (0.176) | (1.530) | (1.171) | (0.176) |
| Country×year FE | yes | yes | yes | yes | yes | yes |
| District FE | yes | yes | yes | yes | yes | yes |
| Individual controls×year FE | yes | yes | yes | yes | yes | yes |
| Obs. | 6,201 | 6,201 | 6,201 | 6,201 | 6,201 | 6,201 |
| Heterogeneity | # Ebola facilities | Difference predicted observed prevalence | Representation in government | # Ebola facilities | Difference predicted observed prevalence | Representation in government |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are clustered at the district level.

Table G.3: First-Stage Regressions: Ebola Exposure and Vote Share Presidential Candidate

| | Ebola Prevalence | High Group × Ebola Prevalence | | |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Simulated Ebola prevalence | 1.670** | | | |
| | (0.664) | | | |
| High × simulated Ebola prevalence | | 1.327** | 0.962*** | 5.314*** |
| | | (0.505) | (0.039) | (1.041) |
| Low × simulated Ebola prevalence | | -0.082 | -0.367 | 0.111 |
| | | (0.976) | (0.228) | (0.090) |
| District FE | yes | yes | yes | yes |
| Country-year FE | yes | yes | yes | yes |
| Obs. | 122 | 122 | 122 | 122 |
| Heterogeneity | | # Ebola facilities | Difference predicted observed prevalence | Representation in government |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Regressions run at the district-year level. Standard errors are clustered at the district level.

Table G.4: First-Stage Regressions: Ebola Exposure and Vote Share Presidential Candidate

| | Ebola Prevalence | Low Group × Ebola Prevalence | | |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| High × simulated Ebola prevalence | | 0.019 | 0.188** | -0.256 |
| | | (0.058) | (0.081) | (0.208) |
| Low × simulated Ebola prevalence | | 5.746** | 4.879*** | 0.983*** |
| | | (2.197) | (1.090) | (0.086) |
| District FE | yes | yes | yes | yes |
| Country-year FE | yes | yes | yes | yes |
| Obs. | 122 | 122 | 122 | 122 |
| Heterogeneity | | # Ebola facilities | Difference predicted observed prevalence | Representation in government |

Note: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Regressions run at the district-year level. Standard errors are clustered at the district level.