

Working Paper No. 174

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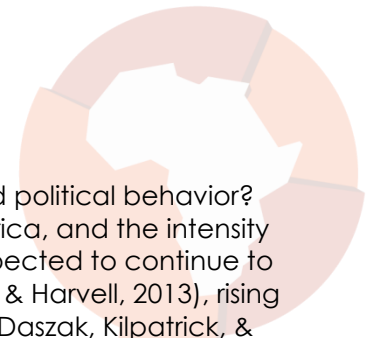
## **Abstract**

How do widespread public health crises affect political behavior? This article examines the impact of the 2014 West African Ebola outbreak on political participation in Sierra Leone. In contrast to the effects observed following conflict and natural disasters, I present evidence that exposure to Ebola virus disease substantially decreased political participation, measured in self-reported political activity using data from an Afrobarometer survey conducted near the end of the outbreak. To account for selection and endogeneity concerns, I undertake falsification and coefficient stability approaches in addition to controlling for levels of political activity in the 2012 national election. The negative effect seems driven in part by a reduction in interest in public affairs, highlighting the role of a psychological rather than resource-based mechanism in mediating the relationship between exposure to the disease and participation.

## **Acknowledgements**

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## I. Introduction

How do widespread public health crises affect individual attitudes and political behavior? Infectious diseases are ubiquitous in many countries in sub-Saharan Africa, and the intensity and frequency of outbreaks are increasing (Smith et al., 2014) and expected to continue to increase in the face of climate change (Altizer, Ostfeld, Johnson, Kutz, & Harvell, 2013), rising antibacterial resistance (Jones et al., 2008), and deforestation (Wolfe, Daszak, Kilpatrick, & Burke, 2005). In the case of Ebola virus disease (EVD), these outbreaks are becoming more deadly (Diehl et al., 2016). While the negative consequences of persistent disease burden on economic outcomes is well known, as in the case of malaria (Sachs & Malaney, 2002) or HIV/AIDS (Whiteside, 2002), the consequences of disease crises on political development is less clear. In this article, I explore the relationship between Ebola exposure in the West African Ebola outbreak and political participation using individual responses from Afrobarometer Round 6 (2015) survey data in Sierra Leone.

This work joins a growing body of literature examining the consequences of exposure to large-scale crises and traumatic events on political participation, collective action, cooperation, and a variety of prosocial preferences. The majority of this literature identifies a consistent, positive, and arguably causal link between the two. In a recent overview and meta-analysis of post-conflict scenarios, Bauer et al., (2016) find that individuals and communities more affected by wartime exposure to violence consistently exhibit higher levels of cooperation and altruism. These results hold across a variety of settings, whether the outcome measures come from survey data as in the case of Bellows and Miguel (2009) or from experimental games as in Voors et al. (2012), and across varying types of violence. Blattman (2009) provides evidence that this increase in prosociality can sometimes be observed in its effects on political participation.

Similar effects are also observed in the aftermath of natural disasters. Rodriguez, Trainor, & Quarantelli (2006) report an increase in prosociality and coordination in Louisiana in the weeks following Hurricane Katrina in 2005. Blocker, Rochford, & Sherkat (1991) identify an increase in political participation, in the form of local collective action, petitions, and protest behavior, following a large flood. Similarly, Cassar, Healy, & von Kessler (2017) find that villages affected to a greater extent by a tsunami in Thailand were more trusting than those less affected, and Fair, Kuhn, Malhotra, & Shapiro (2017) identify an increase in political knowledge and turnout as a result of the 2010-2011 floods in Pakistan.

While there is, to my knowledge, no comparable evidence on the impact of disease-related disasters, the evidence from the psychological literature on disease exposure would preliminarily suggest the opposite effect. Murray and Schaller (2010) and Schaller and Murray (2008) provide cross-country evidence that disease burden decreases extraversion and openness. In the lab, Mortensen, Becker, Ackerman, Neuberg, & Kenrick (2010) demonstrate that priming individuals with images of germs and information on the spread of disease also decreases extraversion and social interaction. Together, these articles provide evidence for the behavioral immune system – behavioral responses developed over time to mitigate exposure to and costs related to harmful pathogens (Hart, 1990).

In line with these theories, and in contrast to the effects observed following civil war or natural disasters, I observe that increased exposure to Ebola decreases political participation in Sierra Leone. I present preliminary evidence that this change in participation is largely driven by a decrease in political engagement, or interest in public affairs, rather than a reduction of capacity through economic channels – though Ebola does appear to increase economic insecurity.

## II. The legacy of the West African Ebola outbreak

The outbreak began in Guéckédou Prefecture, Guinea, in December 2013. Before its final containment in mid-2016, more than 28,616 individuals from 10 countries would be infected with Ebola virus. Of these reported cases, the majority occurred in three countries: Liberia,



Guinea, and Sierra Leone (World Health Organization, 2016). Nearly half (14,000) occurred in Sierra Leone. Accompanying this widespread loss of life was substantial economic disruption, which included reduced agricultural yields, diminished tourism, and stagnant foreign investment (Bowles, Hjort, Melvin, & Werker, 2016; Thomas et al., 2015).

Underlying these gross macroeconomic consequences are the traumatic experiences of victims, survivors, communities, and health workers. The immediate psychological costs of these experiences have been relatively well documented. In a small sample of survivors, Hugo et al. (2015) find that each of the 74 respondents had lost a family member to Ebola, with a majority witnessing their deaths. A full 20% reported "clinically important post-traumatic reactions between three and four weeks post discharge." Yadav and Rawal (2015) also highlight the challenges of survivors in overcoming these experiences, describing survivors as being in "a state of fear, grief, stress, and shame" while the outbreak was still ongoing.

Of course, these reactions were not limited to survivors and their families. Health-care workers, primary caregivers, and other members of affected communities have also been identified as secondary victims of the crisis (Reardon, 2015). Van Bortel et al. (2016) expand beyond those immediately exposed – survivors, contacts, and carers – to identify the potential for Ebola to disrupt local communities through the deaths of community leaders, disruptions in the local economy, community fracturing, and the disruption of social and health services, each of which could have consequences for psychosocial health over the long term.

The international community was cognizant of these potential challenges. Beyond stemming the outbreak, there was consistent concern among international and domestic health researchers and practitioners surrounding the potential mental health challenges that would linger after transmission of the virus had been stopped (Shultz, Baingana, & Neria, 2015; Boscarino & Adams, 2015). By late 2015, the World Bank had already set aside more than \$3 million to provide psychosocial support services in Liberia alone (Reardon, 2015).

### III. Literature review and conceptual framework

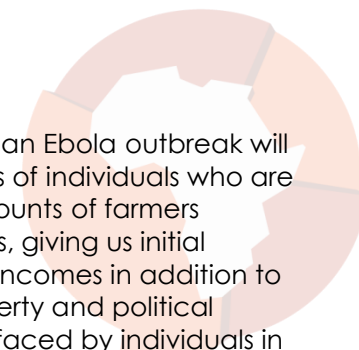
Through which mechanisms should we expect exposure to Ebola to have an impact on involvement in civic affairs? I begin by briefly reviewing the literature on the determinants of political participation, pulling from examples in the West and in less developed countries. I then contrast the potential effects of Ebola or other epidemiological shocks to those experienced during civil conflict or natural disasters, highlighting similarities and differences between the three. Finally, I look to the psychology and public health literatures to identify disease-specific pathways that could also influence political participation.

#### *Crises and individual determinants of political participation*

What determines political participation at the individual level?<sup>1</sup> Seminal work on participation in the United States by Brady, Verba, & Schlozman (1995) emphasizes three main dynamics that could diminish an individual's participation in politics: "because they can't, because they don't want to, or because nobody asked" (p.271). The first emphasizes the role of resources in the form of time, money, and social capital, arguing that individuals with fewer of these resources will be less likely to participate in political affairs and that the distribution of these resources within individuals will impact the manner by which they participate. The second is linked to psychological interest or engagement in politics, while the third highlights the role of social networks and community-level factors.

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<sup>1</sup> Note that while much of the literature uses the terms "political participation" and "political engagement" interchangeably, I distinguish between the two here. Conceptually, I refer to *participation* as actual behaviors (e.g. voting, attending community meetings) and use *engagement* to refer to self-reported interest in political and public affairs.



Clearly, an epidemiological disaster of the scale seen in the West African Ebola outbreak will likely have economic implications and alter the resource endowments of individuals who are directly and indirectly affected by the disease. Indeed, there are accounts of farmers abandoning fields and others leaving employment to escape the crisis, giving us initial reason to believe the disaster would have some impact on individual incomes in addition to country-wide changes in the economy (BBC, 2014). Early work on poverty and political participation by Huntington & Nelson (1976) highlights the challenges faced by individuals in poverty in engaging in public affairs. Other resource-based factors are likely unaffected by Ebola, at least in the short term. For example, education appears to be a driver of civic participation in many contexts (Galston, 2004; Kam & Palmer, 2008; Berinsky & Lenz, 2011). However, levels of education among the individuals surveyed here are unlikely to have changed in the short time frame between the outbreak and survey response.<sup>2</sup> This leads us to our first two hypotheses: That exposure to Ebola will increase economic insecurity and that economic insecurity will decrease political participation.

Additionally, the trauma of exposure to extreme illness and death are likely to elicit psychological reactions. One intuitive effect is that exposure to trauma would lead to an increase in post-traumatic stress disorder (PTSD) and/or depression. This dynamic has been documented in adolescents following Hurricane Mitch in Nicaragua (Goenjian et al., 2000) and as a result of the negative health consequences of disease, as is observed in survivors of Legionnaire's disease (Lettinga et al., 2002). Moreover, Ojeda (2015) demonstrates that, in the United States, depression is associated with diminished political participation.

However, other studies also suggest that trauma, such as the loss of a loved one or other challenging life events, can result in positive outcomes such as optimism and increased extraversion, in a theory dubbed "post-traumatic growth" (Tedeschi & Calhoun, 2004). The literature on conflict and disasters finds moderate support for this theory when looking at prosociality in general (Bauer et al., 2016). Whether this increase in prosociality translates into parallel shifts in political participation appears to be context-specific. For instance, Blattman (2009), exploiting quasi-random abduction into violent groups, finds strong evidence that ex-combatants have higher levels of political involvement than non-combatant peers. Similarly, Bateson (2012) finds that crime victimization at the individual level leads to increased political participation across numerous contexts. However, other studies that also identify an increase in prosociality as a result of violence find little evidence of changes in political behavior (Voors et al., 2012; Cassar et al., 2017).

A parallel literature identifies similar effects from natural disasters such as hurricanes (Rodriguez et al., 2006), floods (Blocker et al., 1991; Fair et al., 2017), tsunamis (Cassar et al., 2017), and earthquakes (Boittin, Mo, & Utych, 2017). The implication of this theory of post-traumatic growth is that to the extent that exposure to Ebola is a comparable trauma to wartime exposure to violence or natural disasters, we should observe an increase in prosociality as proxied by political engagement.

A third, related effect that has been observed is the tendency of individuals to rally around some political institution when facing foreign-policy crises or militarized interstate disputes. Studies considering American foreign policy crises find modest increases in presidential approval following crises when there is major coverage in leading news outlets (Oneal & Bryan, 1995; Baker & Oneal, 2001). While it would be difficult to observe a country-wide shift (as opposed to individual-level variance) in participation with the cross-sectional data employed here, this effect has also been documented following the 2015 earthquake in Nepal. Boittin, et al. (2017) exploit differences in public opinion surveys conducted immediately before and after the earthquake and find that respondents report higher levels of support for political institutions after the disaster.

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<sup>2</sup> Although it would not be surprising to observe changes in educational choices of individuals moving forward.





Together, these dynamics – depressive reactions, post-traumatic growth, and “rally ‘round the flag” – suggest divergent hypotheses, namely: Ebola will decrease political engagement through depressive reactions or Ebola will increase political engagement through post-traumatic growth or a “rally ‘round the flag” effect. The implications of these hypotheses also result in varying predictions for the net effect of Ebola on political participation. If we expect political engagement to be strongly and positively correlated with participation, depressive reactions would yield a reduction in participation, while post-traumatic growth would increase participation.

### *Disease-specific pathways*

Of course, wartime violence, natural disasters, and the Ebola outbreak differ along a number of potentially salient dimensions. Importantly, while civil-war violence may be targeted toward a particular group of people by another group of people, and exposure to natural disasters is largely idiosyncratic, Ebola virus disease is highly contagious, easily transferred from person to person, and lethal. Moreover, Ebola is also characterized by higher information costs regarding identification of a potential threat. Whereas an individual may be able to make a judgment of whether or not a stranger is a member of an opposition group in civil war, for instance, the same cannot be said of hosting EVD. The implications of these costs are explored in the psychological literature on the behavioral immune system. At the country level, individuals have lower levels of extraversion and openness in places with greater historical disease burden (Schaller & Murray, 2008). Lab experiments artificially inducing fear of disease transmission also find that individuals primed with information on germs and infectious disease are less extraverted, less open, and less agreeable compared to non-primed participants (Mortensen et al., 2010). Moreover, the effects of this priming increased with a participant's self-reported perception of vulnerability to disease. The observations in each of these papers are consistent with a theory of a behavioral immune system (Schaller & Park, 2011), which suggests that at the individual level, avoidance of interpersonal interactions can be used as a strategy to limit the physiological cost of infection.

On political outcomes, there is also evidence that individuals who experience higher levels of disgust when primed with pathogen cues or who are avoidant of situations with a high risk of infection are more likely to hold conservative views (Terrizzi, Shook, & McDaniel, 2013). The literature poses two distinct hypotheses for this effect, which Tybur et al. (2016) summarize and test empirically. The first, based on intragroup dynamics, argues that social norms have developed over time to diminish the threat posed by local pathogens. To avoid the costs of contracting a pathogen, individuals naturally respond to these threats by strengthening adherence to those cultural norms.<sup>3</sup> The second argues that individuals from outgroups pose a larger threat to one's health because they are more likely to harbor pathogens to which one's ingroup has not yet developed immunity. Thus, to avoid exposure to communicable diseases, the exposure-minimizing individual would adopt practices that would limit interaction with members of outgroups. Broadly, this ideology takes the form of social dominance orientation, a sub-construct of social conservatism.<sup>4</sup> Tybur et al. (2016) distinguish between these two mechanisms, finding strong support for the former and little for the latter.

Both of the measures discussed in these works gauge political attitudes, not behavior. As I discuss below, the measure of political participation used in this analysis is constructed from

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<sup>3</sup> In the case of Sierra Leone, several norms (such as the consumption of bushmeat and burial practices) encourage physical contact with the dead. However, the intuition for this argument still holds if it is an unconscious, cognitive response developed evolutionarily.

<sup>4</sup> While this effect might hold for many diseases in general, it is unclear whether the same effect would be observed in the case of the Ebola outbreak, where an individual would be more likely to contract the virus from existing social contacts rather than strangers.



political actions taken by an individual following the outbreak of Ebola. These activities will usually entail interaction with members of one's community in addition to strangers. There is relatively little research on the impact of exposure to disease on political behavior,<sup>5</sup> but an intuitive argument would be that pathogen-avoiding behavior would decrease social interaction of all forms. In the case of Ebola, which is characterized by high information costs and physical costs (death), the potential negative effect of Ebola on political engagement is quite large.

From the literature, we observe several pathways through which epidemiological disasters might impact political participation. Of these, two predict positive effects. The theory of post-traumatic growth would hypothesize that exposure to the trauma of Ebola would lead to an increase in prosociality and, through that mechanism, an increase in political participation. Similarly, the "rally 'round the flag" effects observed in Nepal and elsewhere hypothesize an increase in engagement. However, taking behavioral responses designed to mitigate exposure to pathogens or resource-based effects into account, we could expect a marked decrease in the level of political engagement by those who have been most affected by Ebola.

#### IV. Data overview

The individual-level data used in the analysis are from the Afrobarometer Round 6 (2015) survey in Sierra Leone. Responses were collected between 22 May and 10 June, 2015, several months after the peak of the outbreak, though several reported incidents of the disease were recorded after this time (Shultz, Espinel, Espinola, & Rechkemmer, 2016). This is a nationally representative survey of 1,191 individuals and covers 14 districts, 90 chiefdoms, and 150 local enumeration areas, which are typically a single town or neighborhood.

The Round 6 survey asks several questions related to engagement. These include interest in public affairs; frequency of discussing politics; membership in community and religious groups; willingness to attend community meetings, join together with others to raise an issue, attend a demonstration or protest march, and refuse to pay tax; and contact with members of the community, including government officials at local levels. The survey also asks a number of questions related to political participation in the 2012 national elections, which I use as outcome measures in a falsification test. A full list of questions can be found in the Sierra Leone Afrobarometer Round 6 (2015) questionnaire.

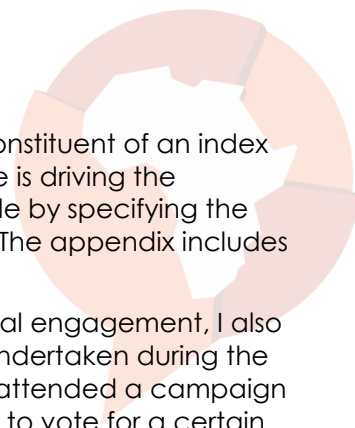
The survey also asks respondents six questions about their exposure to Ebola. For a majority of respondents, we know whether or not a family member or close friend was either infected or killed by EVD and how it impacted their lives across a number of domains. These include whether, due to Ebola, the respondent was unable to attend school, to work or earn income, to attend social gatherings, or to get medical care. Average responses by chiefdom for having a close friend or relative infected with Ebola is positively correlated with the number of laboratory-confirmed and suspected cases of EVD by chiefdom.<sup>6</sup>

"Political participation" is a broad and multifaceted construct. For the purposes of this analysis, I model participation as a latent variable, for which we can generate a proxy by measuring the covariation that is shared among the survey items conceptually linked to participation (attendance of community meetings, membership in community and religious groups, and reaching out to local and national politicians and political groups). To generate this measure, I use principal components analysis and employ the first principal component for the group of variables as an index. I conduct a similar analysis to generate an index of Ebola exposure using the six items described above. This index is used as my primary treatment variable. For the main individual-level analysis, I also present results that replace –

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<sup>5</sup> Exposure to disease being distinct from health generally, where there is a body of work that considers the consequences of health on participation. See Ojeda and Pacheco (2017) for an overview.

<sup>6</sup> This is based on chiefdom-level incidence data pulled from Fang et al. (2016) replication data.



in the case of outcome indices – the dependent variable with each constituent of an index in turn to ascertain whether, and to what extent, an individual response is driving the observed relationship. I perform similar checks on the treatment variable by specifying the same regression with each of the six Ebola questions as the treatment. The appendix includes the factor loadings for each index in tables A.2-A.5.

For use in the falsification exercise and to control for pre-disaster political engagement, I also construct a similar index using responses to questions about activities undertaken during the 2012 national elections. These include whether the respondent voted, attended a campaign rally, met with a candidate or campaign staff, tried to persuade others to vote for a certain presidential or legislative candidate or political party, or worked for a candidate or party. In each of the primary regressions, I include a large number of control variables to account for potential confounding factors. The majority of these are pre-treatment measures, such as age, gender, ethnicity, and language, which are, for practical purposes, time invariant. Other controls, such as occupation, material of housing, and education, are theoretically time variant, though it is unlikely that these responses would change very much in the short period of time between the end of the outbreak and collection of survey responses.

The survey enumerators also collect a number of enumeration area (EA) characteristics. These include measures such as whether the EA has access to the electricity grid, has piped water and sewerage, and receives cell-phone service, and whether banks, police stations, schools, and post offices exist within the sampling area. For the purposes of this analysis, these variables are combined using the same principal components method I describe above. The motivation here is to reduce the degrees of freedom in the estimations while capturing a potential important confounder, local capacity.

## V. Empirics

My empirical strategy considers the proposed relationships in three steps. First, I employ naive OLS regressions to identify the correlation between Ebola exposure and the outcomes of interest. Second, I employ three approaches – falsification, controlling for previous political activity, and coefficient stability – to present suggestive evidence that this relationship is causal and not an artifact of pre-disaster characteristics of individuals and political geography. Finally, I undertake a simple mediation exercise to disentangle the relationship between the identified impacts of Ebola and theoretical expectations.

I begin with the OLS regressions testing whether individuals with greater exposure to Ebola exhibit higher levels of political participation as estimated by equation (1):

$$Y_i = \beta_0 + \beta_1 ebola\_exp_i + X'_i \beta + X'_d \beta + \epsilon_i \quad (1)$$

where  $Y_i$  represents the index of political participation;  $ebola\_exp_i$  is the Ebola exposure index;  $X'_i$  is a vector of individual socio-demographic controls including age, age-squared, gender, education, and a series of occupation, religion, sector (of employment), and ethnicity dummies; and  $X'_d$  is a vector of enumeration area characteristics that measure local state capacity. The OLS regressions presented here are survey-weighted to account for sampling design and employ robust standard errors, clustered by enumeration area.<sup>7</sup>

As can be seen in Table 1, we find a negative correlation between Ebola exposure and the measure of political participation – a finding at odds with the literature on the effects of civil-war exposure and natural disasters. This relationship is observed in the raw correlation, and

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<sup>7</sup> Regressions without survey weights and alternative standard errors are also presented in the appendix (Table C.2), though the results are qualitatively similar throughout; directionality and magnitude are consistent to approximately two decimal points in most regressions.



when controlling for individual characteristics, and is robust to the inclusion of PSU fixed effects.<sup>8</sup>

**Table 1: Ebola exposure and political engagement**

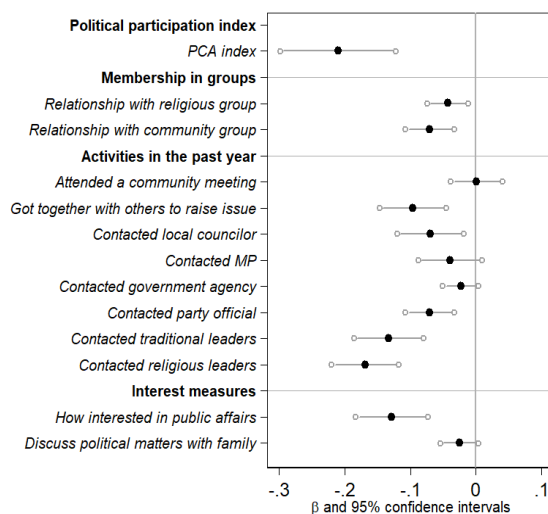
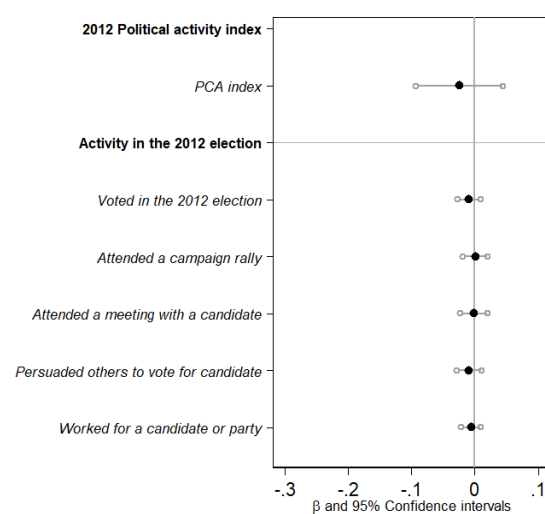
	Political participation index			
	(1)	(2)	(3)	(4)
Ebola exposure index	-0.2505*** (0.046)		-0.2097*** (0.045)	-0.2044*** (0.041)
Age		0.0961*** (0.025)	0.1043*** (0.025)	0.1298*** (0.026)
Age <sup>2</sup>		-0.0006* (0.000)	-0.0007* (0.000)	-0.0009** (0.000)
Sex (Male = 1)		0.4548** (0.147)	0.4892** (0.146)	0.4491** (0.151)
Education		0.0717* (0.035)	0.0820* (0.037)	0.1263** (0.043)
R <sup>2</sup>	0.053	0.200	0.231	0.380
Occupation FE		Y	Y	Y
Sector FE		Y	Y	Y
Religion FE		Y	Y	Y
Language FE		Y	Y	Y
Ethnicity FE				150
Observations	993	993	993	993

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ . Robust standard errors in parentheses.

Note: This table displays the observed relationship between the first principal component index of Ebola exposure on an index of political engagement. A higher value indicates being more politically engaged. Column (1) is the raw correlation between these two measures. Column (2) is a multivariate regression of controls that does not include Ebola exposure. Column (3), our baseline specification, is the fully controlled OLS regression estimating the relationship between Ebola exposure and political engagement. PSU fixed effects are added in Column (4). Controls include the vector of individual characteristics and PSU controls and fixed effects as defined in eq. (1). Coefficients are survey-weighted with robust standard errors clustered at the PSU level.

To test for robustness to index composition, Figure 1a presents the Ebola exposure coefficient for the same regression specification with the outcome variable in each case replaced with a subcomponent of the political engagement index. In all but one case, attendance of community meetings, the observed relationship is negative. Of these, only two fail to meet statistical significance by conventional thresholds, and it should be noted that each of these likely experience some degree of pinning as the mean value for each of these is very low (very few respondents contacted an MP or government agency regardless of Ebola exposure).

<sup>8</sup> It might be concerning that the inclusion of PSU fixed effects does not do more to diminish the coefficient, given that one might assume that levels of Ebola exposure are likely to be highly collinear within PSU. However, even within villages there was significant variation in exposure to the disease.

**Figure 1: Ebola exposure and political participation subcomponents****(a) Political participation index****(b) 2012 political activity index**

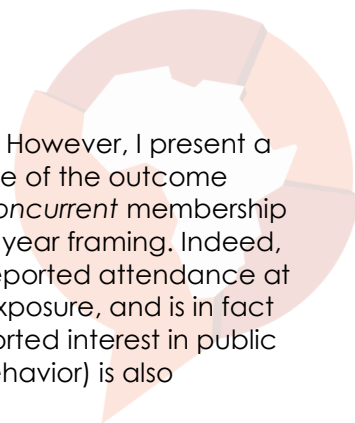
Note: Figure 1a plots the coefficients and 95% confidence intervals for Ebola Exposure taken from specifications following eq. (1) with the index of political participation and each individual component of that index as the dependent variable. Figure 1b plots the coefficients and 95% confidence intervals for Ebola exposure from eq. (2) with the index of political activity in the 2012 national election and each individual component as the dependent variable.

An additional concern is that the results are being driven by the composition of the treatment variable, Ebola exposure. Appendix Table B.1 presents the results of estimations that replace our main independent variable with each of the components of the index. Results are negative and significant for four of the six components. However, for the two components that we would intuitively associate with trauma (having a close friend or relative who died or was infected with Ebola), the association is positive but insignificant, though standard errors are much larger.

This highlights an additional divergence from the literature on post-traumatic growth.<sup>9</sup> As an additional robustness check, I regress political participation against the ecological level of Ebola, testing whether individuals in chiefdoms with higher numbers of confirmed and suspected Ebola cases are less politically active than those with fewer. Appendix Table D.4 presents these specifications with robust, clustered, and survey-weighted standard errors. However, the focus of this paper is on the individual-level effects of Ebola. As there is substantial intra-community variation in level of exposure, the survey measures remain our primary treatment variables.

It is perhaps unsurprising that having one's social and economic life disrupted by a viral outbreak – or any other disaster – would have a negative effect on political and community engagement in the short term, especially as components in both the treatment and

<sup>9</sup> Note that Table D.3 in the appendix presents the same specifications with application to Liberia as a check on the external validity of the findings presented here. Using the Liberian data, it appears that, in contrast to the findings here, having a friend or family member who died or was infected with Ebola is negatively and significantly correlated with political participation, whereas being unable to attend gatherings or receive medical care due to Ebola are negative, but not significant. However, there are substantial differences in the manner in which domestic and international communities responded to the outbreak in the two countries. Assessing the variation in impact between countries is beyond the scope of this article.



outcome indices ostensibly measure disruption in social participation.<sup>10</sup> However, I present a few arguments that suggest this is not driving the results. First, while some of the outcome variables are framed as “in the past year,” several measures include *concurrent* membership in religious and community groups, and should be less affected by the year framing. Indeed, the opposite appears to be true; out of all of our outcome variables, reported attendance at community meetings has the lowest observed correlation with Ebola exposure, and is in fact (weakly) positive. And, as will be discussed later, a measure of self-reported interest in public affairs (excluded from the index as it measures attitudes rather than behavior) is also negatively and significantly correlated with Ebola exposure.

### Assessing causality

The associational evidence presented in the preceding section is consistent with the hypotheses posed in Section III. However, to consider these relationships causal, selection into Ebola exposure must be exogenous, or conditionally unconfounded by unobservable characteristics. There are a number of reasons one might suspect that involvement in local political affairs could be driving exposure to Ebola. First, knowledge of a family member or close friend who is infected will be positively correlated with the size of a respondent's social network. In essence, as the set of people an individual is close friends with increases, mechanically, so does the likelihood of knowing someone who contracted or died from Ebola. Second, in many cases, local organizations responded to the crisis on their own or supported humanitarian efforts. Membership in these organizations could therefore increase exposure to Ebola. Both of these pathways would generate a positive bias, with selection into Ebola exposure causing an increase in the observed relationships between both engagement and tolerance. Alternatively, one could also argue that communities with effective local institutions would be better able to prevent the spread of Ebola into their communities, driving the observed correlation between Ebola exposure and political participation downward. This is more consistent with the results observed in the OLS specifications and conforms in part to an evolutionary model.<sup>11</sup>

### Falsification and controlling for previous political activity

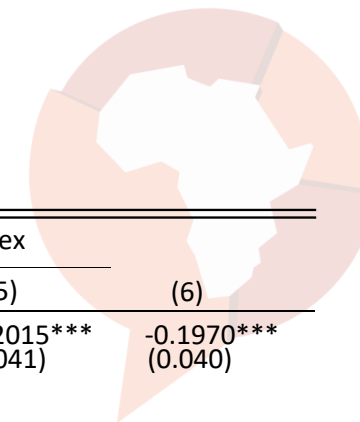
As a falsification exercise, I conduct a similar analysis with an index of political participation taken from an individual's actions in the 2012 national elections, as in equation (2), where  $Y_{i,t-1}^*$  is the index of political activity in 2012.

$$Y_{i,t-1}^* = \beta_0 + \beta_1 \text{ebola\_exp}_i + X_i' \beta + X_d' \beta + \epsilon_i \quad (2)$$

Figure 1b and Table B.3 in the appendix present the coefficients for the relationship between Ebola exposure and the 2012 political participation index and its constituent parts – whether or not they attended a rally, attended a meeting with a candidate or campaign, attempted

<sup>10</sup> Of course, we have relatively little literature to which we can compare; much of the existing literature examines outcomes several years or even decades after the initial shock. Bauer et al. (2016) find that the effects of conflict exposure on measures of altruism appear to be increasing over time, but their meta-analysis includes only two studies that are within two years of the initial conflict.

<sup>11</sup> I say “in part” because there are several evolutionary pathways through which a shock like Ebola would change the observed strength of local institutions. The first is that communities with stronger institutions *ex-ante* have a higher “fitness,” or capacity to endure a shock (selection). The second is that communities that were better able to adapt to shocks, by developing stronger institutions, would be more likely to persist and be observed in the sample. This second, adaptive mechanism would identify the reverse effect (exposure to Ebola would be correlated with better local institutions) assuming that the selection mechanism holds. However, it is unclear if these community-level dynamics would be captured at the individual level, and the capacity to adapt is likely also a function of baseline community institutions.

**Table 2: OLS controlling for political activity in 2012**

	Dependent variable: Political participation index					
	(1)	(2)	(3)	(4)	(5)	(6)
Ebola exposure index	-0.1980*** (0.041)	-0.2112*** (0.046)	-0.2088*** (0.046)	-0.2046*** (0.042)	-0.2015*** (0.041)	-0.1970*** (0.040)
Pre-Ebola political activity index	0.4797*** (0.052)					
2012: Voted in election		0.3222 (0.188)	0.3091 (0.186)	0.1784 (0.182)	0.1561 (0.181)	0.1262 (0.184)
2012: Attended campaign rally			0.8524*** (0.156)	0.3924* (0.160)	0.2948 (0.160)	0.1557 (0.160)
2012: Attended meeting with candidate				1.0717*** (0.158)	0.9527*** (0.156)	0.7587*** (0.169)
2012: Persuaded others					0.3887* (0.155)	0.2355 (0.154)
2012: Worked for candidate or party						0.9580*** (0.196)
$R^2$	0.343	0.237	0.275	0.323	0.328	0.353
Individual controls	Y	Y	Y	Y	Y	Y
Occupation FE	Y	Y	Y	Y	Y	Y
Sector FE	Y	Y	Y	Y	Y	Y
Religion FE	Y	Y	Y	Y	Y	Y
Language FE	Y	Y	Y	Y	Y	Y
Ethnicity FE	Y	Y	Y	Y	Y	Y
Observations	988	988	988	988	988	988

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ . Robust standard errors in parentheses.

Note: This table displays the observed relationship between the first principal component index of Ebola exposure on the contemporary (2016) political engagement index. These regressions each include the vector of controls as defined in eq. (1) and additional controls on political participation in the most national recent election (2012). Column (1) controls for an index of these measures. Columns (2) through (6) add each measure sequentially. Also note that five respondents have missing values for these survey items. For comparison, an OLS estimation analogous to that presented in Column 3 of Table 1 among the subgroup of 980 yields comparable results, estimating the Ebola exposure coefficient to be -0.203 (on the contemporary political engagement index) and a standard error of 0.035. The largest difference between coefficients and the standard regression is 0.0239 or one one-hundredth the standard deviation of the outcome index.

to persuade others, or worked for a candidate or political party. For each of these measures, Ebola exposure has a relatively precisely estimated null effect. While these by necessity measure somewhat different outcomes, it seems likely that political activity of one variety during the election season – barring some mediating event in the intervening period – would be somewhat correlated with political engagement along other domains later. The consistent null results in each case offer reassurance that the correlation we observe between Ebola exposure and post-outbreak political engagement is not simply statistical artifact.

Another way to approach this is to use the measure of political activity on the right-hand side of the estimating equation, as in equation (3).

$$Y_i = \beta_0 + \beta_1 ebola\_exp_i + \beta_2 Y_{i,t-1}^* + X_i' \beta + X_d' \beta + \epsilon_i \quad (3)$$

The logic behind this approach is similar to that employed by lagged dependent variable estimators – that in the absence of some other, causal impact, the lag of a variable should account for a very large portion of the variation in the same measure in the following period. What we see in Table 2 is that this index is indeed very highly correlated with contemporaneous political participation and that the inclusion of this parameter dramatically improves the amount of variance in the outcome that we capture ( $R^2$ ) while moving the coefficient of Ebola exposure very little. What this again implies is that the negative effect of Ebola on political participation is unlikely to be the result of imbalanced exposure to Ebola on pre-disaster levels of participation.

### *Coefficient stability approaches*

To further test the sensitivity of these results to selection into Ebola exposure based on unobservable characteristics, I employ strategies developed in Altonji, Elder, & Taber (2005) and Oster (2016) and presented in González and Miguel (2015). The intuition presented in these articles is that we can get a sense of the potential selection into treatment from omitted variables based on the nature of selection on observed covariates. To operationalize this intuition, we first estimate the coefficient of Ebola exposure on our outcome of interest in an uncontrolled regression and the coefficient for fully controlled regressions, as in equation (1). Then, with the assumption that unobservable variables share the same level of explanatory power with observable characteristics, the following equation is a consistent estimator of the effect of Ebola on Y:

$$\hat{\beta} = \hat{\beta}^* - (\hat{\beta} - \hat{\beta}^*) \times \frac{R_{max} - R^*}{R^* - R} \quad (4)$$

This estimator, taken from González and Miguel (2015) and based on the work of Oster (2016), considers changes in the coefficient of interest from the uncontrolled regression ( $\hat{\beta}$ ) to the controlled regression ( $\hat{\beta}^*$ ) and the changes in R-squared from the same regressions ( $R^*$  and  $R$ ). The adjusted coefficient from this estimator ( $\hat{\beta}$ ) yields an upper (or lower) bound for an identified set of potential coefficients. The coefficient from the OLS regression acts as the other bound. If the estimated bounds exclude zero, this would suggest an effect robust to the exclusion of omitted variables. The difficulty in implementing this procedure is identifying an appropriate level of  $R_{max}$ , or the theoretical upper bound of explanatory power.

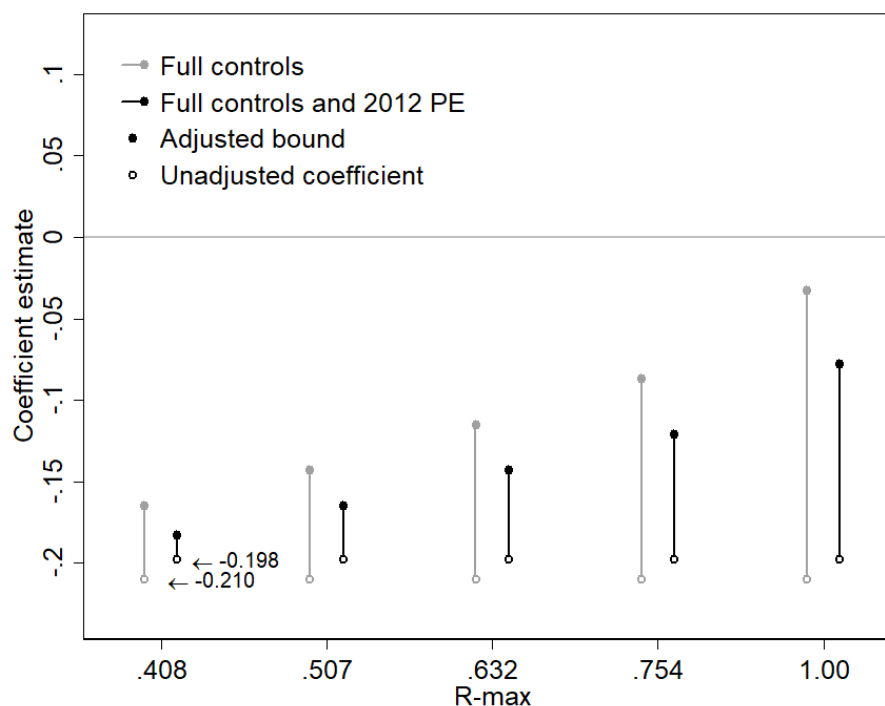
Panel A in Table 3 presents the results of this analysis using the specification in equation (1) as the control regression. Panel B replicates this analysis with the inclusion of the index of political activity in the 2012 election. The first two columns provide the coefficients, standard errors, and  $R^2$  values for these regressions, while the final three columns present stable bounds for the coefficient of Ebola exposure on political participation at varying levels of  $R^2$  derived using values of  $R_{max}$  from the methods suggested by Altonji et al. (2005) and Oster (2016) –  $2R^*$  and  $2.2R^*$  respectively. For these values, the directionality of all coefficients in the identified set for political participation remains the same. Naturally, the identified set for Panel B is smaller because of the increase in  $R^2$  relative to the difference in coefficients.



**Table 3: Selection based on unobservables**

	OLS		Stability bounds		
	$\beta^R$	$\beta^F$	Bellows & Miguel 2015	Oster 2016	Maximum
<i>Panel A: Full controls</i>					
Ebola exp.	-0.251*** (0.046)	-0.210*** (0.045)	[-0.169, -0.210]	[-0.146, -0.210]	[-0.033, -0.210]
$R^2$	0.053	0.231	.	.	.
$R_{max}$	.	.	0.408	0.507	1.000
<i>Panel B: Full controls and political activity in 2012</i>					
Ebola exp.	-0.251*** (0.046)	-0.198*** (0.041)	[-0.145, -0.198]	[-0.123, -0.198]	[-0.078, -0.198]
$R^2$	0.053	0.343	.	.	.
$R_{max}$	.	.	0.632	0.754	1.000

Note: The first two columns present the coefficients and standard errors for Ebola exposure, first in a regression with no controls, denoted  $\beta^R$ , and second with the full set of controls from equation (1). The following three columns present bounded estimates from  $\beta^*$  to  $\beta^{**}$  and the value used as the maximum achievable R-squared. Panel A uses the fully specified estimation from (1) to estimate  $\beta^F$  and the  $R^2$  value, while Panel B also includes the index of political engagement in the most recent national election.

**Figure 2: Coefficient stability bounds**

Note: Each pair of lines displays the impact of Ebola exposure on an index of political engagement under different levels of  $R_{max}$ , or “the maximum variation that can be explained in a regression of a dependent variable of interest. ...” (González & Miguel, 2015). An  $R_{max}$  of 1.00 assumes that all the variation in political participation would be able to be captured by survey measures. The first line of each pair uses  $R^2$  and  $\beta^*$  values from the specification in eq. (1). The second, darker line takes these values from a similar specification that also includes measures of political participation in the 2012 elections.

Even in the most conservative case, where we assume that we can perfectly capture the variance in political participation ( $R_{max}=1$ ), the set of coefficients is wholly below zero (Figure 2), though I note that this is itself an unrealistic assumption. The difficulties in collecting survey data in West Africa in terms of measurement error, sample sizes, geographic coverage, etc. increase statistical noise in the estimation of socioeconomic outcomes. As mentioned in González and Miguel (2015), McKenzie (2012) considers autocorrelation of many outcomes – including income, expenditures, math test scores, and language test scores – between two periods and across numerous countries and finds that they are “typically lower than 0.50 with many around 0.30.” The Altonji et al. and Oster methods suggest that a reasonable upper bound on  $R^2$  would be approximately 0.41 or 0.51, respectively, in the case of the basic controls, or 0.63 and 0.75 when also including pre-disaster political activity as a control. The bounds from these estimates, coupled with strong evidence of null effects in the falsification test, suggest that the relationships we observe here are indeed causal and not an artifact of bias from selection on unobservables.

### Additional robustness checks

**Evaluating response bias:** One concern that I discuss briefly in the previous section is response bias. If it is the case that individuals who are less likely to participate in civic affairs respond in a systematically different way to questions regarding exposure to Ebola – if they were deliberately overstating the degree to which Ebola affected their lives, for example – the estimates presented here would be biased. A simple way to evaluate the likelihood of this bias is to shift the main treatment variable (Ebola exposure) to the left-hand side of the equation. Table C.1 in the appendix presents two additional specifications that regress the vector of control variables against Ebola exposure. In each case, F-statistics are fairly low,<sup>12</sup> suggesting little evidence of systematic bias in responses.

**Weighting and standard error construction:** An additional concern is that the results presented above are sensitive to either the weighting we place on them or the manner by which standard errors are calculated. Table C.2 presents specifications analogous to Column 1 of Table 1 without survey weighting; with and without clustered standard errors at enumeration, chiefdom, and district levels; and using quantile regression evaluated at the median. Under no alternative weighting or standard error construction scheme would we change our inference.

**Multiple imputation:** All surveys include some level of missingness or non-response. To account for any potential impact this might have on the estimates, I also replicate the specifications in Table 1 on data that have been multiply imputed. These results are presented in appendix Table C.4 and do not indicate that this potential source of bias would change our inference.

**Distributional robustness:** To probe the robustness of the above findings to non-normality in the distribution of the outcome variable and potential outliers, I also present (in appendix Table C.5) the results of quantile regressions (Koenker & Bassett, 1978), evaluated at the 10<sup>th</sup>, 25<sup>th</sup>, 50<sup>th</sup> (median), 75<sup>th</sup>, and 90<sup>th</sup> percentiles. The observed relationship is negative and significant at all points in the outcome distribution, though larger at the higher end, suggesting exposure to Ebola had a larger negative effect on individuals who are generally more politically engaged.

**Over-controlling:** Achen (2005) argues against the inclusion of a large number of control variables in multivariate regressions in favour of more parsimonious specifications. Recently, Lenz and Sahn (2017) highlight that in many studies, statistical significance is achieved using covariate adjustment. To demonstrate that covariate manipulation is not driving the results here, Table C.6 presents the bivariate regression between political participation and Ebola exposure and sequentially adds additional covariates. In all specifications, the coefficient for our main treatment variable remains significant and between -0.22 and -0.17.

<sup>12</sup>  $F = 3.2$  for the baseline specification, and  $F = 2.2$  with the addition of PSU fixed effects.

## VI. Evaluating potential mechanisms

In the preceding section, I demonstrate a strong, negative relationship between Ebola exposure and political participation. Given the divergence between this finding and much of the literature on trauma, distinguishing the mechanism by which this particular type of crisis affects civic engagement becomes all the more important. In Section III, I highlight several potential pathways. Of these, three are both consistent with the observed net effect of Ebola exposure on participation and testable with the data at hand: resources (economic security), interest in public affairs, and support for democratic institutions.

To evaluate these mechanisms, I undertake a very simple mediation exercise testing 1) whether Ebola appears to influence the potential mediator  $M$ , as in equation (5), and 2) to what extent controlling for this variable mediates the relationship between Ebola exposure and political participation, as in equation (6).

$$M_i = \beta_0 + \beta_1 \text{ebola\_exp}_i + X_i' \beta + X_d' \beta + \epsilon_i \quad (5)$$

$$Y_i = \beta_0 + \beta_1 \text{ebola\_exp}_i + \beta_2 M_i + X_i' \beta + X_d' \beta + \epsilon_i \quad (6)$$

Ebola exposure appears to be conditionally correlated with each of the three mediators in the manners predicted by theory: Ebola appears to increase economic insecurity, to diminish satisfaction with the way democracy is performing in Sierra Leone, and to decrease interest in public affairs, or political engagement (Table 4).

**Table 4: Mediation analysis**

	Reference		Mediators ( $M$ )	
	OLS	Economic insecurity	Support for democracy	Political engagement
<i>Panel A: Political participation as DV; mediator as post-treatment control</i>				
Ebola exp.	-0.2097*** (0.045)	-0.1890*** (0.043)	-0.2015*** (0.044)	-0.1588*** (0.041)
$M$		-0.1250** (0.045)	0.2192 (0.167)	0.4046*** (0.064)
Individual controls	Y	Y	Y	Y
District controls	Y	Y	Y	Y
Observations	993	993	993	993
<i>Panel B: Mediator as outcome variable</i>				
Ebola exp.	-0.2097*** (0.045)	0.1657*** (0.036)	-0.0374*** (0.007)	-0.1257*** (0.028)
Individual controls	Y	Y	Y	Y
District controls	Y	Y	Y	Y
Observations	993	993	993	993

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ . Robust standard errors in parentheses.

Note: This table presents the results of a simple mediation exercise. Panel A displays coefficients for Ebola exposure with the index of political engagement as the dependent variable as well as the coefficient for the potential mediator  $M$  – economic insecurity, support for democracy as it's practiced in Sierra Leone, and support for the incumbent president in the second, third, and fourth columns, respectively. Panel B displays the coefficient of Ebola exposure in specifications with each mediator as the dependent variable. Note: Six observations for economic insecurity are imputed, as are 24 for political engagement. The same specifications for the non-imputed subsamples yield comparable results, which are available by request.

However, controlling for these post-treatment indicators, we observe different results. While Ebola is associated with increased economic insecurity, this does not appear to greatly reduce the observed relationship between Ebola and political participation. This is also true for support for democracy – the coefficient for Ebola reduces by less than .01 compared to the baseline specification. In contrast, adding in the measure of political interest reduces the coefficient of Ebola exposure by approximately 25%, offering suggestive evidence that of the three testable pathways, a decrease in interest in public affairs appears to be driving some of the relationship between Ebola and participation, though I note that there is still a large portion of the total effect for which we are unable to account.

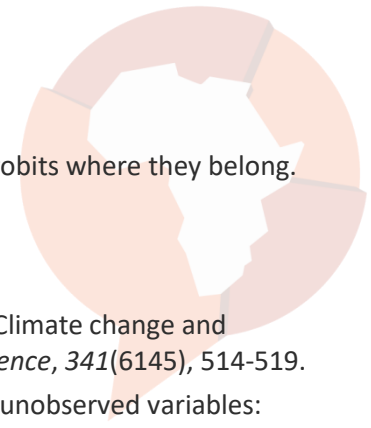
This result, of course, still begs the question, Why does Ebola exposure lead to a decrease in *interest* in politics? With the data at hand, it is difficult to arrive at a concrete answer. Turning to theory, however, there are two main mechanisms that would be consistent with this result: 1) depressive reactions & PTSD, as in Goenjian et al. (2000) and Ojeda (2015), and 2) pathogen-avoidant responses theorized as the behavioral immune system (Schaller & Park, 2011).

## VII. Conclusion

The negative impacts of disease burden on economic development are well documented. In the preceding sections, I have demonstrated that, in the case of the 2014/2015 West African Ebola outbreak, epidemiological disasters can also have detrimental effects on political development. Individual responses from approximately 1,000 Afrobarometer (2015) survey participants suggest that citizens with greater exposure to Ebola are significantly less politically engaged – less likely to participate in community groups, undertake collective action with fellow citizens, and reach out to community and political leaders. These results are robust to extensive individual and geographic controls, falsification, and selection from unobservables.

The negative effect seems, in part, driven by a reduction in interest in public affairs, highlighting the role of a psychological rather than resource-based mechanism in mediating the relationship between exposure to the disease and participation. This result is largely consistent with potential mechanisms highlighted in the literature: depressive/PTSD reactions and pathogen-avoidant behavioral responses, though we cannot differentiate between them with the data at hand. While these hypotheses are not mutually exclusive, this finding is most consistent with the many accounts of the outbreak that highlight the lingering consequences of the disaster on the mental and social health of survivors, families, health-care workers, and members of communities most affected (Yadav & Rawal, 2015; Shultz et al., 2015; Reardon, 2015; van Bortel et al., 2016; Boscarino & Adams, 2015; Hugo et al., 2015). While Ebola exposure is negatively correlated with economic insecurity and satisfaction with democracy, these proposed alternative mechanisms do not mediate the relationship between exposure to the disease and participation.

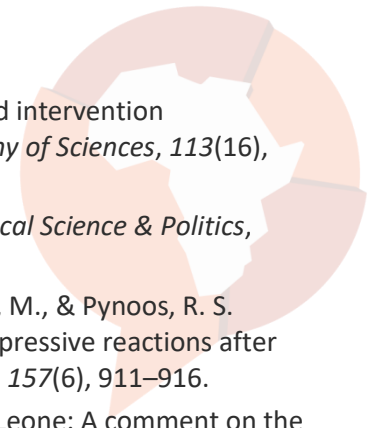
Regardless of mechanism, the observed effect of the Ebola outbreak on political engagement demonstrates that the impacts of disease can go well beyond immediate health consequences. In weak institutional environments, epidemiological disasters would appear to have a much stronger potential to undermine social capital formation and advances toward democratization than either natural disasters or conflict.



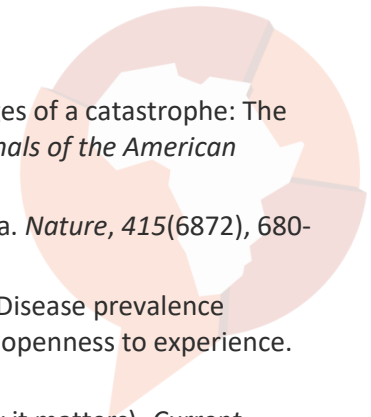
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## Appendix

This appendix provides supplementary information to *Disease, disaster, and disengagement: Ebola and political participation in Sierra Leone*. Summary statistics and principal components analysis for index construction can be found in Section A. Section B presents full regressions tables for the main effects presented in the article. Section C presents the analysis for further robustness checks referred to in the paper, and Section D portrays additional relationships that we can evaluate using the data, including the effect of Ebola exposure on measures of economic insecurity or “lived poverty.”

### A. Summary statistics and index construction

**Table A.1: Summary statistics**

	Mean	Std. dev.	Min	Max	N
<i>Panel A: Control variables</i>					
Age	39.71	14	18	99	1185
Sex (Male = 1)	0.49	.5	0	1	1191
Education	3.49	2.5	1	10	1188
Religiosity	5.46	1.8	1	7	1155
State capacity	-0.00	2	-3.9	4.2	1191
<i>Panel B: Political engagement outcomes</i>					
Political engagement index	-0.00	2	-3.4	6.5	1145
How int. in public affairs?	1.55	1.2	0	3	1146
Discuss public affairs?	0.80	.73	0	2	1139
Rel. with religious group	2.44	.89	1	4	1182
Rel. with community group	2.09	1.1	1	4	1180
Attended a community meeting	2.43	1.2	0	4	1179
Got together with others to raise an issue	2.13	1.3	0	4	1178
Past yr: Contacted local gov. councillor	0.68	1	0	3	1176
Past yr: Contacted MP	0.38	.84	0	3	1174
Past yr: Contacted government agency	0.22	.65	0	3	1177
Past yr: Contacted party official	0.35	.82	0	3	1175
Past yr: Contacted traditional leaders	1.39	1.3	0	3	1176
Past yr: Contacted religious leaders	1.70	1.3	0	3	1182
<i>Panel C: 2012 Political activity</i>					
Political engagement index (pre-Ebola)	0.00	1.5	-1.7	2.8	1175
2012: Voted in national election	0.75	.43	0	1	1191
2012: Attended campaign rally	0.43	.49	0	1	1180
2012: Attended meeting with candidate	0.45	.5	0	1	1182
2012: Persuaded others	0.37	.48	0	1	1177
2012: Worked for candidate or party	0.19	.39	0	1	1181
<i>Panel D: Ebola exposure</i>					
Ebola exposure index	0.00	1.9	-3.5	2	1102
Close friend or relative died from Ebola	0.34	.47	0	1	1137
Close friend or relative infected with Ebola	0.36	.48	0	1	1142
Due to Ebola: Unable to attend school	2.02	1.2	0	3	1151
Due to Ebola: Unable to work/earn income	1.93	1.2	0	3	1161
Due to Ebola: Unable to attend social gath.	2.04	1.2	0	3	1162
Due to Ebola: Unable to get medical care	1.95	1.2	0	3	1160

*Note: The summary statistics presented here are only for continuous and ordinal variables. While I discuss several in the body of the text, for the full coding of ordinal variables, please refer to Afrobarometer Round 6 codebook.*

## PCA indices construction

**Table A.2: Political engagement PCA index**

Eigenvalue	( $\rho$ )	Difference	Observations
4.176	0.418	2.497	1,145
Variable	Eigenvector	Unexplained	
Rel. with religious group	0.303	0.62	
Rel. with community group	0.325	0.56	
Attended a community meeting	0.324	0.56	
Got together with others to raise an issue	0.367	0.44	
Past yr: Contacted local gov. councillor	0.311	0.59	
Past yr: Contacted MP	0.302	0.62	
Past yr: Contacted government agency	0.255	0.73	
Past yr: Contacted party official	0.283	0.67	
Past yr: Contacted traditional leaders	0.355	0.47	
Past yr: Contacted religious leaders	0.321	0.57	

Note: This table displays summary characteristics of the political engagement index, including Eigenvalue,  $\rho$ ,  $p$ , and Eigenvectors or factor loadings for each component as well as the unexplained variance for each.

**Table A.3: Pre-Ebola political engagement PCA index**

Eigenvalue	( $\rho$ )	Difference	Observations
2.306	0.461	1.301	1,175
Variable	Eigenvector	Unexplained	
2012: Voted in national election	0.116	0.97	
2012: Attend campaign rally	0.487	0.45	
2012: Attended meeting with	0.525	0.36	
2012: Persuaded others	0.492	0.44	
2012: Worked for candidate or party	0.480	0.47	

Note: This table displays summary characteristics of the pre-Ebola engagement index, including Eigenvalue,  $\rho$ ,  $p$ , and Eigenvectors or factor loadings for each component as well as the unexplained variance for each.

**Table A.4: Ebola exposure PCA index**

Eigenvalue	( $\rho$ )	Difference	Observations
3.521	0.587	1.651	1,102
Variable	Eigenvector	Unexplained	
Close friend or relative infected with Ebola	0.120	0.95	
Close friend or relative died from Ebola	0.128	0.94	
Due to Ebola: unable to attend school	0.483	0.18	
Due to Ebola: unable to work/earn income	0.498	0.13	
Due to Ebola: unable to attend social gath.	0.500	0.12	
Due to Ebola: unable to get medical care	0.488	0.16	

Note: This table displays summary characteristics of the Ebola exposure index, including Eigenvalue,  $\rho$ ,  $p$ , and Eigenvectors or factor loadings for each component as well as the unexplained variance for each.

**Table A.5: Economic insecurity PCA index**

Eigenvalue	( $\rho$ )	Difference	Observations
2.470	0.548	1.922	1,185
Variable	Eigenvector	Unexplained	
Past yr: not enough food?	0.490	0.34	
Past yr: not enough clean water for home use	0.463	0.41	
Past yr: not enough medicines/treatment	0.499	0.32	
Past yr: not enough fuel to cook	0.331	0.70	
Past yr: not enough cash income	0.432	0.49	

Note: This table displays summary characteristics of the economic insecurity index, including Eigenvalue,  $\rho$ , and Eigenvectors or factor loadings for each component as well as the unexplained variance for each.

## B. Tabular presentation of main effects

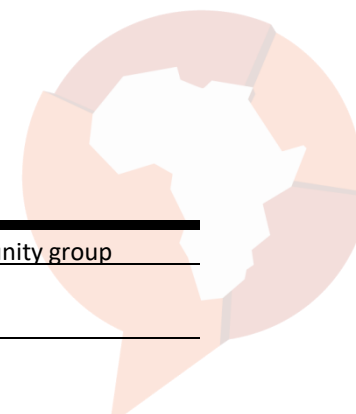
**Table B.1: Ebola index subcomponents and political participation**

	Friend/family		Due to Ebola unable to:			
	Infected	Dead	Attend school	Work	Attend gath.	Get med. care
Ebola measure	0.0877 (0.162)	0.0654 (0.165)	-0.2902*** (0.058)	-0.3133*** (0.058)	-0.2950*** (0.059)	-0.2982*** (0.061)
Age	0.0877*** (0.024)	0.0880*** (0.024)	0.0928*** (0.023)	0.0970*** (0.024)	0.0971*** (0.024)	0.0939*** (0.024)
Age <sup>2</sup>	-0.0005 (0.000)	-0.0005 (0.000)	-0.0006* (0.000)	-0.0006* (0.000)	-0.0006* (0.000)	-0.0006* (0.000)
Sex (Male = 1)	0.3127* (0.139)	0.3146* (0.139)	0.3315* (0.138)	0.3717** (0.139)	0.3439* (0.140)	0.3165* (0.141)
Education	0.0528 (0.031)	0.0527 (0.032)	0.0583 (0.033)	0.0605 (0.032)	0.0665* (0.033)	0.0624 (0.032)
$R^2$	0.315	0.315	0.341	0.343	0.338	0.340
Occupation FE	Y	Y	Y	Y	Y	Y
Sector FE	Y	Y	Y	Y	Y	Y
Religion FE	Y	Y	Y	Y	Y	Y
Language FE	Y	Y	Y	Y	Y	Y
Ethnicity FE	Y	Y	Y	Y	Y	Y
Observations	988	988	988	988	988	988

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ . Robust standard errors in parentheses.

Note: Each row describes the estimated coefficient for the variable in an OLS regression with the political engagement index as the outcome and the Ebola exposure index and each of its subcomponents as the primary treatment variable in turn. These regressions are fully controlled and include the vector of individual controls and enumeration area controls as defined in eq. (1) as well as an index measuring political activity in the previous national election.

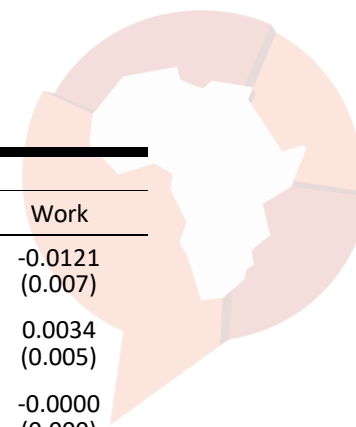


**Table B.2: Ebola index and participation index subcomponents**

	Political engagement index	Rel. with religious group	Rel. with community group
Ebola exposure index	-0.2097*** (0.045)	-0.0484** (0.016)	-0.0675*** (0.019)
$R^2$	0.231	0.178	0.135
Observations	993	993	993
	Attended a community meeting	Got together with others to raise an issue	Past yr: Contacted local gov. councillor
Ebola exposure index	0.0011 (0.019)	-0.0964*** (0.027)	-0.0729** (0.027)
$R^2$	0.223	0.189	0.124
Observations	993	993	993
	Past yr: Contacted MP	Past yr: Contacted government agency	Past yr: Contacted party official
Ebola exposure index	-0.0420 (0.025)	-0.0222 (0.013)	-0.0679*** (0.019)
$R^2$	0.112	0.109	0.148
Observations	993	993	993
	Past yr: Contacted traditional leaders	Past yr: Contacted religious leaders	How int. in public affairs
Ebola exposure index	-0.1333*** (0.028)	-0.1703*** (0.027)	-0.1304*** (0.029)
$R^2$	0.205	0.173	0.121
Observations	993	993	968

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ . Robust standard errors in parentheses.

Note: This table displays the observed relationship between the first principal component index of Ebola exposure on each of the subcomponents of the political participation index. These regressions include the vector of individual controls and enumeration area controls as defined above. Also included here is a self-reported measure of interest in public affairs. This was excluded from the index as it measures an attitude rather than behavior. Results discussed in the rest of the article are robust to its inclusion. These results are available upon request.

**Table B.3: Falsification exercise – OLS Ebola exposure on participation in the 2012 election**

	Index	2012 election participation				
	2012 PE	Vote	Rally	Meeting	Persuade	Work
Ebola exposure index	-0.0460 (0.027)	-0.0112 (0.007)	-0.0048 (0.009)	-0.0073 (0.009)	-0.0155 (0.009)	-0.0121 (0.007)
Age	0.0152 (0.017)	0.0059 (0.005)	-0.0007 (0.006)	0.0086 (0.006)	0.0010 (0.006)	0.0034 (0.005)
Age <sup>2</sup>	-0.0002 (0.000)	-0.0000 (0.000)	-0.0000 (0.000)	-0.0001 (0.000)	-0.0000 (0.000)	-0.0000 (0.000)
Sex (Male = 1)	0.2558* (0.103)	0.0141 (0.027)	-0.0272 (0.034)	0.1092** (0.034)	0.0612 (0.033)	0.0827** (0.027)
Education	0.0458 (0.026)	-0.0049 (0.007)	0.0134 (0.009)	0.0096 (0.009)	0.0053 (0.009)	0.0150* (0.007)
<i>R</i> <sup>2</sup>	0.108	0.127	0.083	0.096	0.094	0.102
Occupation FE	Y	Y	Y	Y	Y	Y
Sector FE	Y	Y	Y	Y	Y	Y
Religion FE	Y	Y	Y	Y	Y	Y
Language FE	Y	Y	Y	Y	Y	Y
Ethnicity FE	Y	Y	Y	Y	Y	Y
Observations	988	988	988	988	988	988

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ . Robust standard errors in parentheses.

Note: This table displays the observed relationship between the first principal component index of Ebola exposure on an index of political actions taken in the 2012 election and each of its subcomponents. These regressions include the vector of controls as defined in eq. (1). Note, five respondents have missing values for these survey items. For comparison, an OLS estimation analogous to that presented in Column 3 of Table 1 among the subgroup of 980 yields comparable results, estimating the Ebola exposure coefficient to be -0.203 (on the contemporary political engagement index) and a standard error of 0.035. This suggests that subgroup composition is not driving the null effects here. More detail on this result is available upon request.



## C. Additional robustness checks

### Evaluating response bias

**Table C.1: Evaluating response bias**

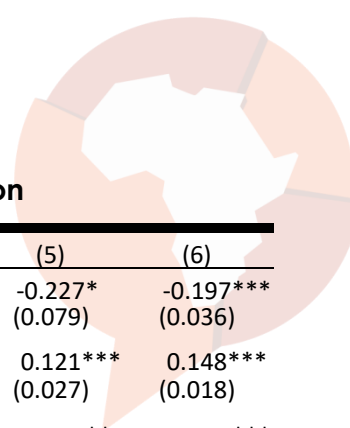
	Ebola exposure index	
	(1)	(2)
Age	0.0286 (0.021)	0.0332 (0.022)
Age <sup>2</sup>	-0.0004 (0.000)	-0.0005* (0.000)
Sex (Male = 1)	0.1678 (0.125)	0.1708 (0.124)
Education	0.0652* (0.032)	0.0154 (0.034)
$R^2$	0.145	0.358
F-statistic	3.2	2.2
Occupation FE	Y	Y
Sector FE	Y	Y
Religion FE	Y	Y
Language FE	Y	Y
Ethnicity FE	Y	Y
PSU FE		150
Observations	993	993

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ . Robust standard errors in parentheses.

Note: Column (1) regresses the vector of individual controls against the Ebola exposure index using ordinary least squares. Column (2) repeats this specification with the addition of 150 PSU fixed effects.

## Weighting and standard error construction

Table C.2: Robustness to weighting and standard error construction



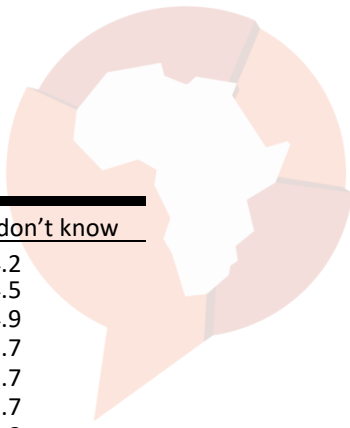
	(1)	(2)	(3)	(4)	(5)	(6)
Ebola exposure index	-0.210*** (0.045)	-0.227*** (0.035)	-0.227*** (0.040)	-0.227*** (0.049)	-0.227* (0.079)	-0.197*** (0.036)
Age	0.104*** (0.025)	0.121*** (0.022)	0.121*** (0.023)	0.121*** (0.023)	0.121*** (0.027)	0.148*** (0.018)
Age <sup>2</sup>	-0.001* (0.000)	-0.001*** (0.000)	-0.001*** (0.000)	-0.001*** (0.000)	-0.001** (0.000)	-0.001*** (0.000)
Sex (Male = 1)	0.489** (0.146)	0.412** (0.128)	0.412** (0.131)	0.412** (0.144)	0.412* (0.176)	0.384** (0.147)
Education	0.082* (0.037)	0.110** (0.034)	0.110** (0.035)	0.110** (0.037)	0.110* (0.041)	0.116*** (0.033)
Observations	993	993	993	993	993	993
R <sup>2</sup>	0.231	0.239	0.239	0.239	0.239	
Individual controls	Y	Y	Y	Y	Y	Y
Survey weighting	Y					
Robust VCE	Y	Y	Y	Y	Y	Y
No. clusters	150		150	90	14	
Quantile						.50

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ . Robust standard errors in parentheses.

Note: Column (1) presents the standard baseline specification presented throughout this article. Column (2) removes survey weighting and retains robust standard errors. Columns (3) through (5) cluster standard errors at the enumeration area, chiefdom, and district level, respectively. Column (6) presents the results of a quantile regression evaluated at the median.

## Multiple imputation

Table C.3: Missing values



	Observations	Total	% missing/don't know
Political participation index	1145	1191	4.2
Close friend or relative infected with Ebola	1142	1191	4.5
Close friend or relative died from Ebola	1137	1191	4.9
Due to Ebola: Unable to attend school	1151	1191	3.7
Due to Ebola: Unable to work/earn income	1161	1191	2.7
Due to Ebola: Unable to attend social gath.	1162	1191	2.7
Due to Ebola: Unable to get medical care	1160	1191	2.8
Age	1185	1191	.55
Sex (Male = 1)	1191	1191	0
Education	1188	1191	.27
Religiosity	1155	1191	3.3
Occupation	1184	1191	.64
Sector	1143	1191	4.4
Religion	1162	1191	2.7
Language	1191	1191	0
Ethnic community/group/tribe	1190	1191	.092
State capacity	1191	1191	0

Note: For each main variable, this table presents the total number of not missing, refused, or "don't know" responses and the percentage that are missing, refused, or "don't know." Most of the missingness stems from "don't know" responses, particularly for the Ebola exposure index. This likely reflects the fact that the outbreak was still winding down at the time of the survey.

Table C.4: Multiple imputation and Ebola exposure and political engagement

	Political participation index			
	(1)	(2)	(3)	(4)
Ebola exposure index	-0.2330*** (0.042)		-0.1966*** (0.042)	-0.1969*** (0.039)
Age		0.1084*** (0.021)	0.1129*** (0.021)	0.1396*** (0.023)
Age <sup>2</sup>		-0.0007** (0.000)	-0.0008** (0.000)	-0.0010*** (0.000)
Sex (Male = 1)		0.5231*** (0.143)	0.5647*** (0.143)	0.5219*** (0.143)
Education		0.0868** (0.033)	0.0944** (0.035)	0.1042* (0.041)
R <sup>2</sup>				
Occupation FE		Y	Y	Y
Sector FE		Y	Y	Y
Religion FE		Y	Y	Y
Language FE		Y	Y	Y
Ethnicity FE		Y	Y	Y
PSU FE				150
Observations	1191	1185	1185	1185

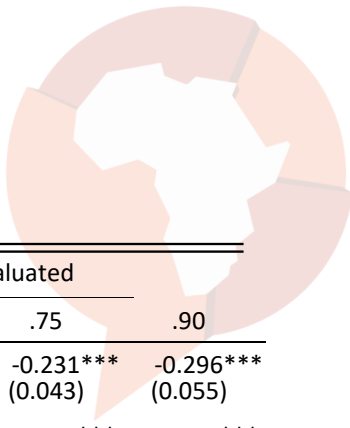
\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ . Robust standard errors in parentheses.

Note: This table displays the observed relationship between the first principal component index of Ebola exposure on an index of political engagement with all variables multiply imputed using five imputations estimated from chained regressions. A higher value indicates being more politically engaged. Column (1) is the raw correlation between these two measures. Column (2) is a multivariate regression of controls that does not include Ebola exposure. Column (3), our baseline specification, is the fully controlled OLS regression estimating the relationship between Ebola exposure and political engagement. PSU fixed effects are added in Column (4). Controls include the vector of individual characteristics and PSU controls and fixed effects as defined in eq. (1). Coefficients are survey-weighted with robust standard errors clustered at the PSU level.



## Distributional robustness

Table C.5: Quantile regression results



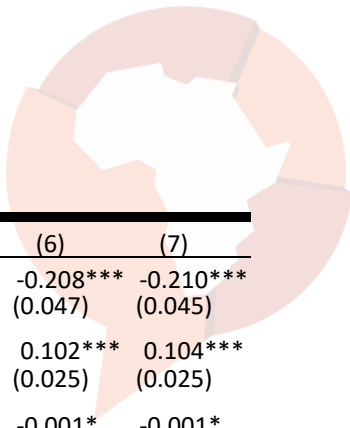
	Baseline	Quantile [Ebola exp.] evaluated				
	OLS	.10	.25	.5	.75	.90
Ebola exposure index	-0.227*** (0.035)	-0.175*** (0.037)	-0.193*** (0.036)	-0.197*** (0.036)	-0.231*** (0.043)	-0.296*** (0.055)
Age	0.121*** (0.022)	0.038 (0.020)	0.111*** (0.019)	0.148*** (0.018)	0.134*** (0.025)	0.124*** (0.027)
Age <sup>2</sup>	-0.001*** (0.000)	-0.000 (0.000)	-0.001*** (0.000)	-0.001*** (0.000)	-0.001*** (0.000)	-0.001*** (0.000)
Sex (Male = 1)	0.412** (0.128)	0.095 (0.140)	0.530*** (0.147)	0.384** (0.147)	0.403** (0.142)	0.530* (0.234)
Education	0.110** (0.034)	0.079* (0.037)	0.083* (0.036)	0.116*** (0.033)	0.141*** (0.034)	0.097 (0.060)
R <sup>2</sup>	0.239					
Occupation FE	Y	Y	Y	Y	Y	Y
Sector FE	Y	Y	Y	Y	Y	Y
Religion FE	Y	Y	Y	Y	Y	Y
Language FE	Y	Y	Y	Y	Y	Y
Ethnicity FE	Y	Y	Y	Y	Y	Y
Observations	993	993	993	993	993	993

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ . Robust standard errors in parentheses.

Note: This table presents the results of five quantile regressions and the unweighted OLS baseline regression for comparison. The last five columns estimate  $Y_i = X'_{i,d}\beta + \varepsilon_{i,d,\tau}, Q_\tau(Y_i|X_{i,d}) = X'_{i,d}\beta$ ,  $\tau \in (0, .25, .50, .75, .90)$ . These estimations evaluate the relationship between Ebola exposure and political engagement at different quantiles of the political engagement index. Namely, the OLS regression overestimates the effect at the 10th percentile and underestimates the relationship at the higher end of the distribution. However, the relationship is negative and significant at all levels.

## Over-controlling

Table C.6: Robustness to over-controlling




	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Ebola exposure index	-0.229*** (0.048)	-0.210*** (0.046)	-0.210*** (0.046)	-0.213*** (0.046)	-0.219*** (0.045)	-0.208*** (0.047)	-0.210*** (0.045)
Age	0.094*** (0.026)	0.102*** (0.025)	0.101*** (0.025)	0.101*** (0.025)	0.103*** (0.025)	0.102*** (0.025)	0.104*** (0.025)
Age <sup>2</sup>	-0.001* (0.000)	-0.001* (0.000)	-0.001* (0.000)	-0.001* (0.000)	-0.001* (0.000)	-0.001* (0.000)	-0.001* (0.000)
Sex (Male = 1)	0.516*** (0.129)	0.549*** (0.143)	0.557*** (0.144)	0.509*** (0.144)	0.521*** (0.145)	0.488** (0.147)	0.489** (0.146)
Education	0.022 (0.033)	0.032 (0.036)	0.039 (0.036)	0.061 (0.036)	0.070* (0.035)	0.073* (0.036)	0.082* (0.037)
Observations	993	993	993	993	993	993	993
R <sup>2</sup>	0.167	0.180	0.182	0.193	0.208	0.226	0.231
Occupation FE		Y	Y	Y	Y	Y	Y
Sector FE			Y	Y	Y	Y	Y
Religion FE				Y	Y	Y	Y
Language FE					Y	Y	Y
Ethnicity FE						Y	Y

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ . Robust standard errors in parentheses.

Note: Columns (1) through (7) in this table sequentially add additional covariates used in the baseline regression specification with the political engagement index as the outcome variable. Controlling for additional covariates slightly decreases the relationship between Ebola exposure and political engagement, though the significant, negative relationship is stable across all specifications.

## D. Additional findings

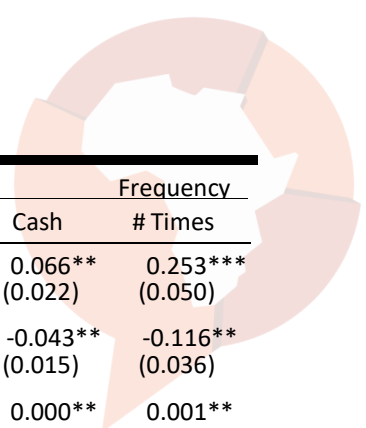
**Table D.1: Disentangling trauma and general exposure**



	Political participation		
	(1)	(2)	(3)
Ebola exposure index	-0.210*** (0.045)	-0.238*** (0.042)	-0.238*** (0.043)
Close friend or relative infected with Ebola		0.466* (0.183)	
Close friend or relative died from Ebola			0.480** (0.182)
Age	0.104*** (0.025)	0.105*** (0.026)	0.106*** (0.026)
Age <sup>2</sup>	-0.001* (0.000)	-0.001* (0.000)	-0.001* (0.000)
Sex (Male = 1)	0.489** (0.146)	0.463** (0.144)	0.466** (0.142)
Education	0.082* (0.037)	0.079* (0.037)	0.077* (0.037)
R <sup>2</sup>	0.231	0.240	0.241
Occupation FE	Y	Y	Y
Sector FE	Y	Y	Y
Religion FE	Y	Y	Y
Language FE	Y	Y	Y
Ethnicity FE	Y	Y	Y
Observations	993	993	993

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ . Robust standard errors in parentheses.

Note: This table displays the observed relationship between the first principal component index of Ebola exposure on the contemporary (2016) political engagement index. Columns (2) and (3) also include one subcomponent (whether the respondent had a friend/family member who was either infected or died from Ebola, respectively) in the specification.

**Table D.2: Ebola exposure and economic insecurity**


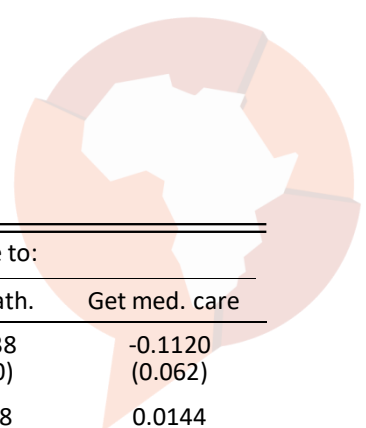
	PCA	Components					Frequency
	Index	Food	Water	Medicine	Fuel	Cash	# Times
Ebola exposure index	0.167*** (0.036)	0.114*** (0.027)	0.098*** (0.027)	0.146*** (0.024)	0.008 (0.021)	0.066** (0.022)	0.253*** (0.050)
Age	-0.066*** (0.018)	-0.011 (0.013)	-0.056*** (0.013)	-0.041** (0.014)	-0.024 (0.015)	-0.043** (0.015)	-0.116** (0.036)
Age <sup>2</sup>	0.001*** (0.000)	0.000 (0.000)	0.001*** (0.000)	0.000** (0.000)	0.000 (0.000)	0.000** (0.000)	0.001** (0.000)
Sex (Male = 1)	-0.099 (0.107)	0.005 (0.086)	-0.128 (0.087)	-0.043 (0.077)	-0.065 (0.074)	-0.015 (0.080)	0.155 (0.176)
Education	-0.077* (0.033)	-0.078** (0.024)	-0.033 (0.024)	-0.041 (0.022)	-0.007 (0.024)	-0.037 (0.023)	-0.097* (0.048)
R <sup>2</sup>	0.221	0.190	0.136	0.232	0.083	0.215	0.195
Occupation FE	Y	Y	Y	Y	Y	Y	Y
Sector FE	Y	Y	Y	Y	Y	Y	Y
Religion FE	Y	Y	Y	Y	Y	Y	Y
Language FE	Y	Y	Y	Y	Y	Y	Y
Ethnicity FE	Y	Y	Y	Y	Y	Y	Y
Observations	987	989	993	993	991	993	922

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ . Robust standard errors in parentheses.

Note: This table displays estimates of Ebola exposure on measures of economic insecurity. A first principal component index is the dependent variable in the first column; the following five columns replace each component as the outcome variable. Each component/question asks the respondents how often in the past year they have gone without a particular item. The final column regresses Ebola exposure against an additional measure (excluded from the index) asking how frequently the respondent went without the highest-order (from left to right in this table) item.

## External validity? Ebola in Liberia

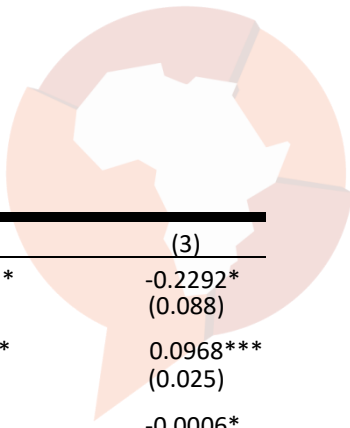
Table D.3: Ebola exposure and political engagement – Liberia



	Friend/family		Attend school	Due to Ebola unable to:		
	Infected	Dead		Work	Attend gath.	Get med. care
Ebola measure	-0.4043** (0.134)	-0.3545* (0.137)	0.0910 (0.057)	0.0110 (0.060)	-0.0638 (0.060)	-0.1120 (0.062)
Age	0.0164 (0.018)	0.0163 (0.018)	0.0141 (0.017)	0.0148 (0.017)	0.0148 (0.017)	0.0144 (0.017)
Age <sup>2</sup>	-0.0002 (0.000)	-0.0002 (0.000)	-0.0002 (0.000)	-0.0002 (0.000)	-0.0002 (0.000)	-0.0002 (0.000)
Sex (Male = 1)	0.0999 (0.107)	0.0935 (0.109)	0.1094 (0.109)	0.1037 (0.111)	0.0962 (0.110)	0.0995 (0.109)
Education	-0.0069 (0.042)	-0.0077 (0.043)	-0.0013 (0.044)	-0.0033 (0.044)	-0.0033 (0.043)	-0.0016 (0.043)
R <sup>2</sup>	0.351	0.349	0.344	0.342	0.343	0.345
Occupation FE	Y	Y	Y	Y	Y	Y
Sector FE	Y	Y	Y	Y	Y	Y
Religion FE	Y	Y	Y	Y	Y	Y
Language FE	Y	Y	Y	Y	Y	Y
Ethnicity FE	Y	Y	Y	Y	Y	Y
Observations	926	932	932	932	932	932

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ . Robust standard errors in parentheses.

Note: This table displays the observed relationship between the first principal component index of Ebola exposure on an index of political engagement using comparable data from Round 6 Afrobarometer (2015) in Liberia. A higher value indicates being more politically engaged. Column (1) is the raw correlation between these two measures. Column (2) is a multivariate regression of controls that does not include Ebola exposure. Column (3) is the fully controlled OLS specification estimating the relationship between Ebola exposure and political engagement. Controls include the vector of individual characteristics and enumeration area controls and fixed effects as defined in eq. (1).

*Alternative measures of Ebola exposure***Table D.4: Ecological level of Ebola and political participation**


	(1)	(2)	(3)
Ebola cases in chiefdom (1000s)	-0.2904*** (0.066)	-0.2904*** (0.063)	-0.2292* (0.088)
Age	0.1150*** (0.023)	0.1150*** (0.024)	0.0968*** (0.025)
Age <sup>2</sup>	-0.0008** (0.000)	-0.0008** (0.000)	-0.0006* (0.000)
Sex (Male = 1)	0.3928** (0.129)	0.3928** (0.129)	0.4682** (0.147)
Education	0.1091** (0.035)	0.1091** (0.036)	0.0843* (0.037)
Occupation FE	Y	Y	Y
Sector FE	Y	Y	Y
Religion FE	Y	Y	Y
Language FE	Y	Y	Y
Ethnicity FE	Y	Y	Y
Observations	993	993	993

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ . Robust standard errors in parentheses.

Note: Column (1) includes robust standard errors, Column (2) clusters standard errors by 150 PSUs, and Column (3) includes survey weighting and clustered standard errors.



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