THE ECONOMIC JOURNAL

The Economic Journal, **127** (*December*), 2581–2616. Doi: 10.1111/ecoj.12430 © 2016 Royal Economic Society. Published by John Wiley & Sons, 9600 Garsington Road, Oxford OX4 2DQ, UK and 350 Main Street, Malden, MA 02148, USA.

PATHOGENS, WEATHER SHOCKS AND CIVIL CONFLICTS*

Matteo Cervellati, Uwe Sunde and Simona Valmori

This article documents a statistically strong and quantitatively relevant effect of high exposure to infectious diseases on the risk of civil conflicts. The analysis exploits data on the presence and endemicity of multi-host vector-transmitted pathogens in a country, which is closely related to geo-climatic conditions due to the specific features of these pathogens. Exploiting within-country variation over time shows that this effect of pathogen exposure is significantly amplified by weather shocks. The results indicate health shocks and the outbreak of epidemics as a potential channel, while we find no evidence that the effect works through alternative channels like income, population dynamics, or institutions.

Civil conflicts cause considerable social and economic disruption and account for most of the conflict-related casualties after the Second World War. A recent literature (discussed below) has isolated socio-economic, institutional and geo-climatological factors that increase the likelihood of civil conflict. This article provides a first systematic investigation of the hypothesis that the overall health conditions relating to the disease environment, in terms of the exposure of humans to infectious pathogens, might be a relevant but so far largely overlooked determinant of civil violence.

From a theoretical perspective, health conditions influence the opportunity costs of violence. In a standard model of production *versus* predation, payoffs in both alternatives are affected by health. Individuals with poorer health and higher exposure to potentially fatal infections are more willing to take risks and discount the future more. Moreover, the permanent exposure to the risk of infections from potentially deadly, vector-transmitted diseases lies largely outside the control of individuals. This leads to passive and fatalistic attitudes regarding the chances to survive to old age and, thus, to limited future orientation and lower incentives to sustain the short-term costs

* Corresponding author: Uwe Sunde, LMU Munich, Geschwister-Scholl Platz 1, D-80539 München, Germany. Email: uwe.sunde@lmu.de.

The authors thank two referees and the editor, Frederic Vermeulen, for very helpful comments and suggestions on how to improve the article, and Margherita Fort, Andrew Oswald, Torsten Persson, Dominic Rohner, Mathias Thoenig, David Weil and Fabrizio Zilibotti for very useful discussions. The authors are also grateful to Roland Benabou, Lukas Buchheim, Antonio Ciccone, David de la Croix, Grigory Egorov, Joan Maria Esteban, Elena Esposito, Bernd Fitzenberger, Eliana la Ferrara, Stelios Michalopoulos, Omer Moav, Gerard Padro-i-Miquel, Elias Papaioannou, Jean-Philippe Platteau, Adam Przeworski, Debraj Ray, Anthony Strittmatter and seminar participants at Universities of Bologna, Freiburg, Göttingen, Modena, the Barcelona Workshop 'Towards Sustained Growth', the Zurich Workshop on the Economics of Conflict, the Economic Fluctuations and Growth Group at the NBER Summer Institute, the Workshop on Conflict and Development in Namur, the Workshop on the determinants of Civil Conflicts in Madrid, the second LEPAS conference in Alicante, the ESEM-EEA in Oslo, the workshop on civil conflicts in Bergamo, SCALA – University of St. Gallen and Warwick for helpful comments. We are grateful to the Centre for Research on the Epidemiology of Disasters (CRED) for supplying the data on natural disasters. Financial support from the European Union through the Career Integration Grant (618641 DISCON, Uwe Sunde) and Einaudi Institute for Economics and Finance (Matteo Cervellati) is gratefully acknowledged.

of peaceful cooperation in repeated interactions.[1] In addition, while negative health shocks may make fighting more difficult and costly, they also increase the exposure and vulnerability to predation. In the context of organised violence, a low opportunity cost of violence can also facilitate the recruiting of rebels and mercenaries.

International organisations repeatedly warn about the crucial role of health conditions and health shocks as the main risk factors for social and political disruption and ultimately civil violence. They emphasise the importance of their prevention and timely containment.[2] This is particularly relevant to epidemic diseases where outbreaks and the associated increase in the risk of death can cause temporary spikes in violence.[3] However, whether and how the exposure to pathogens affects the risk of civil violence and whether this effect is amplified by short-term shifts in pathogen exposure are questions that have not yet been investigated empirically.

This article investigates the role of disease exposure for civil conflict using cross-country panel data over the past half century. The analysis is based on a novel measure of disease exposure, which has several properties that make it particularly suited for this purpose. The measure uses cross country information about the exposure to multi-host vector-transmitted (MHV) pathogens from different sources and periods in time. We concentrate attention on the presence of pathogens at the extensive margin in terms of the number of MHV diseases with a significant risk of death that have ever been diagnosed or, alternatively, that are endemic in a country. MHV pathogens grow in human and non-human hosts and can be transmitted only through specific vectors. The pathogens of this class include diseases that seriously affect health and are recurrently epidemic. Vaccines are generally not available yet and cures are often difficult to administer. Eradication of MHV pathogens has proved difficult since these infectious agents exploit multiple hosts for their survival. As a result, no MHV pathogen included in the baseline measures has been successfully eradicated at the country level during the observation period.[4] Given their exclusive reliance on weather-sensitive vectors for transmission, the presence and endemicity of these pathogens is closely linked to the exogenous country-specific bio-climatological environment. Because of the serious health consequences and the difficulties related

[1] This has been documented in many contexts and using different methodologies, see, for example, Becker and Mulligan (1997) and Guiso *et al.* (2013) for predictions in economics, Aspinwall (2005) in social psychology, Lammers and van Wijnbergen (2008) for experimental findings and the studies of Lorentzen *et al.* (2008), Oster (2012), Adda and Lechene (2013) and Goudie *et al.* (2014) (with very different data and contexts) in empirical economics. Consistent with this, individuals in countries with lower life expectancy are less patient and more willing to take risks, as documented by recent representative evidence by Falk *et al.* (2015) for 76 countries.

[2] For instance the UN guidelines stating the codes of behaviour for disaster management programmes stress the importance of timely interventions also in view of the fact that 'the immediate effect of epidemics is of course that they cause illness and death. Secondary effects are social and political disruption and economic loss' (Disaster and Risk Management Guides – Epidemic, United Nations Platform for Space-based Information for Disaster Management and Emergency Response, UN, http://www.un-spider.org/disaster-management-guides/epidemic, last accessed 18 February 2016).

[3] This is illustrated by the civil violence triggered by the 2014 Ebola epidemic. In that episode, as reported by international media, even the mere fear of epidemics sparked, and 'as the numbers of dead has surged, so has the violence' (*Washington Post*, 19 September 2014, available at: washingtonpost.com/news/morning-mix/wp/2014/09/19/why, last accessed 18 February 2016).

[4] Malaria is the only exception, see the discussion below.

to their prevention and control, fighting these pathogens has become a key priority for international organisations such as the WHO.

We use the number of MHV pathogens ever detected (or, alternatively, endemic) in a country as count index of MHV pathogen exposure at the extensive margin. This provides a simple, time-invariant measure of pathogen exposure, which is of comparable quality across countries and little affected by measurement error, in particular in the context of civil violence. As discussed in detail in Section 1, the specific features of MHV pathogens and the construction of count indices based on the mere presence or endemicity of such infectious agents in a country limits by construction the potential for reverse causality and measurement error, compared to data on disease prevalence in terms of affected individuals or casualties. The resulting measures of country specific disease environment have the additional advantage that they can also be constructed by using information from historical sources on the global distribution of pathogens.

The empirical analysis proceeds in two steps. In the first step, we explore the existence of a reduced-form effect of the exposure to MHV pathogens, which proxies the total latent disease burden faced by the population, on civil conflicts. The empirical results document that, on a global scale, a harsher disease environment implies a quantitatively relevant increase in the risk of civil conflicts of 8–10 percentage points for a one-standard deviation increase in the measure of pathogen exposure.[5] Compared to an unconditional probability of conflict of 17%, this corresponds to an increase of around 50%. The findings are confirmed with alternative measures based on different codings, with different subsets of pathogens and with measures based on information from the early twentieth Century, well before the beginning of the observation period for conflicts. The analysis accounts for non-linear trends and the role of past conflicts, and exploits variation within world regions. The exposure to pathogens also affects the frequency of civil conflicts in terms of conflict onsets, their duration, the incidence of internal conflicts for the control of government, of small localised struggles and the extent of individual level violence in terms of homicides. Extensive robustness checks suggest that the results are unlikely to be driven by reverse causality running from civil violence to the global distribution of MHV pathogens in the last century. The baseline estimates are essentially unaffected by the inclusion of an extensive set of economic, demographic, institutional and geographical control variables that have been found relevant in the literature, as well as to the inclusion of additional geographical controls. The evaluation of the potential bias from omitted variables suggests that the explanatory power of unobserved heterogeneity required to explain away the effect of the disease environment would have to be several times that of all observed covariates in extensive specifications of the empirical model.

In the second step of the analysis, we move beyond a cross-country perspective and test the hypothesis that health shocks may trigger violence. The analysis exploits the link between short-term fluctuations in weather conditions, which affect the prevalence of disease vectors and the associated short-term variation in the effective exposure to

[5] In terms of interpretation, the results of this analysis correspond to the effect of additional MHV pathogens being present in a country compared to a country that does not face the burden of these pathogens.

MHV pathogens. The empirical approach relies on year-to-year variation in weather shocks within a country over time. Specifically, it exploits the effects of exogenous variation in weather in interaction with the disease environment while accounting for country and time fixed effects, thus following the logic of a difference-in-differences intention-to-treat analysis. We consider short-term fluctuations in terms of droughts and heat waves, that is, periods of exceptionally low precipitation that create standing water which can serve as breeding grounds for the vectors and unusually warm conditions that favour the reproduction of vectors and pathogens. The findings document a robust effect of droughts and heat waves in interaction with the disease environment, raising the probability of conflict incidence by 3 to 5 percentage points, which corresponds to an increase of 30% to 50% compared to the unconditional mean. The results are robust to the consideration of extensive controls including the interaction between weather shocks and a large set of other potentially relevant country-specific characteristics, as well as interactions between the exposure to pathogens and alternative short-term triggers of conflicts like shocks to income, population, or the quality of institutions. Finally, we explore the channel behind the intention-to-treat results. Exploiting again within-country variation over time, we find that droughts and heat waves increase the likelihood of epidemics in interaction with the disease environment, while we find no evidence for potential alternative channels such as income, population density, institutions, or rents from natural resources. To test more explicitly the role of health shocks, we exploit the weather shocks and their interaction with the exposure to pathogens as instruments for the out-break of epidemics. The results provide a first piece of evidence that the interaction between the disease environment and weather shocks trigger epidemics, while the so-instrumented epidemics significantly increase the risk of civil conflicts.

The article contributes to several branches of the literature. Among the determinants of conflicts that have been documented are the role of income and poverty, weak or non-democratic institutions, political instability, specific features of the geographic environment, ethnic fragmentation and polarisation, and genetic diversity (Fearon and Laitin, 2003; Collier and Hoeffler, 2004; Montalvo and Reynal-Querol, 2005, as well as Collier and Rohner, 2008; Collier *et al.*, 2009; Esteban *et al.*, 2012; Arbatli *et al.*, 2013; among others). This article complements these works by providing a first attempt of a systematic investigation of the disease environment as a relevant, but so far neglected, country-specific determinant of civil conflict.

The article also relates to investigations of the short-term triggers of conflicts. Previous studies have exploited exogenous variation in weather conditions that have been interpreted as economic shocks (Miguel *et al.*, 2004, Couttenier and Soubeyran, 2014; Couttenier and Berman, 2015). Blattman and Miguel (2010) and Couttenier and Soubeyran (2015) provide recent surveys of the literature. Consistent with the evidence shown in the present article, health-related shocks can also be interpreted as negative economic shocks (in a broader sense) in view of their impact on labour productivity (or the opportunity costs for supplying labour to production rather than predation), or on the costs of medication and medical treatment, and more generally on living conditions (above and beyond income). The findings thus provide first evidence on a health channel and are also relevant for the ongoing debate regarding the potential consequences of climate change for civil conflicts.

In a broader perspective, the results relate to the literature on the interaction between the disease environment and long-run comparative development. The role of geography and the long run exposure to diseases across human societies has been emphasised by Diamond (1997). Using disaggregate data for Africa, Alsan (2015) documents the influence of the geographic suitability for the tsetse fly, the vector that transmits trypanosomiasis, on the incentives to use domesticated animals, and thereby on population density and the degree of political centralisation. Cervellati and Sunde (2011, 2013) have investigated the reduced form effect of health on economic growth in the last half a century. The results in this article contribute to this literature by adding to the picture the previously unexplored potential role of the exposure to pathogens for conflicts and indirectly for institutional quality as a complementary channel that links diseases and health to long-term development.

The article is structured as follows. Section 1 describes the measurement of pathogen exposure and the data. The empirical results for the disease environment are reported in Section 2 and the results for the interaction between disease environment and weather shocks are reported in Section 3. Section 4 concludes.

1. Data and Measurement

1.1. *Exposure to Pathogens: Conceptual Background*

Infectious agents affecting humans can be classified in terms of the host and the transmission channel. In terms of host, pathogens can be classified as human only (like HIV) if the pathogen has only humans as a reservoir, zoonotic (like plague or anthrax) if the host of the pathogen is an animal, or multi-host if the pathogen uses both humans as well as non-humans as a reservoir. In terms of transmission, pathogens can either be transmitted directly from human to human (like HIV or influenza), or through a vector (like the mosquito *Aedes Aegypti* for dengue or *Anopheles* for malaria).[6]

The analysis in this article focuses on the class of MHV pathogens, which turns out to have several characteristics that make them particularly appropriate for the purpose of this article. The fact that these pathogens exploit multiple hosts as reservoirs (including sylvatic species) for their survival makes them difficult to treat and control even in the more developed countries. Vaccines are generally not available for MHV pathogens, which have proved resurgent even after long periods of extensive campaigns.[7] In some countries, extensive campaigns that were heavily based on the the use of insecticides and the treatment of affected cases helped in controlling the spread of MHV diseases but the difficulty of eliminating the vectors and of eradicating the pathogen from all potential hosts is still a substantial obstacle for eradication. As a consequence, with the relevant exception of malaria, none of the deadly MHV pathogens has been successfully eradicated at the country level during the observation

[6] A complete classification of pathogens affecting humans, including the infectious agent, the taxonomic group, the host category and the vectors is available from the Ecological Archive E88-114-A1 at: http://esapubs.org/Archive/ecol/E088/114/appendix-A.htm (last accessed: 18 February 2016).

[7] Exceptions are malaria, yellow fever and typhus epidemic for which vaccines are available although, especially for yellow fever, the vaccine provides incomplete protection, is unstable and heat sensitive. After infection, no cure of the disease is currently available.

period and even the prospects of successful eradication at subnational levels in the near future vary considerably across pathogens.[8] While health infrastructure and active campaigns are key determinants of prevalence in terms of the number of affected cases, the lack of successful eradication implies that if a MHV pathogen has ever been present in a country in the past it is present still today.[9]

The second relevant feature of MHV pathogens is that they are transmitted from human to human only through specific vectors. As a consequence, the endemicity of these pathogens, that is, the presence of the pathogen in the population in a stationary state without external inputs, requires specific bio-climatological conditions that are suitable for the respective transmission vectors. This is different from directly transmitted diseases such as HIV-AIDS or influenza, a MHV pathogen cannot be endemic in a country unless the specific vector is present and finds sufficiently suitable biological conditions to survive, get infected and spread. A relevant implication is that while outbreaks of localised civil violence affect the prevalence of diseases in the population, they cannot make a pathogen of the class of MHV pathogens endemic in a country if vectors and suitable conditions for their survival are absent. For the same reason, MHV pathogens are affected little by migration of infected humans, economic activities, and trade, and are the least globalised diseases with humans as reservoirs, as documented by Guernier *et al.* (2004) and Smith *et al.* (2007). As a result, the worldwide distribution of MHV pathogens is essentially determined by climate and geography.

Third, MHV pathogens have severe health consequences. From the 17th until the early twentieth century, vector-transmitted pathogens have been estimated to be responsible for more human deaths than all other causes combined (Gubler, 1991). A high exposure to MHV diseases also indicates a high exposure to several non-vector transmitted diseases since the geo-climatic conditions favouring the endemicity of MHV diseases are also a favourable environment for other pathogens. MHV agents are also co-factors for other potentially fatal diseases.[10] Individuals exposed to many MHV

[8] In this respect, malaria is an exceptional MHV pathogen, since the only non-human host of the pathogen is the same mosquito that also acts as vector for the pathogens. For this reason, the elimination of the vector allowed the full eradication of malaria from OECD countries. As a consequence, malaria is excluded from some of the measures used in the empirical analysis. The results are essentially unaffected by the inclusion of malaria, however, as it does not fundamentally change the overall informational content of the measure of disease environment within the subset of developing countries, as discussed below.

[9] While good health infrastructure and active campaigns are key determinants of disease prevalence in terms of affected cases they were, so far, not sufficient for the full eradication of these diseases even in the more developed countries like the US. In the empirical analysis, we perform several checks to investigate the stability of the measures of pathogens exposure over time and across different alternative codings.

[10] For instance, countries exposed to dengue are likely to be exposed also to other diseases that are transmitted by the same mosquito (*Aedes Aegypti*), such as yellow fever. More specifically, the climatic conditions favouring the bio-diversity of human pathogens in this class also favour the bio-diversity in the other pathogen classes, as documented by Smith *et al.* (2007). In fact, the countries with the highest exposure to MHV diseases account for about two thirds of the countries with the highest number of all other infectious disease pathogens. Out of the countries in the highest quartile in terms of MHV, 65% are also in the highest quartile of all other infectious pathogens. The exposure to MHV diseases is a significant predictor of several types of health outcomes and of the risk of observing different types of epidemics (Cervellati *et al.*, 2012). In addition, a high exposure to these pathogens increases the susceptibility of individuals to infections with other diseases and thus implies a higher vulnerability of the population to health shocks. Likewise, individuals that have already a weakened immune system due to other infections, e.g. with HIV, face higher risks in areas with higher pathogen exposure. See, for example, Gopinath *et al.* (2000) and Karp and Auwaerter (2007) and references therein.

pathogens face a faster accumulation of health deficits and exhibit higher levels of frailty and mortality even in the context of similar health care institutions (Rockwood and Mitnitski, 2007).

1.2. *Measurement of Pathogen Exposure*

Addressing the question whether and how the exposure to diseases affects the risk of civil violence requires a measure of pathogen exposure that includes diseases with relevant health consequences, that reflects the exogenous, country-specific, disease environment while not being affected by the occurrence of localised civil conflicts, and that is based on reliable data of comparable quality for a large number of countries. To this end, we construct count indices reflecting the number of MHV pathogens present in a country at the extensive margin according to different criteria.

As a benchmark, we consider a count index of all MHV pathogens that are present in a country. More specifically, to build the measures of the country-specific pathogen exposure we collected information on whether a particular MHV pathogen has ever been detected, i.e. reported or diagnosed, in a country. The MHV pathogens are dengue, yellow fever, leishmaniasis visceral, relapsing fever, typhus epidemic, angiomatoses, filariasis-brugia malayi, leishmaniasis (mucocutaneous and cutaneous), malaria, onchocerciasis, trypanosomiasis africanis and trypanosomiasis (American sleeping sickness). Between zero and twelve of these pathogens can be observed in a country. Table B1 in online Appendix B reports information on all MHV diseases, including the vectors, the spread, and the prospects of treatment and eradication for the MHV pathogens.[11] We also use a restricted set of MHV pathogens that involve a significant risk of death to focus on the pathogens with the most severe health consequences.

Data on the presence of MHV pathogens are available at high and comparable quality for most countries. The raw data on the global distribution of human pathogens are from the Global Infectious Disease and Epidemiology Online Network (GIDEON) database.[12] Importantly, we only exploit information on the presence of a pathogen in a country at the extensive margin (i.e. a count of MHV pathogens in a country) and not information on the current spread, prevalence, or number of affected cases. Given their relevance for human health, these diseases are carefully monitored also in the developing world. Information on their mere presence in a country is subject to much lower measurement error than data on current prevalence or general health outcomes, which are likely to suffer from poor measurement in the countries with worse health systems, or with incomplete coverage due to, for example, civil conflict. The use of count indices also delivers a conservative measure of the actual disease exposure since it provides a comparable measure regardless of whether a

[11] The main sources of information are the GIDEON database and the World Health Report (2008) available at: http://www.who.int/whr/2008/whr08_en.pdf (last accessed: 18 February 2016) and the report of the International Task Force for Disease Eradication (1993 updated in 2008) whose summary results are available at: http://www.cartercenter.org/resources/pdfs/news/health_publications/itfde/updated_disease_candidate_table.pdf (last accessed: 18 February 2016).

[12] This data set provides information for more than 300 human infectious agents in more than 200 countries. The data are available upon subscription at http://www.gideononline.com.

country has a functioning health system and faces civil violence in a given year. Since the presence of the pathogen in a country is necessary for an infection, cross country differences in the count indices are informative on cross country differences in the latent burden of these diseases faced by the population without suffering from the obvious concerns of endogeneity associated with measures of disease prevalence at the intensive margin.

As a drawback, a coding of count indices based on the mere presence can also be influenced by isolated cases that might reflect migrants, refugees, or tourists, however. In addition, such an index does not account for the possibility of successful eradication of diseases, since a pathogen would be recorded if it has ever been present in the past, even if it is not any longer present. This definition of pathogen exposure at the extensive margin might thus not necessarily be informative about the actual latent prevalence of cases in the population. Therefore, as an alternative measure of pathogen exposure we coded the presence of MHV pathogens based on whether a pathogen is currently endemic in a country. This measure does not rely on prevalence in terms of diagnosed cases but only on information on whether the pathogen is coded by GIDEON to be endemic in (at least part of) the territory of a country. Endemicity requires that a pathogen can reproduce itself in a population without the need for external inputs.[13] Despite their conceptually very different codings of exposure to MHV pathogens, the resulting count indices display a very high correlation (above 0.9).[14] Since the presence of MHV pathogens in a country is mainly driven by the country specific bio-climatological conditions and in light of the difficulties of eradication (MHV pathogens have not been successfully eradicated at the country level), the high similarity of the resulting measures should be expected.[15]

Another advantage of the proposed measures of pathogen exposure is that it is possible to combine information from historical sources on the worldwide distribution of infectious diseases from the late nineteenth and early twentieth centuries and construct a count index for a subset of these pathogens.[16] The historical index follows the exact same logic as the benchmark index by being based on the presence of each pathogen at the extensive margin in a country and not on the prevalence or the

[13] Technically, a disease is in an endemic steady state if the basic reproduction rate of the infectious agents in the population times the share of the population that is not immune equal one. GIDEON codes a disease as endemic or potentially endemic if 'autochthonous cases of this disease are reported or have been reported in recent years, or if there is a considerable likelihood of contracting the disease, by virtue of ongoing presence of the infecting agent in local reservoirs/vectors'.

[14] The mismatch between the two measures is typically due to cases in which these vector-transmitted pathogens have been diagnosed in countries with non-suitable habitats (e.g cases of dengue in Sweden), for instance related to returning travellers. In fact, one of the reasons the GIDEON platform was set up was to support diagnoses of anomalous and infrequent cases of infections with symptoms that are uncommon in a given country.

[15] An alternative strategy would be to exploit information on the (predicted) presence of the vectors for each pathogen in each country. This alternative is prevented by lack of reliable cross country data for all countries and pathogens. The information on endemicity delivers, however, conceptually equivalent information, since a necessary condition for endemicity of a MHV pathogen in a country is the presence of the vectors that transmit the pathogen. In this respect, the index based on endemicity also has the advantage of limiting the problem of false positives.

[16] The original sources are Craig and Faust (1943) and Simmons *et al.* (1944), see also for an application using these data for different sets of diseases. For comparability with the baseline measures based on GIDEON data, we again restrict attention to the subset of MHV pathogens.

presence of the pathogen in specific areas within these countries. The historical data are subject to some limitations, however. Information is available only for a subset of countries and it is not possible to code the presence of a pathogen discriminating between actual diagnosed cases or endemicity. Also, the historical sources do not report information on modern classifications of pathogens such that, for instance, we cannot distinguish between different forms of leishmaniasis. In addition, for some pathogens, the information on their presence is only based on the presence of the respective vectors, even if information on clinical cases is missing or not fully reliable. This is particularly the case for relapsing fever and yellow fever, which thus cannot be included in the index constructed using historical data. The inferior medical knowledge and the more limited information available during the first part of the twentieth century also implies a lower coverage of pathogens and lower accuracy compared to the GIDEON data. Nevertheless, the historical sources allow the construction of measures of the disease environment that are predetermined to the civil conflicts in the period 1960–2010.

Finally, we constructed a database on the occurrence of epidemics caused by vector-transmitted and weather sensitive pathogens in each year for the period 1960–2007. The raw data are extracted from the EM-DAT: The OFDA/CRED International Disaster Database, and from the GIDEON database. Due to the lack of reliable cross-country panel data on prevalence in terms of affected cases and the serious problems of measurement error and endogeneity with such data, we exploit information on whether an epidemic was observed at the extensive margin in a given country and year. Information on the occurrence of epidemics will be used to investigate the potential role of short-term health shocks exploiting year-to-year exogenous variation in weather in IV settings.

1.3. *Data on Civil Conflicts and Covariates*

The benchmark source for information on civil wars and conflicts is the data from the UCDP/PRIO Armed Conflict Data set provided by the Peace Research Institute of Oslo (PRIO) for the period 1960–2007 (version v4, 2012). The baseline-dependent variable, denoted 'Civil Wars', refers to the incidence of internal armed conflicts with at least 25 battle-related deaths per year and more than 1,000 battle-related deaths over the entire duration of the conflict. Internal armed conflicts in a country are defined as a 'contested incompatibility concerning government and/or territory with the use of armed force between two parties, of which at least one is the government of a state'. The sample used in the analysis includes up to around 140 countries. We also explore the role of exposure to pathogens on other types of civil conflicts in terms of smaller conflicts, conflicts for the control of government and individual violence in terms of homicides.

There have been warnings by epidemiologists on the existence of relevant interactions between the exposure to a high number of endemic vector-transmitted pathogens and weather conditions.[17] The literature has emphasised the role of weather shocks in terms of unusually warm periods associated with droughts for facilitating the reproduction of transmission vectors and in creating standing water that serves as a

[17] See the WHO's World Health Report (2008) available at: http://www.who.int/whr/2008/whr08_en.pdf (last accessed: 18 February 2016).

breeding ground for most vectors.[18] The effects of pathogen exposure on the opportunity cost of conflicts might therefore be amplified by weather shocks. To test this conjecture in the second part of the article, we use weather data on temperature and precipitation at yearly frequencies as well as information on exceptional weather conditions in terms of the occurrence of episodes of droughts and heat waves. These variables are taken from the OFDA/CRED International Disaster Database. Droughts are climatological conditions corresponding to an 'extended period of time characterised by a deficiency in a region's water supply that is the result of constantly below-average precipitation', while heat waves are meteorological events of extreme weather conditions in terms of temperatures above the respective long-run mean.[19] The occurrence of climatological and meteorological shocks offers a source of exogenous (year-to-year) variation which can be exploited in interaction with the country-specific disease environment.

Finally, the analysis in the article uses an extensive set of time invariant and time-varying covariates. A brief description of the main variables of interest as well as their sources is reported in Table A1 in Appendix A. Table A2 reports the data sources of the variables that are used as controls and in the robustness analysis.

2. The Effect of the Disease Environment on Civil Conflict

2.1. *Baseline Results*

The main determinants for the number of MHV pathogens that are present or endemic in a country are essentially the country-specific bio-climatological conditions. To get a first impression of the relationship between the country-specific disease environment in terms of exposure to MHV pathogens and the likelihood of civil conflict outbreaks, we visualise the patterns in the raw data. Panel (*a*) of Figure 1 plots the worldwide distribution of MHV pathogens. Panel (*b*) of Figure 1 depicts, for each country in the sample, the number of five-year periods with at least one civil war. There is a positive unconditional correlation between the number of MHV pathogens in a country at the extensive margin and the average incidence of civil conflicts.[20]

As a first step to a formal empirical analysis of this relationship, we follow the literature on the long-term determinants of civil conflicts and consider panel data, aggregated at five-year intervals, that allow controlling for time-varying covariates and past conflicts. As benchmark we estimate the empirical model:

$$\Pr(Conflict_{i,j,t}|\boldsymbol{X}_{i,j,t}) = F(\boldsymbol{X}_{i,j,t}\boldsymbol{\gamma}), \tag{1}$$

where the dependent variable $Conflict_{i,j,t}$ is a binary indicator variable that takes value 1 if a conflict is observed in period t in country i (belonging to region j), and $\boldsymbol{X}_{i,j,t}\boldsymbol{\gamma}$ denotes the vector of variables of interest and their associated coefficients:

[18] See for example, Reiter (2001) and Süss *et al.* (2008) for the case of mosquitoes and ticks, and Hunter (2003) for waterborne and vector-transmitted diseases in general.

[19] Data on the occurrence of droughts and heat waves are available at yearly frequency from the EM-DAT: The OFDA/CRED International Disaster Database. http://www.emdat.be, Université Catholique de Louvain, Belgium.

[20] See Figure B1 in online Appendix B.

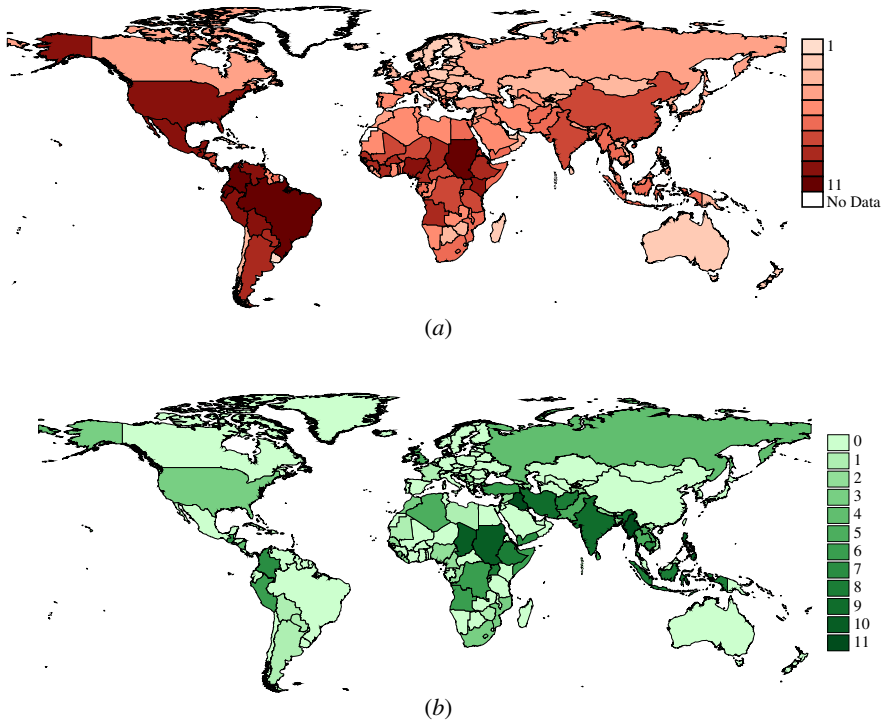

Fig. 1. *The Global Distribution of Multi-host Vector-transmitted Pathogens (MHV) and Civil Wars. (a) MHV Pathogens; (b) Civil Wars (PRIO)*

Notes. Panel (*a*): Multi-host vector-transmitted Pathogens. Panel (*b*): Five year periods with at least one civil war in the period 1960–2010. See Section 1 and Tables A1 and A2 for details and data sources. Colour figure can be viewed at wileyonlinelibrary.com

$$\boldsymbol{X}_{i,j,t}\gamma = \beta Pathogens_{i,j} + \alpha_j + \eta_t + \boldsymbol{Z}'_{i,j,t}\boldsymbol{\delta} \tag{2}$$

and $F(\cdot)$ denotes an appropriate transformation function. In the estimation, we apply probit models as well as other specifications of $F(\cdot)$, such as linear probability or logit models. The variable of main interest, $Pathogens_{i,j}$, is a measure of the disease environment in terms of pathogen exposure (in country i belonging to region j), and β measures the effect of disease exposure on conflict incidence. In addition, the vector of explanatory variables $\boldsymbol{X}_{i,j,t}$ includes binary indicators for world regions, α_j, to account for region specific unobserved characteristics.[21] Period fixed effects, η_t, are included to control for possible trends and waves in conflict incidence thereby accounting, for example, for the increasing number of civil conflicts after the end of the cold war.[22] The vector $\boldsymbol{Z}_{i,j,t}$ includes a large set of

[21] We classify countries in nine sub-regions including on average 12 countries each: Asia, Asia Pacific, North Africa, Sub-Saharan Africa, Middle East, North-America, Central America, South-America and Europe. Alternative specifications using a coarser grouping by continents, or using the regional classification of the World Bank with only seven regions deliver similar results. Details are available upon request.

[22] The proportion of countries with conflicts increases from about 10% in the 1960s to above 20% in the 1990s, see also Collier and Hoeffler (2004) and Blattman and Miguel (2010).

time-invariant and time-varying country characteristics. In particular, the covariates include all the time-invariant and time-varying control variables that have been used in the existing studies on the empirical determinants of civil violence, as discussed in detail below. The time-varying controls are measured at the beginning of the respective five-year periods over which the dependent variable is measured. In the robustness checks we also consider alternative data frequencies (from cross-section to yearly data) and alternative specifications of the vector of explanatory variables. For direct comparability of coefficient estimates, all right-hand side variables (excluding binary variables) are standardised on the respective estimation sample. Standard errors are robust and allow for clustering at the country level and account for heteroscedasticity and serial autocorrelation.

As baseline specification, we estimate probit models and report the marginal effects of the regressors computed at the respective means of the regressors. As discussed in subsection 1.2, the baseline measure, *Pathogens*, restricts attention to the number of deadly MHV pathogens ever detected in a country. Table 1 reports the results of the role of disease environment on the incidence of civil wars. Column (1) reports the unconditional effect. The estimates imply an increase of 10 percentage points in the likelihood of conflict for a one-standard deviation increase in the measure of disease environment. With an unconditional probability of conflict of 17%, this corresponds to an increase in the likelihood of conflict incidence of about 60%.[23]

Column (2) controls for standard time-invariant and time-varying determinants of civil conflict from the existing literature. The 'baseline controls' comprise log population density, ethnic polarisation, log GDP per capita, the share of GDP accounted for by primary commodity exports, the percentage of mountainous terrain, an indicator for non-contingent land, and a democracy index (polity IV). In addition, the specification includes 'geographic controls' such as absolute latitude, the log distance of the country centroid from the nearest coast, the log distance of the country centroid to the nearest navigable river, and the ratio of the population within 100 kilometres of ice-free coast relative to the total population.[24] The description and data sources for the baseline and geographic controls are reported in Tables A1 and A2 in Appendix A.[25]

The increase of 9.2 percentage points in the likelihood of conflict for a one-standard deviation increase in the measure of disease environment corresponds to a rise of more than 50% in the likelihood of conflict incidence compared to the

[23] In the estimations, the measure of disease environment is standardised, as described above. The original variable ranges from 0 to 6 with mean 2.90 (SD 1.56) so that one additional pathogen in a country implies an increase in conflict incidence of almost 40% compared to the unconditional mean.

[24] The inclusion of these covariates follows the literature, see Fearon and Laitin (2003), Collier and Hoeffler (2004), Montalvo and Reynal-Querol (2005), Collier and Rohner (2008) and Collier *et al.* (2009). See also Blattman and Miguel (2010) for a survey of the stylised facts on civil conflicts and the literature.

[25] Tables B3 and B4 in online Appendix B report the summary statistics and the unconditional correlations between the main variables of interest.

Table 1

The Effect of Pathogen Exposure on Civil Wars

Dependent variable	Civil wars								
	Baseline					Currently endemic	All MHV	Coding from historical data	
Pathogen measure	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Pathogens	0.100***	0.092***	0.150***	0.099***	0.056**	0.103***	0.084***	0.055**	0.085***
	(0.023)	(0.025)	(0.036)	(0.035)	(0.022)	(0.029)	(0.026)	(0.024)	(0.024)
Baseline controls	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Geographic controls	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Region fixed effects	No	Yes	Yes	No	Yes	Yes	Yes	Yes	Yes
Time fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sample	Full	Full	Non-OECD	Africa	No Africa	Full	Full	Full	Full
Observations	1,132	1,132	779	373	741	1,132	1,132	1,132	1,132
Number of countries	117	117	87	42	74	117	117	117	117
Pseudo R^2	0.093	0.266	0.251	0.254	0.351	0.265	0.251	0.233	0.258

Notes. The dependent variable is civil war incidence. The baseline measure of *Pathogens* used in columns (1)–(5) is the number of multi-host vector-transmitted (MHV) diseases that are fatal and have ever been recorded in a country according to information from the GIDEON data base. See text and Table A1 for details. Column (6) applies a recoded measure of disease environment that restricts attention to potentially fatal MHV pathogens that are endemic in a country today. Column (7) replicates the estimation with a recoded measure of disease environment that uses information on all MHV pathogens irrespective of their level of fatality. Columns (8) and (9) replicate the baseline estimates restricting attention to the pathogens for which information is available from the historical data sources before the epidemiological revolution (leishmaniasis, dengue, typhus and trypanosomiasis). Column (8) presents estimates for an index of disease environment that is based on the historical data collected before World War II (see text for details). Column (9) is based on a corresponding index constructed with contemporaneous data from GIDEON. The 'baseline controls' comprise log population density, ethnic polarisation, log GDP per capita, the share of GDP accounted for by primary commodity exports, the percentage of mountainous terrain, an indicator for non-contingent land, and a democracy index. The 'geographic controls' include absolute latitude, the log distance of the country centroid from the nearest coast, the log distance of the country centroid to the nearest navigable river, the ratio of the population within 100 kilometres of ice-free coast relative to the total population, see also the text and Tables A1 and A2 for details. All controls, except regional and period indicators, and binary variables (non-contingent land, democracy), are standardised on the respective estimation sample. Marginal effects of probit estimates, standard errors clustered at country level in parenthesis. ***, **, * indicate significance at 1%, 5% and 10% level, respectively.

unconditional mean. In terms of relative quantitative importance, it is one of the main determinants of civil war.[26]

Columns (3)–(5) replicate the analysis for different sub-samples: column (3) reports the results for the sub-sample of less developed countries that are not members of the OECD, column (4) contains the results for countries located in Africa, and column (5) presents results when restricting the sample to countries outside Africa.[27] The results suggest that the role of the disease environment is not driven by the difference between the more and less developed countries. The effect of the disease environment is larger when restricting attention to non-OECD countries than in the full sample, with an increase of 15 percentage points in the likelihood of conflict incidence for a one-standard deviation increase in the disease index, which implies, compared to an unconditional probability of conflict of 23%, an increase of 65%. The point estimate of 0.099 for the Africa sample implies an increase of around 50% in the incidence of civil war compared to an unconditional probability of 20%. Within Africa the variation in the disease environment is one of the few variables that has a significant effect after including geographical and period controls.[28] The result in column (5) shows that the effect is not restricted to Africa.

2.2. *Robustness of Baseline Results*

The findings are robust to alternative estimation methods, data frequencies and specifications. In particular, the results are qualitatively unaffected by the inclusion of past conflict incidence (lagged by one 5-year period). One argument against including previous conflicts is that this might potentially obscure the effect of the disease environment if the disease environment permanently affects the likelihood of conflict, part of which would then be captured by the past conflict control. On the other hand, if conflicts are ongoing or recurrent, one might be concerned about the omission of the history of conflicts. We find that the effect of exposure to deadly pathogens becomes smaller (from 0.091 to 0.051) but the effect remains statistically significant

[26] A more extensive version of Table 1 is presented in Table B5 in online Appendix B, which reports the results for a specification with the baseline controls, the geographic controls and the region and time fixed effects but without accounting for the effect of the disease environment. The baseline explanatory variables are standardised and the coefficient estimates are directly comparable. The results are similar to the findings in the existing literature. Table B5 also presents the respective results for all countries for which data on civil wars and pathogens, but not all control variables, are available. The inclusion of the disease environment contributes at least as much explanatory power as any of the other explanatory variables. Adding additional controls such as landlocked, navigable rivers, temperature and precipitation leaves the results essentially unchanged but implies substantially smaller sample sizes due to missing information, see Table B6 in online Appendix B for details. Comparing the results to those obtained with the full sample that is not restricted by missing observations in some of the control variables and specifications that only contain time fixed effects or a restrictive set of country-specific geographic controls reveal effects of comparable size. Moreover, the estimation of logit models delivers qualitatively identical results. Detailed results are available upon request.

[27] The sample of non-OECD countries essentially coincides with the sample of former colonies (broadly defined), with few exceptions: for instance, Saudi Arabia or China were not colonised; the US and Canada are OECD countries but used to be colonies. For this sample, the mean number of *Pathogens* is 3.37 with standard deviation 1.39.

[28] For the sample of African countries the number of pathogens is 3.99, with standard deviation 1.35. Besides the role of diseases, another significant determinant of civil war is whether a country is a primary commodity exporter, which is more significant than in the sample with all countries. This could reflect a higher stability of regimes with strong economic interests and capabilities to ensure this stability.

when controlling for conflict incidence in the preceding five-year period.[29] The effects are equivalent to an increase in the likelihood of observing a conflict in the current period of about 30% for a one-standard deviation increase in the disease burden. The drop in the coefficient is consistent with the hypothesis that the disease environment has a long-term effect on conflict incidence, part (but not all) of which might work through the lagged dependent variable. The effect of past conflict is 0.59 and highly significant. This implies that the long-run effect of the exposure to pathogens is very similar in the specifications without and with controls for past conflict.[30] Similar, although somewhat less precisely estimated, results emerge when using a linear regression framework, or when estimating a probit random effects model to account for potential country-specific unobserved heterogeneity.[31] Very similar patterns are obtained with data at yearly frequency or when exploiting only variation across countries and allowing for censoring at zero for those countries that had no civil conflict over the entire observation period.[32]

The results are confirmed for alternative definitions of civil violence, including alternative coding of civil wars, onset and duration of civil wars, lower intensity civil conflicts and struggles for the control of government. The results also show that the disease environment has an even slightly larger effect on the likelihood of observing civil violence at a lower intensity.[33] We find some evidence that the positive effect of a higher exposure to harsher disease environment on conflict is not confined to civil conflict but can also be found at the individual level. In particular, a higher pathogen exposure is associated with a significantly higher number of homicides across countries.[34]

2.3. *Measurement of the Disease Environment*

This subsection explores the role of the measurement of the disease environment in terms of different codings of the presence of each pathogen, different sets of diseases and alternative sources of information based on historical records of the global distribution of diseases in the early twentieth Century.

2.3.1. *Endemic pathogens*

The benchmark measure *Pathogens* exploits information on whether a particular MHV pathogen with a high risk of death has ever been detected in a country from the GIDEON database and delivers a conservative measure of the exposure to these

[29] See online Appendix B Table B7 columns (1)–(2) for details.

[30] The corresponding long-run effect of disease environment is $0.051/(1 - 0.59) = 0.12$.

[31] Detailed results for OLS are displayed in columns (3)–(4) of Table B7. Probit random effects results are shown in columns (5)–(6) of Table B7.

[32] See Table B8 in online Appendix B for details.

[33] Detailed results are reported in Tables B9 and B10. Table B11 reports the results for the role of disease environment for the number of conflicts and the duration of conflicts in terms of the average length of conflicts in a given country.

[34] The results, which are reported in Table B12 in online Appendix B, should be taken as purely suggestive as they are based on noisy and potentially incomplete data from the UNODC homicides statistics. These data are not designed for cross-country comparisons and might therefore not be fully reliable. See also Bros and Couttenier (2015) for related work.

pathogens. As discussed in Section 1, a conceptually different coding involves pathogens that are currently endemic in a country. This also allows accounting for possible eradication of diseases. For MHV pathogens the two indices are very similar, with a correlation above 0.9. This is consistent with the view that socio-economic development and civil conflict do not affect the global distribution of vector-transmitted pathogens. The results in column (6) of Table 1 show that the estimates are very similar when using a measure of pathogen exposure whose coding is based on current endemicity. The effect of 0.103 is even slightly larger.

2.3.2. *All multi-host vector-transmitted diseases*

To study the role of the specific diseases, we also consider the broader set of all MHV diseases ever diagnosed in a country irrespective of their degree of fatality and endemicity. For some of these diseases, eradication and effective control may be more of an issue.[35] Specifically, malaria, which is still one of the main killers worldwide, has been treated successfully or even eradicated in some of the more developed countries. Some non-fatal MHV diseases are observed only infrequently in the more developed countries. To deal with these issues conservatively, we rely on the coding based on the criterion of whether the disease has ever been detected in a country – even if it is currently not endemic or has already been eradicated – when constructing the disease index for all MHV diseases. This criterion implies, for example, that several OECD countries like Belgium, Canada, Denmark, France, Germany and Italy are coded as malaria countries, although the disease has been officially eradicated in these countries since at least the 1960s. The results reported in column (7) of Table 1 deliver a significant positive, and very similar, effect of the disease environment on civil war. Additional robustness checks reveal that the results are not driven by any particular pathogen, suggesting that eradication of single pathogens is unlikely to affect the overall finding.[36]

2.3.3. *Historical disease environment*

The historical sources from the nineteenth and early twentieth centuries allow constructing a comparable measure of the worldwide distribution of MHV diseases. Reverse causality, running from the incidence of internal civil conflicts in the past few decades to the global distribution of pathogens more than a century ago is ruled out by construction. The historical information delivers a picture of the global distribution of these pathogens before World War II, which led to an acceleration of the globalisation of human pathogens as a consequence of the massive movements of troops across the world and was followed by a further acceleration since the 1980s with the intensification of world trade, see Smith *et al.* (2007). The globalisation of infectious diseases mainly involved non-vector transmitted pathogens, however. As consequence, one should expect similar findings even when relying on information about the global

[35] In particular, Filariasis-Brugia Malayi and Onchocerciasis (River Blindness) are observed in relatively few countries and have also been subject to successful campaigns of eradication at the local (or sub-national) level.

[36] The results obtained when excluding each single pathogen from the index are reported in Table B13 in online Appendix B. Using principal components analysis also confirms the findings.

distribution of MHV pathogens in the early twentieth century. However, the historical data have lower coverage and precision than the contemporaneous data (see Section 1). The results in Table 1 column (8) confirm the role of diseases for civil wars. The coefficient estimate of 0.055 is smaller than the estimates for the baseline measure. For comparability, we also estimate the effect for an index that is based on the exact same subset of diseases as the historical index but that is constructed using contemporaneous data from GIDEON. The results in column (9) deliver a very similar point estimate compared to the baseline, 0.085.[37]

Taken together, the estimates obtained with different measures of pathogen exposure provide support for the baseline results on the effect of pathogen exposure for civil violence in the last fifty years. These results suggest that the disease environment, as measured by the count of potentially fatal MHV pathogens has a significantly positive and quantitatively relevant effect on civil conflicts. As a consequence of the specific features of these pathogens and the construction of the measure *Pathogens*, the estimated effect is unlikely to be driven by reverse causality running from civil violence to the global distribution of these diseases, as discussed in Section 1. The cross country variation in the index of pathogen exposure is a conservative proxy for the latent burden of the respective diseases since actual pathogen exposure is likely to be overestimated in more developed countries that have better health facilities.[38] Hence, the measures of pathogen exposure at the extensive margin minimise the risk of reverse causality and the point estimates are likely to be a lower bound of the actual effect of the disease burden.

2.4. *Unobservable Country Specific Heterogeneity*

Given the cross-sectional nature of the data, a relevant concern is the potential existence of some third factor that might drive the results. The inclusion of all relevant determinants of conflicts that have been identified in the recent empirical literature to some extent accounts for this potential confound.[39] This does not necessarily rule out

[37] As in the historical indices, the results in column (9) are also based on the respective subset of disease without distinction between the subtypes. The correlation between the indices constructed with historical and contemporaneous data sources is 0.65. We also performed the analysis with an alternative count index from historical sources by including Filariasis (as non-deadly MHV pathogen), although the data for this disease are taken from different, less comparable and potentially less reliable and precise sources. The results confirm the findings of column (8).

[38] In fact, even in developed countries with advanced health infrastructures but with suitable bio-climatological conditions, like for instance the US South, diseases like dengue and yellow fever are still endemic today. However, despite the failure of full eradication of these pathogens in these countries (at the extensive margin), health infrastructure affects the prevalence of the diseases in the population (at the intensive margin). On the other hand, the exposure to pathogens in countries with unsuitable bio-climatic environments for MHV pathogens, like Afghanistan, is low, even after decades of armed conflicts. Of course, civil conflicts and the disruption of health infrastructure imply a larger disease burden at the intensive margin but this does not affect the pathogen exposure at the extensive margin.

[39] The empirical specifications include the typical controls in the literature, as well as the additional geographic factors that may affect the risk of civil conflicts and region specific fixed effects. Even very extensive specifications that include controls for demographic factors, education, inequality, or colonial history consistently deliver a significant effect of the pathogen exposure on civil conflicts. See Table B14 in online Appendix B for more extensive specifications that include additional control variables. However, since not all variables are available for all countries and time periods, the samples differ across specifications.

the existence of other relevant but unobserved, or omitted, variables that could be correlated with the conflict outcome as well as with the disease environment.

One way to account for systematic unobserved heterogeneity across countries is the estimation of a random effects panel model.[40] Alternatively, a linear probability model was estimated that includes the baseline time-varying controls as well as a full set of year and country dummies. The purpose of this model is investigating whether the disease environment is related to the unobserved cross-country heterogeneity that affects conflict and that is captured by the estimated coefficients for the country dummies. It turns out that the time-invariant cross-country heterogeneity captured by the coefficient estimates for the country dummies is significantly related to the exposure to MHV pathogens, suggesting that the exposure to vector-transmitted pathogens is part of the relevant country-specific factors for violence.[41]

Another strategy is to quantify how strong the effect of unobserved heterogeneity in terms of relevant omitted variables needs to be relative to the controlled heterogeneity in observables in order to eliminate the effect of the variable of interest, following the method suggested by Altonji *et al.* (2005).[42] This procedure essentially involves comparing the point estimate of the effect that is obtained with a parsimonious empirical specification (denoted β^R for describing the restricted specification) to the respective point estimate obtained with a more extensive model (denoted β^F for the full specification). The higher the value of the point estimate that is left when conditioning on additional observable covariates, β^F, and the smaller the difference between the estimate of the restricted and the full model, $\beta^R - \beta^F$, the larger is the influence of unobserved heterogeneity that is needed to explain away (and eliminate) the estimated effect of the disease environment.[43] Table 2 presents comparisons of different specifications to gain insights regarding the sensitivity of the results. Column (1) compares the estimate of the specification of column (1) of Table 1, which only includes the disease environment and time dummies but no further covariates, to the respective coefficient estimate of the effect of pathogen exposure obtained with a full model that contains all covariates typically included in the empirical specifications in the literature (summarised under the heading baseline controls), the further geographic controls and the time and region dummies as in column (2) of Table 1.[44] Column (2) of Table 2 contains the most extensive specification, where the full model accounts for a large set of additional covariates from the literatures on long-term development and on the

[40] Columns (5) and (6) of Table B7 in online Appendix B contain the results from random effects estimates.

[41] Table B15 in online Appendix B presents estimation results for regressions of the country dummies (estimated using data at the yearly frequency and in a specification that includes the time varying controls) on the pathogen exposure as well as the other time-invariant country-specific controls.

[42] Bellows and Miguel (2009) applied this approach to civil conflicts and report the details of the methodology when applied to non-binary measures.

[43] The ratio $\delta = \beta^F/(\beta^R - \beta^F)$ thus quantifies how much stronger the effect of potentially relevant but omitted (or unobservable) variables needs to be, relative to the influence of observable variables that are already included as controls in the regression, in order to explain away the entire effect of the disease environment fully.

[44] The baseline and geographic controls include population density, ethnic polarisation, income per capita, democracy, primary exports, the share of mountainous terrain, non-contingent land, absolute latitude, distance from the coast, log mean distance to the nearest inland navigable river (kilometres) and the population in ice-free land.

Table 2

Using Selection on Observables to Assess the Bias from Unobservables

	Civil wars		
Dependent variable	(1)	(2)	(3)
Pathogens restricted model (β^R)	0.100***	0.097***	0.050***
	(0.023)	(0.025)	(0.013)
Pathogens full model (β^F)	0.092***	0.072**	0.036**
	(0.025)	(0.029)	(0.017)
Full model:			
Baseline controls	Yes	Yes	Yes
Baseline geographic controls	Yes	Yes	Yes
Log country size	No	Yes	Yes
Soil suitability	No	Yes	Yes
Roughness	No	Yes	Yes
Longitude	No	Yes	Yes
Temperature	No	Yes	Yes
Droughts	No	Yes	Yes
Landlocked	No	Yes	Yes
Kilometres navigable rivers	No	Yes	Yes
Population in tropical area	No	Yes	Yes
Ethnic fractionalisation	No	Yes	Yes
Religious polarisation	No	Yes	Yes
Predicted genetic diversity	No	Yes	Yes
Oil and diamonds	No	Yes	Yes
Constraints on the executive	No	Yes	Yes
Civil liberties	No	Yes	Yes
Share of European descendants	No	Yes	Yes
Period fixed effects	Yes	Yes	Yes
Region fixed effects	Yes	Yes	Yes
Past conflicts	No	No	Yes
Observations	1,132	1,076	993
Pseudo R^2 (restricted model)	0.093	0.084	0.458
Pseudo R^2 (full model)	0.266	0.320	0.526

Notes. Dependent variable is civil war incidence. Pathogens are defined as in Table 1. Predicted genetic diversity is included in linearly and as a quadratic. See Table A2 in the Appendix for details on the full set of covariates. In each column, the coefficient β^R of the restricted model corresponds to the point estimate of the effect of the variable '*pathogens*', estimated on the sample of the respective full model in the same column, with the restricted specification of column (1) of Table 1, which only includes the disease environment and time fixed effects as explanatory variables. Pseudo R^2 refers to full specification. Marginal effects of probit estimates, standard errors clustered at country level in parenthesis. ***, **, * indicate significance at 1%, 5% and 10% levels, respectively.

determinants of civil conflicts, and for which information is available for a large number of countries. In particular, this specification includes log country size in square kilometres, soil suitability, roughness, longitude, average temperature and risk of droughts, an indicator for landlocked location and the length of navigable rivers in kilometres. The controls for the population and its composition are extended to include the population in tropical areas and measures of ethnic fractionalisation, religious polarisation and (predicted) genetic diversity. Natural resources and economic and political institutions are accounted for by controlling for the reliance on oil and diamonds, the existence of constraints on the executive, the extent of civil liberties and the share of European descendants as proxy for early

economic institutions.[45] The additional variables are not available for all countries and periods in the data set, so that the sample size is slightly reduced and estimates for the restricted model are slightly different.[46] Finally, the specification in column (3) replicates the same estimates as in column (2) but includes past conflicts as an additional control to reflect the fact that conflicts might be recurrent and history dependent. This extensive full model involves a very demanding specification in terms of the number of controls involved in comparison to the existing literature. This specification exhibits a higher explanatory power and a smaller point estimate of the effect of *Pathogens* than in the other specifications. In terms of long run effects, however, the effect of the disease environment are qualitatively and quantitatively similar to those obtained with specifications that do not include past conflicts as additional regressor.[47]

The comparison between the restricted and the full models in Table 2 implies that any relevant unobserved variables should explain more than two times as much variation compared to all the explanatory variables that are included in the full models but not in the restricted ones, in order to explain away the entire effect of the disease environment.[48] Even though this is not a proof that omitted third factors cannot account for the effect of pathogen exposure, these figures suggest that it is very unlikely that the effect of the disease environment on civil conflicts can be fully attributed to unobserved country-specific heterogeneity.

3. Health Shocks and Conflicts: Within Country Variation

3.1. *Diseases and Weather Shocks*

As discussed in Section 1, the literature in epidemiology has pointed out the role of weather conditions for health and the likelihood of epidemics. In particular, the

[45] See Table A2 for variable description and data sources. Some of these variables have recently been used in empirical work, see, for example, Esteban *et al.* (2012) and Arbatli *et al.* (2013). For brevity, Table 2 reports the full specification that accounts for all these covariates jointly, although we have considered intermediate specifications that only include subsets of covariates. The results are confirmed when excluding variables that are potentially endogenous to social conflicts (like e.g. institutions or civil liberties) that could induce problems of bad controls.

[46] For comparability, the β^R is estimated on the same sample used for the respective full model. The estimate of the restricted and full model is therefore directly comparable within a column, while the point estimates are not exactly comparable across columns (due to the slightly different samples).

[47] We also performed further checks including alternative proxies for rent extraction and institutions (like the share of fuel exports among all exports, information on colonial history, or settler mortality) and for socio-economic conditions (like inequality in terms of the Gini index, male education, social fractionalisation). The inclusion of these variables does not alter the effect of the disease environment and deliver very similar results but they are not available for all countries and their inclusion implies very substantial reductions in the sample size across different specifications thereby making the findings not directly comparable to the results in the full sample.

[48] Recently, Oster (2014) has proposed a procedure to obtain bounds for omitted variable bias affecting coefficient estimates that uses changes in the estimated coefficient when adding observable controls as well as changes in R-squared in the context of linear regression. When applying this methodology in the present context using a linear probability estimation framework instead of probit, the δ for the specifications in columns (1) and (2) is 6.14 and 6.5 respectively. Due to the large explanatory power of past conflict incidence, the estimates of δ in column (3) are smaller when using the methodology by Oster (2014). Still, in the most conservative setting, the δ needed to eradicate any effect is larger than 0.5, suggesting that, after controlling for an extensive set of controls and past conflict, any additional (orthogonal) unobserved heterogeneity would have to contribute around 50% additional variation compared to the included controls (and past conflict), which appears highly implausible.

occurrence of periods of droughts and heat waves favours the reproduction and spread of the vectors and thus affects the exposure to vector-transmitted pathogens. Hence, the disease environment may interact with variation in weather conditions in determining conflict. This subsection complements and extends the previous analysis by investigating the hypothesis that the disease environment affects civil violence also in interaction with extreme weather events.

The analysis exploits year-to-year variation to investigate the possible amplification of the effect of the exposure to vector-transmitted pathogens through weather shocks. In the sample, there are about 600 episodes of droughts and heat waves over a horizon of five decades, with an average duration of two and a half months and a standard deviation of seven months. Of these episodes, 85% last around one month, 10% last more than one and less than six months and a minority (less than 5%) last more than six months. In each year, there is typically at most one episode per country, only in about 6% of cases there are several episodes in a given country and year. As a benchmark, we use a binary indicator variable that takes the value one if at least one such weather shock is observed in a given country and year, and zero otherwise. In the estimation sample, this variable has mean (standard deviation) of 0.143 (0.35) and the correlation with the disease environment in terms of pathogens is 0.16.

The variability in weather conditions offers exogenous variation that can be exploited to study the effect of weather shocks and their interaction with the disease environment using panel data models with country and time fixed effects. The baseline estimation framework for the analysis in this section is given by the linear probability model:

$$Conflict_{i,t} = \delta Droughts_{i,t} + \zeta Droughts_{i,t} \times Pathogens_i + \alpha_i + \eta_t + \Gamma X'_{i,t-1} + \varepsilon_{i,t}, \quad (3)$$

where, as above, the dependent variable $Conflict_{i,t}$ is a binary indicator variable taking value one if a civil war is observed in the country i in year t, and $Droughts_{i,t}$ is a binary indicator variable for the occurrence of a drought or heat wave in that country and year. The specification includes country and time fixed effects, α_i and η_t, to account for time-invariant country-specific characteristics and for common time effects across countries.[49] Given that the variation in weather shocks is country-specific and exogenous to civil conflicts, the effect of weather shocks on conflicts δ is identified in this framework. Apart from this effect, which is interesting *per se*, the coefficient of main interest is the effect of weather shocks in the respective year and country in interaction with the pathogen exposure, ζ. Under the null hypothesis $\zeta = 0$ the occurrence of weather shocks has the same effect on conflicts, irrespective of the disease environment in terms of the exposure to multi-host vector transmitted pathogens. While potential interactions between the pathogen exposure and weather

[49] The earlier empirical literature on the role of exogenous weather shocks on civil conflicts estimated panel specifications with country fixed effects and country-specific time trends, see Miguel *et al.* (2004) and Burke *et al.* (2009). More recent work by Hsiang *et al.* (2011) has uncovered the existence of a year-to-year correlation between weather and conflict on a global scale. Couttenier and Soubeyran (2014) find that the inclusion of country-specific time trends, which fails to account for these global effects, might lead to spurious results and suggest the use of country and year fixed effects. In light of this evidence, we use a specification with country and year fixed effects as benchmark and present estimates of specifications that include country-specific time trends in the robustness analysis.

shocks can be identified in this framework, the main effects of any time-invariant country-specific characteristic, including the time-invariant *Pathogens* variable, are subsumed in the country fixed effects. With the inclusion of country and period fixed effects, the coefficients therefore represent difference-in-difference estimates of the effect of weather shocks and their interaction with the exposure to vector-transmitted pathogens on civil conflicts. The identification relies on the occurrence of the weather shock in a country in a year as treatment and compares it to countries that do not experience such a shock in the same year. A causal interpretation of the coefficient of interest requires exogeneity of the weather shocks, and their interaction with the country-specific disease environment, conditional on all included controls. In some specifications, the vector $\boldsymbol{X}$ includes additional time-varying controls as well as the past occurrence of civil conflicts. As a benchmark, we estimate linear probability models with standard errors that allow for clustering on the country-level, heteroscedasticity and serial autocorrelation.

The results in panel (*a*) of Table 3 suggest that droughts and heat waves are positively related to the likelihood of civil conflict, although the effect is not statistically significant at conventional levels, see columns (1)–(4).[50] Columns (5) and (6) report estimates for specifications that include time-varying controls in terms of income per capita, population, primary exports and democracy. The results are similar to before and basically unaffected by the additional control variables.

Panel (*b*) of Table 3 presents the results for the same specification but extended by an interaction term between weather shocks and the disease environment. The coefficient of this interaction term is positive and highly significant regardless of the specification, indicating that weather shocks amplify the effect of the disease environment on the likelihood of civil conflict.[51]

In terms of quantitative importance, in columns (1), (3) and (5) the interaction effect implies that the probability of conflict increases by about 3.6–5 percentage points for a one-standard deviation increase in the number of pathogens in case of a climatological or meteorological shock (in terms of a drought or heat wave). The long-run effects implied by the results in columns (2), (4) and (6) are comparable to (and even slightly larger than) the estimates from the specifications without past conflict controls.[52] Compared to an unconditional conflict probability of 0.129, this implies an increase of 30–50%, which is comparable to the size of the effect of the disease environment in the baseline estimates of Table 1. Similar results are obtained when specifications with country fixed effects and country-specific time trends are estimated.[53] The results for the sub-samples of less

[50] The statistical significance is slightly higher in specifications with country-specific time trends. Once country and time fixed effects (and, *a fortiori*, time-varying controls and past conflicts) are included, significance drops below conventional levels.

[51] For comparability with the results presented above, Table B16 in online Appendix B contains the corresponding results for estimates based on a panel of five-year intervals. In these specifications, *Droughts* represents the number of droughts or heat waves in a given five-year period. One caveat of using data on conflicts and droughts/heat waves aggregated to five-year intervals is that the timing of events and hence the interpretation of the findings is less clean. Nevertheless, the results are qualitatively identical.

[52] The corresponding long-run effects are $0.067 = 0.017/(1 - 0.747)$, $0.057 = 0.014/(1 - 0.756)$, and $0.045 = 0.013/(1 - 0.711)$, respectively. Notice that bias from lagged dependent variable is expected to be limited due to the comparably long time series dimension of 48 years of observations.

[53] See Table B17 in online Appendix B.

Table 3

The Effect of Pathogen Exposure and Weather Shocks on Civil Conflicts

	Civil wars					
Dependent variable	(1)	(2)	(3)	(4)	(5)	(6)
Panel (a)						
Droughts	0.023	0.011	0.011	0.005	0.011	0.005
	(0.017)	(0.008)	(0.012)	(0.007)	(0.012)	(0.007)
Time-varying controls	No	No	No	No	Yes	Yes
Country fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Time fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Past conflicts controls	No	Yes	No	Yes	No	Yes
Observations	10,202	10,202	5,044	5,044	5,044	5,044
R^2 (within)	0.027	0.572	0.044	0.584	0.051	0.584
Number of countries	214	214	145	145	145	145
Panel (b)						
Droughts	−0.012	−0.001	−0.001	0.001	−0.001	0.001
	(0.015)	(0.006)	(0.010)	(0.006)	(0.011)	(0.006)
Droughts × Pathogens	0.050***	0.017***	0.036***	0.014**	0.038***	0.014**
	(0.015)	(0.007)	(0.013)	(0.007)	(0.013)	(0.007)
Time-varying controls	No	No	No	No	Yes	Yes
Country fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Time fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Past conflicts controls	No	Yes	No	Yes	No	Yes
Observations	10,202	10,202	5,044	5,044	5,044	5,044
R^2 (within)	0.031	0.572	0.046	0.584	0.054	0.584
Number of countries	214	214	145	145	145	145

Notes. The dependent variable is the incidence of a civil war in a given year in a given country. The measure of the disease environment (the variable *Pathogens*) are the same as in columns (1)–(6) of Table 1. *Droughts* refers to the occurrence of droughts or heat waves in a respective country-year cell. See also the text and Table A1. Time-varying controls at yearly frequencies include log GDP per capita, log population density, democracy and share of primary exports. Linear probability model estimates, robust standard errors clustered at country level in parenthesis. All controls are standardised on the estimation sample. ***, **, * indicate significance at 1%, 5% and 10% levels, respectively.

developed countries in Africa, Asia and Latin America, or of only African countries deliver point estimates of the interaction of the disease environment with weather shocks that are similar to those for the full sample, presented in panel (*b*) of Table 3.[54]

3.1.1. *Robustness*

We performed several robustness checks with even more extensive empirical specifications. Controlling for temperature and precipitation as well as their interaction with pathogen exposure delivers virtually unchanged results. Considered in isolation, precipitation has a negative and significant effect. However, these results are not robust to different specifications. The most robust result remains the interaction between droughts or heat waves and pathogen exposure, which significantly affects conflicts in all specifications. Even the most extensive (and statistically demanding) specification with lags and leads of the weather variables (and their interactions with

[54] Detailed results reported in Tables B18 and B19.

pathogen exposure) yield a statistically significant and quantitatively very similar effect of the interaction between climatic shocks and pathogen exposure on conflicts.[55] Robustness tests also reveal that the results are not driven by co-factors, such as HIV prevalence, which could have an influence because HIV-infected individuals have a weakened immune system and are more susceptible to other infections.[56]

3.2. *The Channel: Health Shocks*

The results so far qualify the baseline findings and suggest that the occurrence of weather shocks that favour the spread of infected vectors amplifies the effect of the exposure to harsher disease environments on conflict. In this subsection, we move one step further in the investigation of the channel behind the interaction between weather shocks and pathogen exposure. We explore the possibility of interactions between other country-specific characteristics and weather shocks above and beyond the role of the disease environment and then study epidemics as the potential channel.

3.2.1. *Other country-specific characteristics*

The results in the preceding subsection are compatible with the existence of a health-related effect in the short run. To be a compelling interpretation of the findings, the interaction between weather shocks and disease environment should be robust to the inclusion of additional interactions between weather shocks and other country-specific characteristics. For instance, the literature in ecology has found evidence for the gradient theory of bio-diversity, which suggests that absolute latitude is a central determinant of global bio-diversity, also in terms of pathogens. Hence, if the interaction between pathogen exposure and weather shocks is to reflect a health channel and not a finding that relates to other aspects of bio-diversity or geography, the results should be unaffected by the inclusion of additional interactions of weather shocks with absolute latitude. A similar reasoning could be made for other relevant country-specific characteristics, like population shares living in the tropics, land suitability for agriculture, terrain features, ethnic polarisation, or historical institutions which might interact with short-term weather shocks in triggering civil violence.

To investigate this issue, the baseline specification of Table 3 is extended to the inclusion of interaction terms between weather shocks and other country-specific characteristics. Table 4 presents the results of horse races of the interaction between weather shocks and disease exposure and other time-invariant characteristics using extended specifications that include interactions of droughts/heat waves with absolute latitude, the population in tropical areas, land suitability, the share of mountainous terrain, ethno-linguistic polarisation and the extent of the constraints on the executive.

[55] See Table B20 in online Appendix B for details.

[56] In particular, controlling for HIV prevalence leaves the main results unaffected as documented by the results reported in Table B21 in online Appendix B, where HIV prevalence is coded as zero if corresponding data are missing (mostly in the period before the 1980s when HIV was not yet known). Unreported results obtained with a sample restricting to non-missing HIV prevalence data are similar. However, these estimates come under the caveat that the inclusion of HIV prevalence may create problems of bad controls due to the possible endogeneity of HIV as consequence of civil conflicts.

Table 4

Disease Environment and Weather Shocks: Alternative Interactions

	Civil wars					
Dependent variable	(1)	(2)	(3)	(4)	(5)	(6)
Droughts	−0.001	0.001	−0.002	−0.001	0.001	0.001
	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)
Droughts × Pathogens	0.016**	0.015**	0.018**	0.015**	0.016**	0.017**
	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.008)
Droughts ×						
× Absolute latitude	Yes	No	No	No	No	No
× Population in tropical areas	No	Yes	No	No	No	No
× Land suitability	No	No	Yes	No	No	No
× Mountains	No	No	No	Yes	No	No
× Ethnic polarisation	No	No	No	No	Yes	No
× Constraints on the executive	No	No	No	No	No	Yes
Time-varying controls	Yes	Yes	Yes	Yes	Yes	Yes
Country fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Time fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Past conflicts controls	Yes	Yes	Yes	Yes	Yes	Yes
Observations	5,039	4,862	4,771	4,855	4,855	5,012
R^2 (within)	0.583	0.583	0.587	0.587	0.587	0.583
Number of countries	145	139	136	118	118	144

Notes. The dependent variable is the same as in Table 3, the measure of the disease environment (the variable *Pathogens*) is the same as in columns (1)–(6) of Table 1. *Droughts* refers to the occurrence of droughts or heat waves in a respective country-year cell. See also the text and Tables A1 and A2 for a description of variables of interest, covariates and data sources. Time-varying controls at yearly frequencies include log GDP per capita, log population density, democracy and share of primary exports. Linear probability model estimates, robust standard errors clustered at country level in parenthesis. All controls are standardised on the estimation sample. ***, **, * indicate significance at 1%, 5% and 10% levels, respectively.

The results show that the effect of the interaction between weather and the disease environment is robust both qualitatively and quantitatively to the inclusion of interactions of climatological and meteorological shocks with other country-specific characteristics.[57] These findings also suggest that the interaction results specifically relate to the exposure to pathogens and not to some diffuse geographic features or other country-specific characteristics. None of the (unreported) alternative interactions of droughts/heat waves with other country-specific characteristics displays significant effects.[58] The only exception is a positive and marginally significant interaction between droughts/heat waves and the share of mountainous terrain.

[57] Due to space constraints, Table 4 only reports the most extensive specification (corresponding to column (6) of Table 3) for selected variables that proxy for geography, soil suitability for agriculture, tensions in the population and poor institutions. The results are quantitatively and qualitatively identical for more parsimonious panel specifications and when including interactions of weather shocks with any of the country-specific characteristics that are contained in the extensive set of control variables in Table 2.

[58] The same applies to the other country-specific characteristics that are controlled for in Table 2. The findings are also confirmed when restricting attention to developing and African countries. The results are also confirmed when including interactions between droughts/heat waves and the time varying controls (log GDP per capita, log population density, democracy, and share of primary exports). This suggests that the effect is not driven by factors like state collapse.

3.2.2. *Epidemics and other channels*

The existence of an interaction between the exposure to MHV pathogens and weather shocks is consistent with the weather-sensitivity of vectors documented in the medical literature discussed in subsection 1.1. This implies that weather shocks may be associated with local and temporary variations in health conditions and, particularly, with the outbreak of epidemics caused by vector-transmitted infectious agents. To investigate this hypothesis, we use data on the occurrence of epidemics related to vector-transmitted and weather sensitive pathogens in each country and year. The occurrence of epidemics is coded as a dummy variable that equals one if there is at least one epidemic related to vector-transmitted and heat-sensitive diseases in a given country and year. The mean (standard deviation) of these epidemics variable is 0.121 (0.326).

Figure 2 provides a visual illustration of the correlation between pathogen exposure in terms of the count index of MHV pathogens and the average incidence of epidemics per year. The epidemics data reveal a strong, positive correlation between the measures of pathogen exposure, around 0.6. The data also document that, as one would expect, the number of MHV at the extensive margin is informative about the fact that countries with few MHV pathogens face a lower average and maximum number of epidemics than countries with many MHV pathogens. Within each bin of MHV pathogen prevalence, some of the countries with many MHV pathogens exhibit fewer while others exhibit more epidemics but this, as discussed below, might in itself be related to conflict incidence.

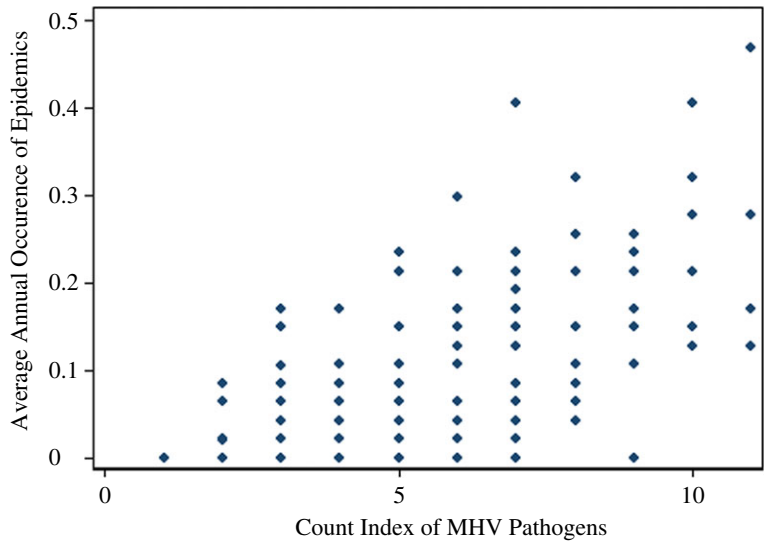

Fig. 2. *Disease Environment and Epidemics*

Note. The Figure depicts the unconditional relation between the count index of of multi-host vector-transmitted (MHV) pathogens and the average annual occurrence of epidemics. See text for details. Colour figure can be viewed at wileyonlinelibrary.com

Table 5

Disease Environment, Weather Shocks, and the Epidemics Channel

	Epidemics					
Dependent variable	(1)	(2)	(3)	(4)	(5)	(6)
Panel (*a*)						
Droughts	0.031**	0.031**	0.028**	0.028*	0.027*	0.027*
	(0.015)	(0.015)	(0.014)	(0.014)	(0.014)	(0.014)
Droughts × Pathogens	0.042***	0.036**	0.046***	0.045***	0.046***	0.045***
	(0.015)	(0.015)	(0.017)	(0.017)	(0.017)	(0.017)
Country fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Time fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Time-varying controls	No	No	No	No	Yes	Yes
Past conflict controls	No	Yes	No	Yes	No	Yes
Observations	9,988	9,774	4,916	4,916	4,916	4,916
Adj. R^2	0.077	0.079	0.101	0.103	0.114	0.115
Number of countries	214	214	145	145	145	145
	Civil wars					
Dependent variable	(1)	(2)	(3)	(4)	(5)	(6)
Panel (*b*)						
Droughts	−0.034	−0.005	−0.014	−0.004	−0.015	−0.004
	(0.030)	(0.012)	(0.019)	(0.008)	(0.019)	(0.008)
Epidemics (instrumented)	0.995**	0.299*	0.644*	0.249*	0.685**	0.258*
	(0.487)	(0.190)	(0.343)	(0.136)	(0.345)	(0.133)
Country fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Time fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Time-varying controls	No	No	No	No	Yes	Yes
Past conflict controls	No	Yes	No	Yes	No	Yes
Observations	9,988	9,774	4,915	4,915	4,915	4,915
Number of countries	214	214	144	144	144	144
First stage F (AR)	7.56	5.70	7.65	7.24	7.23	6.93
p-value	0.006	0.017	0.006	0.008	0.008	0.009

Notes. Panel (*a*): linear probability model. The dependent variable is a binary indicator taking value one if an epidemic caused by vector-transmitted heat sensitive pathogens is observed in a particular country and year. Panel (*b*): 2SLS linear probability model estimates. The dependent variable of the outcome equation is a binary indicator taking value one if a civil war is observed in a particular country and year as in Table 1. The instrumented variable is a binary indicator taking value one if an epidemic caused by vector-transmitted heat sensitive pathogens is observed in a particular country and year. The instrument is the interaction *Droughts* × *Pathogens*. The variable *Pathogens* is the count index for all MHV pathogens as in column (7) of Table 1. *Droughts* refers to the occurrence of droughts or heat waves in a respective country-year cell. Time-varying controls include the same variables as in the baseline time-varying controls: log population density, log GDP per capita, the share of GDP of primary commodities, and democracy. Past conflict controls include conflicts in the previous year. See also the text and Tables A1 and A2 for a description of variables of interest and data sources. Robust standard errors clustered at country level in parenthesis. All controls are standardised on the estimation sample. ***, **, *indicate significance at 1%, 5% and 10% level, respectively.

The results for regressions of epidemics as the dependent variable are presented in panel (*a*) of Table 5. The estimates document that droughts and heat waves are positively and significantly associated with the outbreaks of epidemics. It turns out that particularly the interaction between weather shocks and the exposure to MHV pathogens in a country materialises in a higher prevalence of epidemics, indicating

that the disease environment amplifies the effect of weather. The effect is quantitatively larger and statistically more robust than the main effect.[59] The interaction between weather shocks and the disease burden is essentially unaffected by the inclusion of time-varying controls. The results therefore confirm the hypothesis that weather shocks and disease environment have a strong interaction in causing epidemics. This conjecture has been put forward in the medical literature but has, to our knowledge, not been documented with high frequency cross-country panel data.[60] In addition, it is worth to mention that, inline with what one would expect, population density exhibits a significantly positive effect on epidemics and GDP per capita has a significantly negative effect.

According to the medical literature, the short-term health consequences of a weather shock in a harsh disease environment have many manifestations and therefore are unlikely to be confined to the outbreak of epidemics for which data are available. Nonetheless, it is interesting to consider the role of epidemics as a potential channel by means of a two-stage estimation procedure. Panel (*b*) of Table 5 presents the results obtained from 2SLS estimates, in which epidemics are instrumented using the interaction between weather shocks and the pathogen index as instrument, and for which the specifications in panel (*a*) effectively represent the corresponding first stages. The results suggest that the reduced-form effect of the interaction effect between droughts/heat waves and the exposure to MHV pathogens on conflict works at least in part through epidemics.

The interactions between the disease environment and weather shocks might affect conflict also through alternative channels. Although no specific hypothesis has been put forward in the literature, one could, for instance, conjecture that weather shocks in interaction with a high disease prevalence might be particularly harmful for economic living conditions in countries. Likewise, weather shocks in high disease countries might lead to more pronounced variations in population density, or might affect political participation. To explore these potential alternative channels, Table 6 presents the results of regressions of income per capita, population density, democracy and primary exports on the variation in disease exposure through weather shocks. These specifications thus provide a sort of over-identification test for the health channel. The results reveal no evidence for a significant effect of weather shocks and of the interaction between weather shocks and pathogen exposure on these variables.

A final investigation that can be informative about the channel concerns the existence of alternative interactions between the disease environment and other possible short-term triggers of conflicts in terms of economic conditions, demographic patterns and political institutions. Table 7 reports the results for specifications that include interactions between the disease environment and other time-varying variables such as income per capita, population density, democracy and primary exports. Besides

[59] In light of the construction of epidemics related to vector-transmitted, heat-sensitive diseases, the use of the broader count index based on multi-host vector-transmitted diseases seems more appropriate. Corresponding results for the more restrictive index based on potentially deadly MHV diseases that is used in the previous Tables can be found in online Appendix B, see Table B22.

[60] Consistent with the interpretation of a potential channel, adding the lagged incidence of conflict as additional controls in the baseline specifications of Tables 1 and 3 leaves the main results on the disease burden unaffected but implies a slightly smaller effect of the interaction between weather and pathogens.

Table 6

The Effect of Disease Environment and Weather Shocks on Other Outcomes

	Log GDP per capita		Log population density		Democracy		Primary exports	
Dependent variable	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Droughts	−0.038	−0.013	−0.017	−0.020	0.021	0.001	−0.001	0.001
	(0.031)	(0.010)	(0.025)	(0.022)	(0.024)	(0.009)	(0.004)	(0.001)
Droughts × Pathogens	0.036	0.006	0.017	0.006	0.009	0.001	0.003	0.001
	(0.035)	(0.010)	(0.015)	(0.006)	(0.023)	(0.009)	(0.004)	(0.001)
Time-varying controls	No	Yes	No	Yes	No	Yes	No	Yes
Country fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	5,036	5,036	5,038	5,038	5,035	5,035	5,014	5,014
Adj. R^2	0.745	0.965	0.508	0.685	0.140	0.736	0.068	0.818
Number of countries	145	145	145	145	145	145	145	145

Notes. The measure of the disease environment (the variable *Pathogens*) is the same as in Table 1. *Droughts* refers to the occurrence of droughts or heat waves in a respective country-year cell. See also the text and Tables A1 and A2 for a description of variables of interest, covariates and data sources. Time-varying controls at yearly frequencies include log GDP per capita, log population density, democracy and share of primary exports. Linear probability model estimates, robust standard errors clustered at country level in parenthesis. All controls are standardised on the estimation sample. ***, **, * indicate significance at 1%, 5% and 10% levels, respectively.

Table 7

Disease Environment and Weather Shocks: Alternative Interactions Robustness

	Civil wars					
	(1)	(2)	(3)	(4)	(5)	(6)
Droughts	−0.003	0.001	0.001	−0.001	0.001	−0.001
	(0.011)	(0.006)	(0.006)	(0.010)	(0.006)	(0.006)
Droughts × Pathogens	0.034***	0.013*	0.013**	0.032***	0.013*	0.013**
	(0.012)	(0.007)	(0.007)	(0.011)	(0.007)	(0.007)
Time varying controls:						
Log GDP per capita	−0.054*	−0.012	−0.012			−0.013
	(0.031)	(0.010)	(0.011)			(0.011)
Log GDP per capita × Pathogens	0.017	0.006	0.007			
	(0.018)	(0.005)	(0.006)			
Log population density			−0.003	0.054	0.017	0.003
			(0.015)	(0.051)	(0.014)	(0.015)
Log population density × Pathogens				0.041	0.011	0.008
				(0.058)	(0.015)	(0.016)
Democracy			−0.005			−0.004
			(0.005)			(0.005)
Primary exports			−0.002			−0.003
			(0.005)			(0.005)
Time fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Country fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Past conflicts controls	No	Yes	Yes	No	Yes	Yes
Observations	5,044	5,044	5,044	5,044	5,044	5,044
R^2 (within)	0.052	0.584	0.584	0.048	0.584	0.584
Number of countries	145	145	145	145	145	145

Notes. The dependent variable is the same as in Table 3; the disease environment (the variable *Pathogens*) is the same as in Table 1. *Droughts* refers to the occurrence of droughts or heat waves in a respective country-year cell. See also the text and Tables A1 and A2 for a description of variables of interest, covariates and data sources. Time-Varying Controls at yearly frequencies include log GDP per capita, log population density, democracy, and share of primary exports as indicated. Linear probability model estimates, robust standard errors clustered at country level in parenthesis. All controls are standardised on the estimation sample. ***, **, * indicate significance at 1%, 5% and 10% level, respectively.

confirming the existence of a negative correlation between income per capita and conflicts, which has been documented in the literature, there is no evidence of other significant interactions.

4. Concluding Remarks

This article provides a first systematic investigation of the hypothesis that countries with a high and persistent exposure to (potentially deadly) infectious diseases face a higher risk of violent civil conflicts. The investigation is based on a new index of the worldwide distribution of pathogens in the class of MHV infectious diseases.

The first part of the empirical investigation exploits exogenous differences in the disease environment across countries. The analysis contributes to the growing empirical literature on the determinants of civil conflicts by providing evidence for a potentially relevant channel that complements the economic, social and institutional factors that have been documented previously. The results suggest an important role of the disease environment for civil conflicts, which has not been investigated in the

existing literature. The second part presents evidence from within-country variation by exploiting an interaction with exogenous year-to-year variation in weather conditions. The findings indicate that the so-measured disease environment has an effect on civil conflict incidence also by amplifying the effect of shocks in weather conditions, such as droughts and heat waves. The analysis also reveals evidence for the effect working through a health-related channel, whereas there is no evidence that the effect works through other channels related to income, population, or institutions. The evidence delivers a new rationale for the warnings issued in the medical literature and by international organisations, like the WHO, about the need to intensify the fight against these diseases, and provides a novel and potentially useful piece of information that may be relevant for development programmes.

The findings suggest some promising directions for future research. The analysis so far has exploited year-to-year health shocks at the country level but without relating these shocks explicitly to global warming. A systematic investigation of the role of heat sensitive epidemic pathogens for the effects of climate change on a global scale for civil conflicts is still missing. Such an investigation may add a new relevant facet to the ongoing debate on the consequences of global warming for conflict.[61] In this article, we have collected and exploited data on the global, cross-country, distribution of human pathogens. The hypothesis that poor health conditions, or negative health shocks, might reduce the opportunity costs of violence is not confined to the level of countries, however. The results call for the collection of data at a more disaggregated level. Some recent contributions have proposed the use of disaggregated data for the analysis of the determinants of civil violence.[62] Devising empirical strategies to perform the investigation of the role of exposure to pathogens and health shocks at a more disaggregate level appears a natural direction for further research.

Appendix A. Data Sources and Description of Variables

Table A1

Data Sources and Description of Main Variables of Interest

Variable description and data sources
Pathogens
Baseline measure. Number of MHV (Multi-host Vector-Transmitted) diseases with a significant risk of death that have ever been detected in a country. The included diseases are: dengue, yellow fever, leishmaniasis visceral, relapsing fever, typhus epidemic, trypanosomiasis africanis, relapsing fever. The variable is standardised on the estimation sample. Data source: Global Infectious Disease and Epidemiology Network, GIDEON (http://www.gideononline.com/)

[61] On the role of climate change for health conditions, see e.g. McMichael *et al.* (2006). The existence of a link between climate change and civil conflicts is intensely debated, see, e.g. Burke *et al.* (2009), Sutton *et al.* (2010), Buhaug (2010) and Hsiang *et al.* (2011); see also Hsiang *et al.* (2013).

[62] Harari and La Ferrara (2012) and Michalopoulos and Papaioannou (2013) investigate the determinants of civil conflict using cell-level panel data for African countries, and Rohner *et al.* (2013), Amodio and Chiovelli (2014), Berman *et al.* (2014) and Caselli *et al.* (2015) present studies of particular conflicts in Africa.

Table A1
(*Continued*)

Variable description and data sources
Coding based on endemicity. Number of MHV (multi-host vector-transmitted) diseases with a significant risk of death that are currently endemic in a country. Pathogens are the same as in the baseline measure and variable is standardised on the estimation sample. Data Source: Same as Baseline Measure
Coding based on all MHV. Index of Pathogens recording the number of all MHV disease agents that have ever been detected or that are endemic in a country. The included diseases are: angiomatoses, malaria, onchocerciasis, dengue, yellow fever, leishmaniasis (all types), relapsing fever, typhus epidemic, trypanosomiasis African and American, filariasis brugia-malay, thyphus-epidemic. Data source: Same as baseline measure
Coding from historical data sources. Index of Pathogens recording the number of MHV (multi-host vector-transmitted) diseases that have been detected in historical sources from the mid 19th and early 20th centuries. The index includes typhus, dengue, tripasonomiasis, leishmaniasis. Data sources: Craig and Faust (1943), Simmons *et al.* (1944), and Rodenwald and Bader (1961), see also Murray and Schaller (2010)
Weather shocks and epidemics
Droughts and heat waves. Number of episodes of droughts and heat waves in each year in each country for the period 1960–2006. Data source: EM-DAT: OFDA/CRED International Disaster Database http://www.emdat.be/database)
Epidemics. Number of outbreaks of epidemics of vector transmitted and heat sensitive pathogens in each country and year for the period 1960-2006. Data Sources: EM-DAT: OFDA/CRED International Disaster Database (http://www.emdat.be/database) and Global Infectious Disease and Epidemiology Network, GIDEON (http://www.gideononline.com/)
Measures of civil violence
Baseline: Civil wars. Incidence of civil conflicts with at least 25 battle-related deaths per year and more than 1,000 battle-related deaths over the entire duration of the conflict) for the period 1960–2011. The data are recoded on the respective temporal reference period (5-year panel, yearly frequency, cross section). Data include Type 3 and 4 of the PRIO definition (internal and internationalised internal conflicts), see http://www.pcr.uu.se/research/ucdp/datasets/ for a data description. Data source: UCDP/PRIO Armed Conflict Dataset v.4-2012, 1946–2012, based on Gleditsch *et al.* (2002) and updated by Harbom and Wallensteen (2010)
Intermediate intensity civil conflicts. Incidence of civil conflicts with at least 25 battle-related deaths per year, but not necessarily more than 1,000. Same as baseline measure
Conflicts for Government. Incidence of Civil Wars (see description above) specifically aimed at the control of Government for the period 1960–2011. See http://www.pcr.uu.se/research/UCDP/data_and_publications/datasets.htm for a data description. Same as baseline measure
Coding from correlates of war. Intra-state wars within the recognised territory of a state between a government and non-government forces (civil war), or at least two non-government forces (inter-communal war). Wars involve sustained combat resulting in a minimum of 1,000 battle-related combatant fatalities within a twelve month period without reference to a minimum number of deaths per year. Conflicts with international actors involved in the war are excluded. Data source: Correlates of War Intra-State War Database (v4.0) (http://www.correlatesofwar.org/ datasets.htm)
Coding from Doyle and Sambanis. Civil Conflicts according to the classification by Doyle and Sambanis (2000). Data source: Doyle and Sambanis (2000)
Coding from Fearon and Laitin. Civil Conflicts according to the classification by Fearon and Laitin (2003). Data source: Fearon and Laitin (2003)
Homicides. Average number of Homicides (in thousands) over the period 1995–2011. Data source: UNODC Homicide Statistics (https://www.unodc.org/gsh)

Table A2

Data Sources and Description of Variables: Covariates

Variable and description and data sources
Baseline controls
Log population size: Log of the population at the beginning of the period. Data sources: Penn World Tables 5.6 and Global Development Network Growth Database (World Bank)
Log GDP per capita. Log of real GDP per capita of the initial period (at 1985 international prices). Data sources: Penn World Tables 5.6 and Global Development Network Growth Database (World Bank)
Ethnic polarisation. Index of Ethno-linguistic Polarisation. Data source: Montalvo and Reynal-Querol (2005) and Collier *et al.* (2009)
Primary commodity exports. Primary commodity exports as proportion of GDP. Data source: Global Development Network Growth Database (World Bank)
Log country size: Log of country size in square kilometres. Data source: Physical Geography and Population Data Set, Center for International Development at Harvard University
Mountainous terrain: Percent mountainous terrain. Data source: Physical Geography and Population Data Set, Center for International Development at Harvard University
Non-contingent land: Indicator for non-contingent areas. Countries with territory holding at least 10,000 people and separated from the land area containing the capital city either by land or by 100 kilometres of water were coded as 'noncontiguous'. Data source: Physical Geography and Population Data Set, Center for International Development at Harvard University
Democracy: Democracy score. General openness of the political institutions. Data sources: Polity IV database (2007) and Collier *et al.* (2009)
Geographic controls
(Absolute) Latitude: Data source: CIA World Factbook
Longitude Data source: Physical Geography and Population Data Set, Center for International Development at Harvard University
Log distance of the country centroid to the nearest coast: Data source: Physical Geography and Population Data Set, Center for International Development at Harvard University
Log distance of the country centroid to the nearest navigable river Data source: Physical Geography and Population Data Set, Center for International Development at Harvard University
Ratio of the population within 100 kilometres of ice-free coast relative to the total population: Data source: Physical Geography and Population Data Set, Center for International Development at Harvard University
Percentage of population living in the tropics: Data source: Ashraf and Galor (2013)
Land suitability: geospatial index of the suitability of land for agriculture based on indicators of climate suitability. Data source: Ashraf and Galor (2013)
Elevation: mean elevation of a country in kilometres above sea level, calculated using geospatial elevation data. Data source: Ashraf and Galor (2013)
Roughness: Average degree of terrain roughness across the grid cells within a country. Data source: Ashraf and Galor (2013)
Temperature: Spatial mean of average monthly temperature of a country in degrees Celsius. Data source: Physical Geography and Population Data Set, Center for International Development at Harvard University
Precipitation: Spatial mean of average monthly precipitation of a country in millimetres per square meter. Data source: Physical Geography and Population Data Set, Center for International Development at Harvard University
Additional covariates
Religious polarisation. Index of. Data source: Montalvo and Reynal-Querol (2005)
Ethnic fractionalisation. Index of. Data source: Alesina *et al.* (2003)
Oil and diamond. Dummy variable for the presence of diamonds and oil production in the country. Data source: Esteban *et al.* (2012)

Table A2
(*Continued*)

Variable and description and data sources
Constraints on the executive: 1960-2000 mean of the Index reported by the Polity IV dataset measuring the institutionalised constraints on the decision making power of chief executives. Data source: Ashraf and Galor (2013)
Civil liberties, Index. Data source: Esteban *et al.* (2012)
Share of European descendants: Share of the population whose ancestors lived in Europe in 1500. Data Source: Putterman and Weil (2010)
Predicted genetic diversity: Genetic diversity, prediction based on migratory distance from East Africa. Data source: Ashraf and Galor (2013)

University of Bologna, IZA, CESifo
LMU Munich, IZA, CEPR, CESifo
University of Bologna

Submitted: 30 January 2015
Accepted: 17 March 2016

Additional Supporting Information may be found in the online version of this article:

Appendix B. Technical Appendix and Additional Material.
Data S1.

References

Adda, J. and Lechene, V. (2013). 'Health selection and the effect of smoking on mortality', *Scandinavian Journal of Economics*, vol. 115(3), pp. 902–31.
Alesina, A., Devleeschauwer, A., Easterly, W., Kurlat, S. and Wacziarg, R. (2003). 'Fractionalization', *Journal of Economic Growth*, 8(3), 155–94.
Alsan, M. (2015). 'The effect of the TseTse fly on African development', *American Economic Review*, vol. 105(1), pp. 382–410.
Altonji, J.G., Elder, T.E. and Taber, C.R. (2005). 'Selection on observed and unobserved variables: assessing the effectiveness of Catholic schools', *Journal of Political Economy*, vol. 113(1), pp. 151–84.
Amodio, F. and Chiovelli, G. (2014). 'Ethnicity, migration, and conflict: evidence from contemporary South Africa', mimeo, Universitat Pompeu Fabra, Barcelona.
Arbatli, C.E., Ashraf, Q. and Galor, O. (2013). 'The nature of civil conflict', Working Paper No. 2013-15, Department of Economics, Brown University.
Ashraf, Q. and Galor, O. (2013). 'The "out of Africa" hypothesis, human genetic diversity, and comparative economic development', *American Economic Review*, vol. 103(1), pp. 1–46.
Aspinwall, L.G. (2005). 'The psychology of future-oriented thinking: from achievement to proactive coping, adaptation, and aging', *Motivation and Emotion*, vol. 29(4), pp. 203–35.
Becker, G.S. and Mulligan, C.B. (1997). 'The endogenous determination of time preference', *Quarterly Journal of Economics*, vol. 112(3), pp. 729–58.
Bellows, J. and Miguel, E. (2009). 'War and local collective action in Sierra Leone', *Journal of Public Economics*, vol. 93(11), pp. 1144–57.
Berman, N., Couttenier, M., Rohner, D. and Thoenig, M. (2014). 'This mine is mine! How minerals fuel conflicts in Africa', Research Paper No. 141, OXCarre.
Blattman, C. and Miguel, E. (2010). 'Civil war', *Journal of Economic Literature*, vol. 48(1), pp. 3–57.
Bros, C. and Couttenier, M. (2015). 'Untouchability, homicides and water access', *Journal of Comparative Economics*, vol. 43(3), pp. 549–58.

Buhaug, H. (2010). 'Climate not to blame for African civil wars', *Proceedings of the National Academy of Sciences*, vol. 107(38), pp. 16477–82.
Burke, M.B., Miguel, E., Satyanath, S., Dykema, J.A. and Lobell, D.B. (2009). 'Warming increases the risk of civil war in Africa', *Proceedings of the National Academy of Sciences*, vol. 106(49), pp. 20670–4.
Caselli, F., Morelli, M. and Rohner, D. (2015). 'The geography of inter-state resource wars', *Quarterly Journal of Economics*, vol. 130(1), pp. 267–315.
Cervellati, M. and Sunde, U. (2011). 'Life expectancy and economic growth: the role of the demographic transition', *Journal of Economic Growth*, vol. 16(2), pp. 99–133.
Cervellati, M. and Sunde, U. (2013). 'Life expectancy, schooling, and lifetime labor supply: theory and evidence revisited', *Econometrica*, vol. 81(5), pp. 2055–86.
Cervellati, M., Sunde, U. and Valmori, S. (2012). 'The distribution of infectious diseases and extrinsic mortality across countries', *Mathematical Population Studies*, vol. 19(2), pp. 73–93.
Collier, P. and Hoeffler, A. (2004). 'Greed and grievance in civil war', *Oxford Economic Papers*, vol. 56(4), pp. 563–95.
Collier, P. and Rohner, D. (2008). 'Democracy, development, and conflict', *Journal of the European Economic Association*, vol. 6(2), pp. 531–40.
Collier, P., Hoeffler, A. and Rohner, D. (2009). 'Beyond greed and grievance: feasibility and civil war', *Oxford Economic Papers*, vol. 61(1), pp. 1–27.
Couttenier, M. and Berman, N. (2015). 'External shocks, internal shots: the geography of civil conflicts', *Review of Economics and Statistics*, vol. 97(4), pp. 758–76.
Couttenier, M. and Soubeyran, R. (2014). 'Drought and civil war in Sub-Saharan Africa', Economic Journal, vol. 124(575), pp. 201–40.
Couttenier, M. and Soubeyran, R. (2015). 'A survey of the causes of civil conflicts: natural factors and economic conditions', *Revue Economie Politique*, vol. 125(6), pp. 787–810.
Craig, C.F. and Faust, E.C. (1943). *Clinical Parasitology*, 3rd edn, Philadelphia, PA: Lea & Febinger.
Diamond, J. (1997). *Guns, Germs, and Steel*, New York: WW Norton.
Doyle, M.W. and Sambanis, N. (2000). 'International peacebuilding: a theoretical and quantitative analysis', *American Political Science Review*, vol. 94(4), pp. 779–802.
Esteban, J.M., Mayoral, L. and Ray, D. (2012). 'Ethnicity and conflict: an empirical investigation', *American Economic Review*, vol. 102, 1310–42.
Falk, A., Becker, A., Dohmen, T., Enke, B., Huffman, D. and Sunde, U. (2015). 'The nature and predictive power of preferences: global evidence', Discussion Paper, University of Bonn.
Fearon, J.D. and Laitin, D.D. (2003). 'Ethnicity, insurgency, and civil war', *American Political Science Review*, vol. 97(1), pp. 75–90.
Gleditsch, N.P., Wallensteen, P., Eriksson, M., Sollenberg, M. and Strand, H. (2002). 'Armed conflict 1946–2001: a new dataset', *Journal of Peace Research*, vol. 39(5), pp. 615–37.
Gopinath, R., Ostrowski, M., Justement, S.J., Fauci, A.S. and Nutman, T.B. (2000). 'Filarial infections increase susceptibility to human immunodeficiency virus infection in peripheral blood mononuclear cells in vitro', *Journal of Infectious Diseases*, vol. 182(6), pp. 1804–8.
Goudie, R.J., Mukherjee, S., De Neve, J.E., Oswald, A.J. and Wu, S. (2014). 'Happiness as a driver of risk avoiding behavior', *Economica*, vol. 81(324), pp. 674–97.
Gubler, D.J. (1991). 'Insects in disease transmission', in (G.T. Strickland, ed.), *Hunter's Tropical Medicine and Emerging Infectious Diseases*, 7th edn, pp. 981–1000, Philadelphia: W.B. Saunders.
Guernier, V., Hochberg, M. and Guegan, J.-F. (2004). 'Ecology drives the world-wide distribution of diseases', *PLoS Biology*, vol. 2, pp. 740–6.
Guiso, L., Sapienza, P. and Zingales, L. (2013). 'Time varying risk aversion', Discussion Paper, National Bureau of Economic Research.
Harari, M. and La Ferrara, E. (2012). 'Conflict, climate, and cells: a disaggregated analysis', Working Paper No. 461, IGIER.
Harbom, L. and Wallensteen, P. (2010). 'Armed conflict 1946–2009', *Journal of Peace Research*, vol. 47(4), pp. 577–87.
Hsiang, S.M., Burke, M., and Miguel, E. (2013). 'Quantifying the influence of climate on human conflict', *Science*, vol. 341(6151), pp. 1235367-1-14.
Hsiang, S.M., Meng, K.C., and Cane, M.A. (2011). 'Civil conflicts are associated with the global climate', *Nature*, vol. 476(10311), pp. 438–41.
Hunter, P. (2003). 'Climate change and waterborne and vector-borne disease', *Journal of Applied Microbiology*, vol. 94(Suppl.), pp. 37S–46S.
Karp, C. and Auwaerter, P. (2007). 'Coinfection with HIV and tropical infectious diseases. I. Protozoal pathogens', *Clinical Infectious Diseases*, vol. 45(9), pp. 1208–13.
Lammers, J. and van Wijnbergen, S. (2008). 'HIV/AIDS, risk aversion and intertemporal choice', Discussion Paper No. 2007-098/1, Tinbergen Institute.
Lorentzen, P., McMillan, J. and Wacziarg, R. (2008). 'Death and development', *Journal of Economic Growth*, vol. 13(2), pp. 81–124.

McMicheal, A., Woodruff, R. and Hales, S. (2006). 'Climate change and human health: present and future risks', *Lancet*, vol. 367(9513), pp. 859–69.
Michalopoulos, S., and Papaioannou, E. (2013). 'The long-run effects of the scramble for Africa', Working Paper No. 17620, NBER.
Miguel, E., Satyanath, S. and Sergenti, E. (2004). 'Economic shocks and civil conflict: an instrumental variables approach', *Journal of Political Economy*, vol. 112(4), pp. 725–53.
Montalvo, J.G. and Reynal-Querol, M. (2005). 'Ethnic polarization, potential conflict, and civil wars', *American Economic Review*, vol. 95(3), pp. 796–816.
Murray, D.R. and Schaller, M. (2010). 'Historical prevalence of infectious diseases within 230 geopolitical regions: a tool for investigating origins of culture', *Journal of Cross-Cultural Psychology*, vol. 41(1), pp. 99–108.
Oster, E. (2012). 'HIV and sexual behavior change: why not Africa?', *Journal of Health Economics*, vol. 31(1), pp. 35–49.
Oster, E. (2014). 'Unobservable selection and coefficient stability: theory and evidence', Working Paper No. 19054, NBER.
Putterman, L. and Weil, D. (2010). 'Post-1500 population flows and the long-run determinants of economic growth and inequality', *Quarterly Journal of Economics*, vol. 125(4), pp. 1627–82.
Reiter, P. (2001). 'Climate change and mosquito-borne disease', *Health Perspectives*, vol. 109(1), pp. 141–61.
Rockwood, K. and Mitnitski, A. (2007). 'Frailty in relation to the accumulation of deficits', *Journal of Gerontology: Medical Sciences*, vol. 62(A), pp. 722–7.
Rodenwald, E. and Bader, R.-E. (1961). *World Atlas of Epidemic Diseases*, vols. 1–3, Hamburg: Falk-Verlag.
Rohner, D., Thoenig, M. and Zilibotti, F. (2013). 'War signals: a theory of trade, trust and conflict', *Review of Economic Studies*, vol. 80(3), pp. 1114–47.
Simmons, J., Whayne, T.F., Anderson, G.W. and Horack, H.M. (1944). *Global Epidemiology: A Geography of Disease and Sanitation*, Philadelphia, PA: J.B. Lippincott.
Smith, C.F., Sax, D.F., Gaines, S.D., Guernier, V. and Guegan, J.-F. (2007). 'Globalization of human infectious disease', *Ecology*, vol. 88(8), pp. 1903–10.
Süss, J., Klaus, C., Gerstengarbe, F. and Werner, P. (2008). 'What makes ticks tick? Climate change, ticks, and tick-borne diseases', *Journal of Travel Medicine*, vol. 15(1), pp. 39–45.
Sutton, A.E., Dohn, J., Loyd, K., Tredennick, A., Bucini, G., Solrzano, A., Prihodko, L. and Hanan, N.P. (2010). 'Does warming increase the risk of civil war in Africa?', *Proceedings of the National Academy of Sciences*, vol. 107(25), pp. E102.