Plague and war: Political breakdown and the spread of HIV

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Previous studies of war and infectious disease have generated inconsistent findings on the interrelationships of these phenomena. This study investigates the association of war with HIV prevalence using time-series cross-section (TSCS) analysis of a data set that is more inclusive in both spatial and temporal terms than the data used in previous analyses.

Two design aspects of this study differ from previous efforts. First, I have compiled data from both epidemiological and political science sources, and the result is a larger dataset for analysis, including 17 times periods and the full range of developing countries, not just those in sub-Saharan Africa.

Second, due to the expanded data and as a real-world test of common suggestions for what to do with time-series-cross-section (TSCS), the study uses five commonly recommended statistical procedures to evaluate the data, providing comparison of common TSCS practices.

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1 Introduction

The world has been aware of HIV (Human Immunodeficiency Virus) for over 30 years now, and three decades into the pandemic the disease remains a grave problem, especially in less politically and economically developed countries of the world. Many of the countries of the world most affected by HIV also face significant challenges in their political circumstances. They undergo various forms of war and civil violence. This paper assesses the effect of these challenges upon the change in HIV prevalence and incidence, assessing the relationships quantitatively over 139 countries in the period 1990–2007.

War is known to be potentially full of the possibility for creating or exacerbating upheaval. Understanding the way in which war may relate to an "exterior" problem may allow those in positions of authority to devise ways in which to ameliorate the political irruptions that an epidemic like HIV may be subject to.

This study adds the following to our understanding of the relationship of the relationships of armed conflict and HIV.

- Exploratory data analysis finds that there is a negative relationship between some forms of warfare and HIV prevalence.
- The study uses a *longer time frame* (relative to others), with up to 17 years of annually observed estimates of HIV prevalence. I also test the associations across a *broader range* of countries.
- This article uses a variety of methods for time-series, cross-section analysis (different estimation procedures, different error structures), given that the literature's advises a multiplicity of methods to deal with such data. While the estimation results differ somewhat, the substantive relationships discovered hold over a variety of procedures and specifications. This increases confidence that the imputed relationship exists.

It is important to note that the following is primarily exploratory data analysis. Where possible, I outline my suppositions for why I obtain different results than other studies,

but due to the nature of the data under examination, causal mechanisms are not often directly testable. The results presented should help to drive further theory, hypothesis generation, and data upon which we can test those suppositions.

1.1 Background

Epidemic diseases, like famine and other "natural" disasters, occur via a combination of biological and social factors, both of which have been jointly necessary to drive the problem to crisis levels. The social factors have generally dovetailed with the particular biological characteristics of the disease pathogen that facilitates its spread most effectively.

Conflict situations can spread infections well, depending on the virulence and transmissibility of the pathogen. Cholera, with a fecal-water-oral transmission route, will spread easily when sanitation and purification systems break down. Airborne pathogens can spread easily from person to person via droplet infection routes, and airborne pathogens have spread with particular ease in situations when previously isolated populations are brought into contact with one another, either because of troop movement or refugee flows. Vectors often spread diseases via blood, saliva, urine, or feces containing the pathogen. In World War II, one way in which Allied soldiers in the Pacific theater reduced malarial morbidity and mortality, compared to the Japanese, came from extensive use of DDT to kill mosquito larvae, stopping the transmission of *Plasmodium*. There exists evidence that insect-vector diseases may be a principle cause for the spread of disease in recent wars (Hotez 2013).

Not all diseases are the same, however, and we should not expect that all infections will spread in like fashion under similar social conditions. Epidemics are combinations of the biological and the social. The more specific the circumstances required for transmitting a disease and for keeping the pathogen viable, the less transmissible the pathogen will be.

The human immunodeficiency virus requires quite specific conditions for transmission. It does not spread particularly easily nor is it very robust outside the human body and

fluids. One recent meta-analysis study (Boily et al. 2009) indicates that male-to-female transmission probability per sex act with no commercial sex exposure in low-income countries is 0.30% (1 in 300) and female-to-male probability is 0.38% (about 1 in 250).

To maximize efficient sexual spread through the population, HIV needs at least the following conditions. First, an ability for individuals to meet, associate, and have sexual relations; this requires urban densities of geography and time (a moderate to high number of people per unit of space and a high number of contacts over a given unit of time). Geographic density and socio-economic production conditioned the "epidemic ignition" of HIV (Faria et al. 2014).

Second, HIV requires population of people linked together in a social network. Social networks are particularly important for HIV, and relatively small increases or decreases in the mean number of partners can drastically affect whether HIV spreads widely or remains restricted to a relatively small proportion of a network (Carnegie and Morris 2012).

Growing interest in the relationship between political processes and public health have primarily focused upon aggregate measures of population health, like Disability-Adjusted Life Years (DALYs) (Ghobarah et al. 2004, 2003). This has the unfortunate effect of ignoring the possibility that political events may differently affect the public health depending upon the disease under consideration.

1.2 Conflict

War and disease usually link to one another in a positive direction, with war and violent conflict driving the incidence and prevalence of disease(s) upward.

War and civil conflict disrupt peacetime physical and organizational infrastructure. Much of this infrastructure contributes directly or indirectly to general human health and the prevention of disease. When conflict prevails, sanitation collection, water purification and reclamation systems, waste disposal, food production, and a whole host of systems

suffer damage and destruction.

The diseases that war can exacerbate are those primarily spread via water contamination and airborne/droplet infection. This would include cholera, tuberculosis, influenza, smallpox, typhus, dengue fever, malaria, plague, and yellow fever (World Health Organization N.d.). Other diseases—difficult to spread via water, air, or casual human contact—are less likely to spread in war, conflict, or "humanitarian emergency."

War and violent conflict create refugee and migrant flows. Migrant populations can bring with them infections that are endemic to their home area, but which are relatively unknown elsewhere. In moving to a new area, migrants face new disease environments to which they have relatively low immunity, and epidemic conditions can develop quickly in host or refugee populations. The conditions in refugee settlements are often lacking in terms of sanitation, public health, and medicine.

Ghobarah et al. (2003, 2004) find that civil conflict has long-term effects that can equal the immediate, proximate negative effects of a civil war. They find that DALYs lost as a result of a particular year's civil conflicts (1999, in their case) are equal to the lost DALYs from after-effects of wars from 1991–97. They also find that "AIDS rates" [sic] did not increase in the case of civil war (2003, 200). What the present study can add to their work is an appreciation of trends over space *and* time.

The usual hypothesis in previous studies relating war and HIV draws from the more general relationship between epidemic disease spread and war. That is, researchers expect that the presence of violent conflict will associate with higher HIV prevalence.

Published evidence of the relationship of conflict and HIV's spread is mixed, and the answer to the question of how one affects the other often depends upon whether the analysis occurs at the micro- or macro-level (Paxton 2012). Early studies, primarily qualitative examinations of a small set of countries, indicated a positive relationship: with the increase of conflict, especially civil or intrastate war, HIV prevalence also increased (UNAIDS 1998; Hankins et al. 2002; Ostergard 2002; UNAIDS 2004). Two potential

causal stories dominate:

1. In war, especially that with an ethnic character, forces for one side could use rape to dilute racially and pollute ethnic antagonists. Should some men be HIV-positive, so much the better.

2. In refugee camps, because people are displaced from the social and institutional structures that mitigate risky behavior, women in particular would be at greater risk for sexual assault and violence, and this would facilitate more efficient spread of HIV.

By the first decade of the 2000s, based on the foregoing, it had become common among professional development workers and global HIV activists to assert that the violence of war and concomitant disruptions like refugee flows increased overall HIV prevalence (see, for example, Salama and Dondero 2001; International Save the Children Alliance 2002; Smith 2002; Carballo and Solby 2001; Hankins et al. 2002).

More recent quantitative studies, however, have been mixed in their conclusions. Spiegel et al. (2007), argued (to some controversy) that conflict related-rape occurring among refugee populations did not appear to increase HIV prevalence. Notably, Spiegel et al. did not employ any standard political science measures for concepts like conflict, polity type, and other political variables. Iqbal and Zorn (2010), on the other hand, tested linear regression models alongside spatial analysis, and the study found positive relationships and spatial "neighborhood" effects for war upon HIV. Iqbal and Zorn restricted their analysis to sub-Saharan Africa and to HIV prevalence from 1997-2005, measured every other year ($N = 181$).

In line with the work of Davis and Kuritsky (2002) and Mock et al. (2004), I am less convinced by such logic (i.e., War $\Rightarrow$ Rape $\Rightarrow$ Increased HIV). HIV differs from diseases like cholera, plague, smallpox and influenza in at least two important respects. HIV's primary mode of transmission is human sexual contact and its secondary mode is direct

blood transfer. Under peacetime conditions, the virus needs a highly connected network to spread widely in a human population, and it is not clear that such conditions can be maintained in time of war. Carnegie and Morris (2012) found that small changes in the mean number of partners (e.g., from 1.9 to 1.7 at any particular time) can have radical effects on the amount of the population linked together and most able to sexually spread a disease. Although there exists little direct evidence about sexual activity in war, we can question the logic of whether people engage in "normal" sexual activity, much less the extensive, dense, simultaneous networks needed to spread HIV.

War and violent conflict have two opposing effects for potential sexual interaction. First, they mobilize fighting forces and deploy them throughout the theater of the conflict. As noted above, some research has advanced the idea that soldiers are either more likely to harbor HIV within the ranks and that they also use rape and sexual violence as part of the weapons of war. Given that rape and HIV did not seem to be at higher incidence in civilian populations subject to military violence Spiegel et al. (2007), we should be suspicious of this mechanism for a relationship between epidemic and war.

Second, in the region where war occurs, civilian movement proves dangerous and risky. Remaining where one is, if a non-combatant, may also prove risky, if the fighting is near enough to where one is. To create refugee flows, however, the costs and dangers of movement during time of war — decreased social stability of leaving one's village or neighborhoods, potential for rape or other sexual assault, loss of possessions and properties resulting from their abandonment and so forth — have to outweigh the costs of staying where one is. Until those costs are high, war will result in a preference for "hunkering down" in people's current circumstances to wait out the conflict. Anecdotally, this is what happened in the case of Angola, during its civil war from 1975–2001.

The political institutions and governance within a country affect the qualities and intensity of an HIV response. Lieberman (2009) analyzed the ways that ethnic group boundaries affect the effort that political leaders put into anti-HIV policies and the

resulting effects that this has on the spread of the disease. Importantly, more rigid ethnic boundaries lead to a sense of in-group/out-group membership, with leaders more willing to pursue HIV policies that benefit members of their own ethnic group. If we consider ethnic war as the most intense expression of ethnic barriers among different groups, then we could expect ethnic war might result in an overall increase in HIV prevalence.

In sum, I do not intend to say that HIV would be *impossible* to spread during conflict, but sufficient evidence exists on the importance of sustained sexual networks that we can question factors like war that disrupt those networks. On this logic, I expect that although the *process* may be hard to assess quantitatively, the *outcome* of violent conflicts is that they do not exacerbate the spread of HIV (the total effect is negligible or negative).

2 Methods

Data used in this paper come from standard sources in political science and political sociology, combined with other data obtained from UNAIDS, United Nations, and World Bank databases. All data are public and widely available.

I restrict analysis to the set of "developing" countries, as defined by the World Bank's country income categories of low, low-middle, and high-middle income countries. This makes 139 countries potentially available for analysis, over the period from 1990–2007. I further separate out the sub-Saharan African countries to perform analysis on them apart from that on the full set of developing countries. The HIV epidemic has affected this region of the world more severely than any other, and the epidemic here differs from that in the rest of the world.

2.1 Dependent Variable: HIV prevalence

Data on HIV prevalence was taken from the UNAIDS (2008) national-level, time-series estimates of HIV prevalence; I use the midpoint estimate of HIV prevalence, rather than

the high and low estimates also provided.

Graphical analysis of HIV prevalence shows some interesting differences between and within the two subsets of data I examined, sub-Saharan Africa and non-African developing countries. Figure 1 shows histograms and kernel density curves by country for all states in sub-Saharan Africa, and Figure 2 is an aggregate curve for all of sub-Saharan Africa. In aggregation, we can see that most country-years in sub-Saharan Africa have low overall HIV prevalence, and the modal range is between 0 and 1 percent. The distribution of values, however, is quite broad, ranging out to estimates in the high-20 percentiles. Disaggregated by country, as in Figure 1, we see much variation in the levels and trends of prevalence different countries face. Some have maintained fairly low levels of HIV (e.g., Angola or Ghana), some have experienced changing levels of prevalence (Uganda), and others have had mostly high prevalence (Botswana, Zambia, Zimbabwe).

Figure 1 and 2 here.

For the developing world *sans* sub-Saharan Africa, the story in Figures 3 and 4 is a different one. Most significantly, the range of HIV prevalence in these countries is a full order of magnitude lower than in sub-Saharan Africa, with values ranging from 0–3 percent (as compared to 0–30 percent). The aggregate curve is also (perhaps) less "bumpy" than in Figure 2. When comparing the by-country graphs, there do appear to be more countries in the non-sub-Saharan Africa set that have a greater range of values, indicating changes in the size of epidemics over time. These, however, have also remained largely between 0 and 3 percent.

Figure 4 and 3 here.

2.2 Explanatory variables

2.2.1 Conflict episodes

For the measure of various types of armed conflict, I used the Major Episodes of Political Violence (MEPV) data set (Marshall 2006). In this case, MEPV provides the most relevant dataset for several reasons: (1) it draws upon other common conflict datasets, including the COW and SIPRI datasets, so it makes use of relevant information in other sources; (2) the author of MEPV is one of the co-authors for Polity IV, which I use for a control variable and which should augment conceptual consistently across datasets; and (3) MEPV is sensitive to the characteristics of modern warfare, requiring fewer threshold deaths than other datasets, counting civilian deaths rather than just military force deaths, and separating intra-state warfare into ethnic and civil types.

The MEPV provides data for all country-years covered in the HIV prevalence data, and it separates conflicts according to a magnitude criterion and a type criterion. In terms of magnitude, there are (1) wars and (2) episodes of armed violence. I have conducted this analysis primarily upon the cases coded as wars, because "the designation of 'war' carries with it a stronger institutional, or institutionalized, component and more definite objectives" (Marshall 2006). Political science definitions of "war" have also traditionally been more definite and consistent, following those initial criteria established in the Correlates of War project.

War variable measures have three-year lags in the regressions shown below. I chose three year lags for information and epidemiological reasons. Since HIV is a disease that can take a while to manifest in an obvious way, one needs to build in some time lag to account for the long "dormant" period of the virus. Epidemiological surveillance is unlikely to continue full functioning during war, especially if resources available to a government are limited. Even if surveillance continues at previous levels, it may take a government time to catalog, process, and publish information on the presence of HIV, in the face of

warfare. The three-year-lag provides a chance for the virus, surveillance, and information to catch up. I have tested these models with one- and two-year lags, and the results are not substantially different.

(To give some idea of an increase in MEPV scores mean, in Table 1, I present MEVP scores for some recent episodes of state violence so that the interpretation of these results makes more intuitive sense.)

Table 1 about here.

2.3 Control variables

With respect to the control variables, with the exception of the variable measuring polity, these were taken from the World Bank and the UN High Commissioner for Refugees (UNHCR). Data availability imposes some restrictions upon what can be used. In several cases, plausible controls (such as total health spending/gov't health spending as a percentage of gross domestic product (GDP); literacy rate; or urban population percentage) were not available for more than 2 or 3 of the 17 total years in this dataset. Inclusion of these variables would severely limit the time-element of this study, cutting the number of cases drastically. In line with the advice of Achen (2005), I have not included every possible control variable, a la so-called "garbage can regressions," but I have included a set of controls that covers in concept and previous statistical analysis the alternative structural factors for levels of HIV prevalence.

2.3.1 Political regime

I use the Polity IV (Marshall and Jaggers 2006, rev. 2007) data to measure the political character of the country-years. For both sub-Saharan Africa and other developing nations, the mean scores are just slightly above the scale's midpoint of 0 (0.1 and 1.5 respectively) but the standard deviations are rather large. In both sets of countries, the polity scores distribute bimodally. Most country-years are either quite democratic or autocratic, and

(comparatively) few fall in the middle range of scores. The set of non-sub-Saharan Africa developing countries has relatively even autocratic and democratic peaks, while Sub-Saharan Africa has a comparatively taller autocratic peak.

2.4 Models

I conducted analysis via linear regression of HIV prevalence upon the foregoing independent and control variables, using several regression techniques to assess the robustness of the findings. I also used two different error estimation procedures, both often recommended for TSCS analysis.

For this data, covering a relatively large number of countries (from 46–139) and years (17), where $T < N$, and where most of the independent variables of interest are relatively stationary over time, the fixed effects estimator provides a commonly accepted way to examine "un-modeled factors" particular and idiosyncratic to individual countries. With the sort of data employed here, Green et al. (2003) indicate that including fixed effects can help to address some of the problems that come from unobserved "interdependencies among the observations" (441).

This is not to say that the fixed effects method is universally recommended for TSCS. Beck and Katz (1995) argue that for much of this type of data, a better procedure would use pooled ordinary least squares (OLS) for coefficient estimates and then to use a standard error technique devised to account for the fact that the data is actually composed of multiple panels of countries observed over the same time period(s): the panel-corrected standard error (PCSE). They recommend PCSE because more conventional Huber-White "robust" standard errors (RSE) treat the potential heteroskedasticity introduced by having multiple panels as a problem rather than as an integral and interesting aspect of TSCS data. RSEs are thus likely to be smaller than the "true" standard error (Beck and Katz 1996, 21). For the sake of comparison, I present results estimated with both RSE and PCSE, although PCSE are generally more appropriate for modelling the error structure

in data like mine.

Beck and Katz (1996) suggest that when examining TSCS data, in addition to using OLS and PCSE, one should include a lag of the dependent variable (LDV) on the independent variable side of the regression equation. This allows for "dynamic" (serially independent) error structures. It takes account of the level of the dependent variable in the previous time period, and effectively de-means the dependent variable. One refers to this as taking account of the "unit root." However, this only works when the unit root is not approximately equal to one. When the unit root is close to one (that is, $\gamma \approx 1$ in $Y_t = \gamma Y_{t-1} + \varepsilon$), virtually all the variation in the dependent variable would be "sucked up" by the previous values of that variable. In this case, we have to find other ways to de-mean the dependent variable, and the most common is to take the time-period differences of both sides for variables that do not change much (factors that are "big and slow-moving"). This allows one to see the difference that changes in the independent variables make on changes in the dependent variable. Such is the method of first differences.

Beck and Katz's LDV will not work in this dataset. As Achen (2000) notes, in a situation where there is both serial correlation in the regression equation and heavy trending in the LDV, "...the autoregressive term does not conduct itself like a decent, well-behaved proxy. Instead, it is a kleptomaniac, picking up the effect, not only of excluded variables, but also of the *included* variables if they are sufficiently trended. As a result, the impact of the included substantive variables is reduced, sometimes to insignificance" (7, emphasis in original).

In this data, when estimating the equation where Y is regressed on its immediately previous value, the coefficient is essentially equal to one, suggesting that yearly values of the prevalence of HIV depend largely on the values of previous years. This should not be surprising. However, once we include the lagged dependent variable in any of the models and procedures presented below, even well-accepted causal factors, like per capita income and aid assistance fail to be statistically or substantively significant. Fixed effects

regressions to some extent get around this limitation, putting all country idiosyncratic factors including past HIV prevalence into the separately estimated country intercept. Indeed, fixed effects regression can be combined with PCSE to achieve the desired estimation strengths of those multiple intercepts, as well as accounting for the potentially different error structures that each country unit can reasonably be expected to have (Nathaniel A. Beck, personal communication). Achen (2000) also suggests that country dummies/fixed effects can address some of the bias present, that between countries.

The first-difference results presented in Table 4 provide another way of attempting to account for influential past values of the dependent variable. By presenting three different regression procedures and two error structures, I test whether the results coming out of the estimations are substantively robust. That is, if we see similar effects across models, we can be more sure than what we have found is "true".

I use the following equations to estimate the regressions presented in Table 2, Table 3, and Table 4.

$$Y_{it} = \beta_0 + \beta_i x_{it} + \varepsilon_{it} \tag{1}$$

$$Y_{it} = \alpha_i + \beta_i x_{it} + \varepsilon_{it} \tag{2}$$

$$\Delta(Y_i)_t = \beta_i(\Delta(X_i)_t) + \varepsilon_{it} \tag{3}$$

where the independent and control variables, x, are those described above, i denotes the country, t is the time period, α_i is the vector of country intercepts (fixed effects), and ε_{it} are the random error terms. Equation 1 is the "pooled", standard OLS estimator; equation 2 estimates the fixed/within effects; and equation 3 is the first differences procedure.

3 Results and Discussion

For each model (combination of independent and control variables), I used the regression procedures above and two different methods of calculating the coefficients' standard error (RSE and PCSE). In Tables 2 and 3, Model I tests the independent variables by themselves, and Model II introduces the control variables. Finally, Table 4 examines the effects of differencing for both sub-Saharan Africa and the full set of developing countries for Model II, using pooled and fixed effects procedures with PCSE in all variations.

Table 2 about here.

Table 3 about here.

3.1 War and conflict

Across a variety of model specifications, regression techniques, and error calculations, different types of war also differ in their effects upon the prevalence of HIV. Notably, *intra-state wars, civil or ethnic, are associated with decreased levels of HIV prevalence.* The greater the degree of violence in a civil or ethnic war, the lower the associated level of HIV found in the population three years later.

In sub-Saharan Africa, there are surprising and significant (in terms both statistical and substantial) negative relationships between HIV prevalence and internal warfare. Across model specifications, estimation techniques, and error structures, greater civil and ethnic warfare associated with lower levels of HIV. With eight different sets of results, the coefficient on civil war ranged from -0.209 to -0.911 and was statistically significant (at $p < 0.10$ in seven cases. For ethnic war, the regression coefficient ran from -0.284 to -0.498, with statistical significance in six cases.

For the "average" civil war in sub-Saharan Africa (that is, when the civil war variable is non-zero; the corresponding value is 4.33, or a war about the intensity of the Eritrean Independence War) one should expect that HIV prevalence in those countries would be

from 0.90 to 3.94 percent lower than in the hypothetical case where the war did not occur. For ethnic wars (where the average size was 4.82), the corresponding decreased levels would be between 1.37 and 2.40 percent.

As one would expect, the inclusion of country-specific intercept vectors (the fixed effects model) increases the variation accounted for, as indicated by the R^2 and the "root mean squared error" measures. The use of the PCSE method, which I argued above is more appropriate with this data, increases the significance of the estimated coefficients, not only for the independent variables of interest. Control variables' error terms were either similar or smaller (better).

Model II (the right-hand four columns of the table) includes control variable measurements. This model, again using both pooled and fixed effects estimation and both error calculations, gives coefficient estimates that we can account as more "realistic", in that they take account of alternative possible explanations. Again, the results are relatively clear in indicating that HIV is lower than otherwise would be when there was a civil war or ethnic war in the country three years previous.

Similar results attain for the full set of developing countries (where I have included a dummy variable for membership in the set of sub-Saharan African countries, given the particular intensity of the pandemic in that part of the world). The size of the coefficients is smaller in this full set, but we still see negative, statistically and substantively significant, results here.

The coefficients for the effects of internal wars upon HIV are quite large, in that they indicate the shift in HIV prevalence for *each unit* of the MEVP score for a particular country. One can analogize and better understand from the example of a hypothetical Angola. In a national civil war similar to the Angolan civil war, we would expect to observe an HIV prevalence of 1.5 to 3.9 percent lower. Note that this does not necessarily mean that a civil war like Angola's *decreases* the level of HIV in the population. What it does mean is that when a civil war of the intensity of Angola's occurs, we can expect that

HIV prevalence will be lower than if the war had not occurred. A similar logic and results obtain for the case of ethnic warfare.

This analysis is quite sobering. For all of the misery associated with civil or ethnic conflict, *it appears that this type of violence associates with lower HIV levels.* Civil and ethnic wars have powerful negative effects upon the level of HIV in the population of the state under consideration. In other words, HIV prevalence is *lower* under conditions of ethnic or civil war than in the absence of that war.

As we can see, interstate war does not appear to have a consistent effect upon HIV prevalence. For Sub-Saharan African countries, international war's effects in the full model (Model II) are not statistically significant in the pooled estimation and relatively small in the fixed effects estimation. Further examination shows that there are only ten non-zero cases (i.e., where the magnitude of the international war measure is greater than zero) in sub-Saharan Africa.A similar situation is likely at work for the full set of developing countries, where there are only 87 non-zero cases over the whole data.

3.2 Regime type and HIV

In sub-Saharan Africa, the coefficient of democracy with HIV prevalence is either positive or not significantly different than zero. That is, the more democratic a country is, the higher its HIV prevalence. This is relatively unsurprising. By way of example, the five most democratic countries in sub-Saharan Africa are Botswana, The Gambia, Mauritius, Namibia, and South Africa, and three of these—Botswana, Namibia, and South Africa—are countries with some of the world's highest levels of HIV (ranging from 10 to 21 percent of the adult population). Although in the sparse Model I, the relationship appears quite strong, in the Model (II) with controls, the relationship of polity score to HIV prevalence pretty much disappears. To take an example, for Botswana, with an average polity score of about eight, the increase in HIV prevalence attributable to democracy ends up being about 0.85 percent.

In the whole of the developing world, the results are less clear. The sparse model, which either includes a sub-Saharan Africa control or country-level fixed effects, would indicate a strong and significant relationship. Once controls are included, only one of the four variants of Model II achieves statistical significance, and in any case, the coefficients are small. During the entire period of this study, Brazil's polity score was 8; this yields an overall decrease in HIV prevalence (under the pooled PCSE estimation) of 0.34 percent. Not much, but given that the population prevalence in Brazil was about two-thirds of one percent, a difference of 0.34 percent (as compared to when Brazil was a military autocracy and had a polity score of -4) would certainly be of significant real-world concern.

In the case of the developing world writ large, modeling choices very much influence the implication one can draw from the empirics. The fifth through eighth columns of Table 3 are relatively different, with respect to the estimated effect of democracy and whether the associated error is small enough for it to be statistically significant. One's model and error estimation choices greatly alter the results obtained, with implications for the conclusions drawn. In the case of this data (moderate length time series, many cross-sections, *a priori* reasons for treating the cross sections as heterogenous), the final column (Model IId) makes the most sense, and polity score does not play a substantive or statistically significant role.

3.3 Differencing analysis

As noted above, so-called "unit roots" may confound the sorts of causal arguments that we are able to reasonably sustain about TSCS data, when the period-lagged value of the dependent variable enters into the right-hand side of the regression equation.

Table 4 about here.

In Table 4, there are several aspects to take note of before interpreting the results. First, factors that we know from the previous analyses and other studies to be relevant

predictors of HIV prevalence (i.e., the controls) are relatively small, often not statistically significant, and of small substantive significance. Similarly, on the independent variable measures, the coefficients are generally smaller and with larger standard errors. This is to be expected, as we are now assessing the effects of differences in differences in HIV prevalence, rather than just the effect of differences. The general, "background" levels of these variables have been subtracted out. We are looking at what predicts mean annual increases of 0.23 and 0.12 percent in HIV prevalence, in sub-Saharan Africa and the rest of the developing world.

What we see in Table 4 is that the effects of the right-hand variable differences on the dependent variable's differences are most predictive in sub-Saharan Africa countries. In Column 2, where different intercepts for each country are estimated, independent and control variables both perform in line with the results in Tables 2 and 3.

The differences effect results are somewhat indeterminate, except for two findings. First, among all developed countries, only being among the set of sub-Saharan African countries was statistically significant. In fact, as seen in the pooled regression model for all developing nations (Column 3), the coefficient on that variable (0.119) accounts for almost the entirety of the average change (0.122). Second, within the sub-Saharan African nations, changes in the level of civil or ethnic warfare (which are infrequent, occurring most often at the initiation or cessation of hostilities) associate with changes in HIV prevalence differences. Based on the results here, we would expect that the outbreak of a war comparable to the Congolese civil war would result in 0.18 percent decrease in HIV prevalence change, negating three-quarters of the mean year-on-year increase for sub-Saharan African countries. A shift in ethnic warfare intensity like Rwanda underwent in the early '90s would decrease HIV differences by roughly one third of a percent, equivalent to one-and-a-half years mean HIV differences.

In and of themselves, the results presented in Table 4 might be explained away. In combination with the results on non-differenced models, it appears that intra-state war

processes have at least some degree of relation to lower HIV prevalence.

4 Conclusions

Intrastate violence exhibits lower HIV prevalence, as compared to the (hypothetical) case in which violence does not occur. That finding is robust across a number of estimation procedures and model specifications.

These results may prove sobering, for they suggest the pursuit of some goods may come at the expense of others. That is, lessening civil conflict coincides and perhaps be causally related to concomitant increase in HIV.

If this is the case, the implication to draw is not that we should foment violent intra-state conflict in countries highly affected by the HIV pandemic. Instead, those involved in crafting high-level development policy, as well as those on the front lines of providing services, must put more attention and effort into anti-HIV measures when those take place in a more peaceful environment. The presence and consolidation of peace does not allow development professionals to let their guard down against this disease—indeed, they must be even more vigilant and work even harder against HIV.

There may be a trade-off between the consolidation of peaceful societies and managing one of our most deadly pandemics. With awareness of the trade-off, countries and organizations may be able to avoid goring themselves upon the horns of this dilemma.

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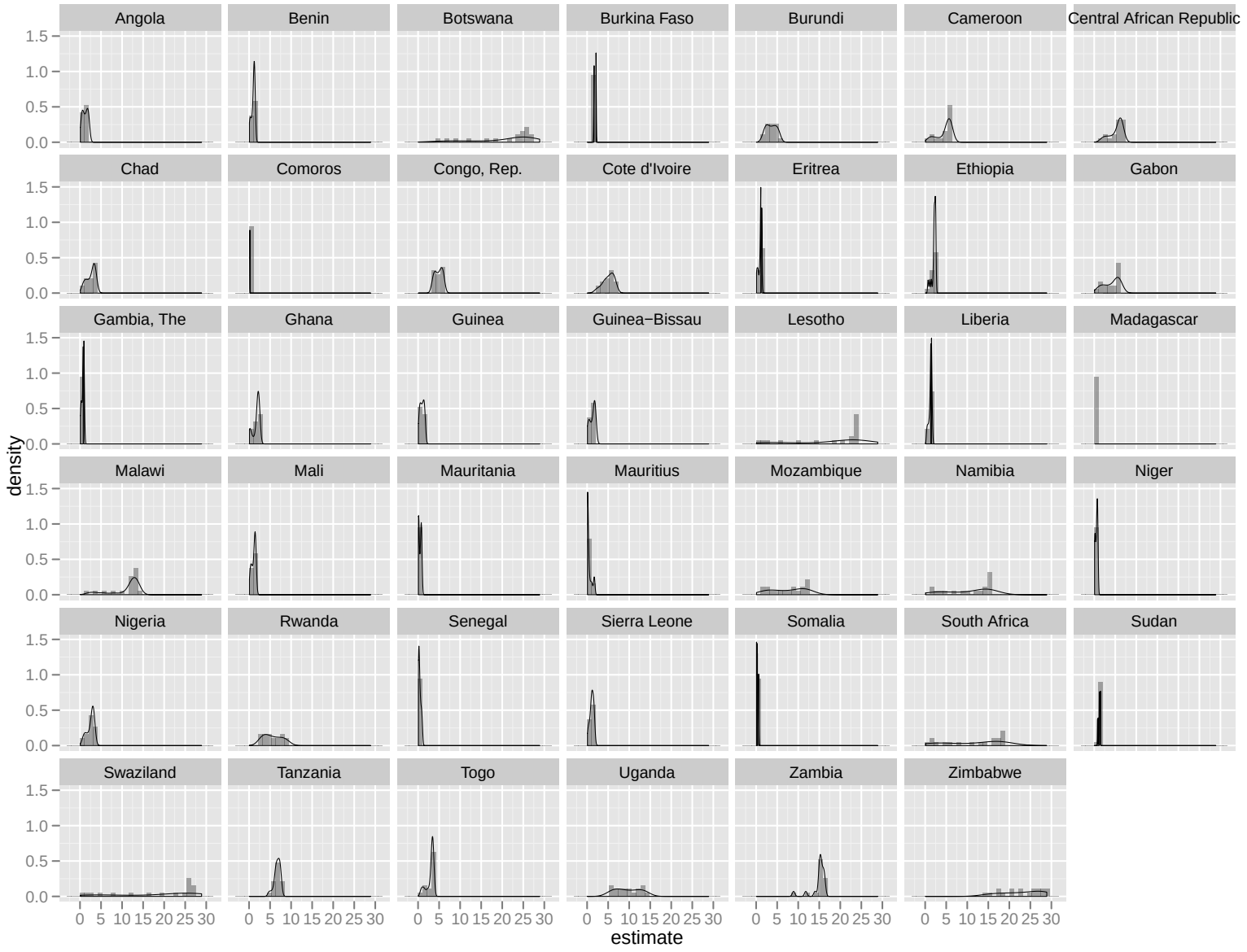

Figure 1: Histogram and kernel density of HIV prevalence, by country, Sub-Saharan Africa

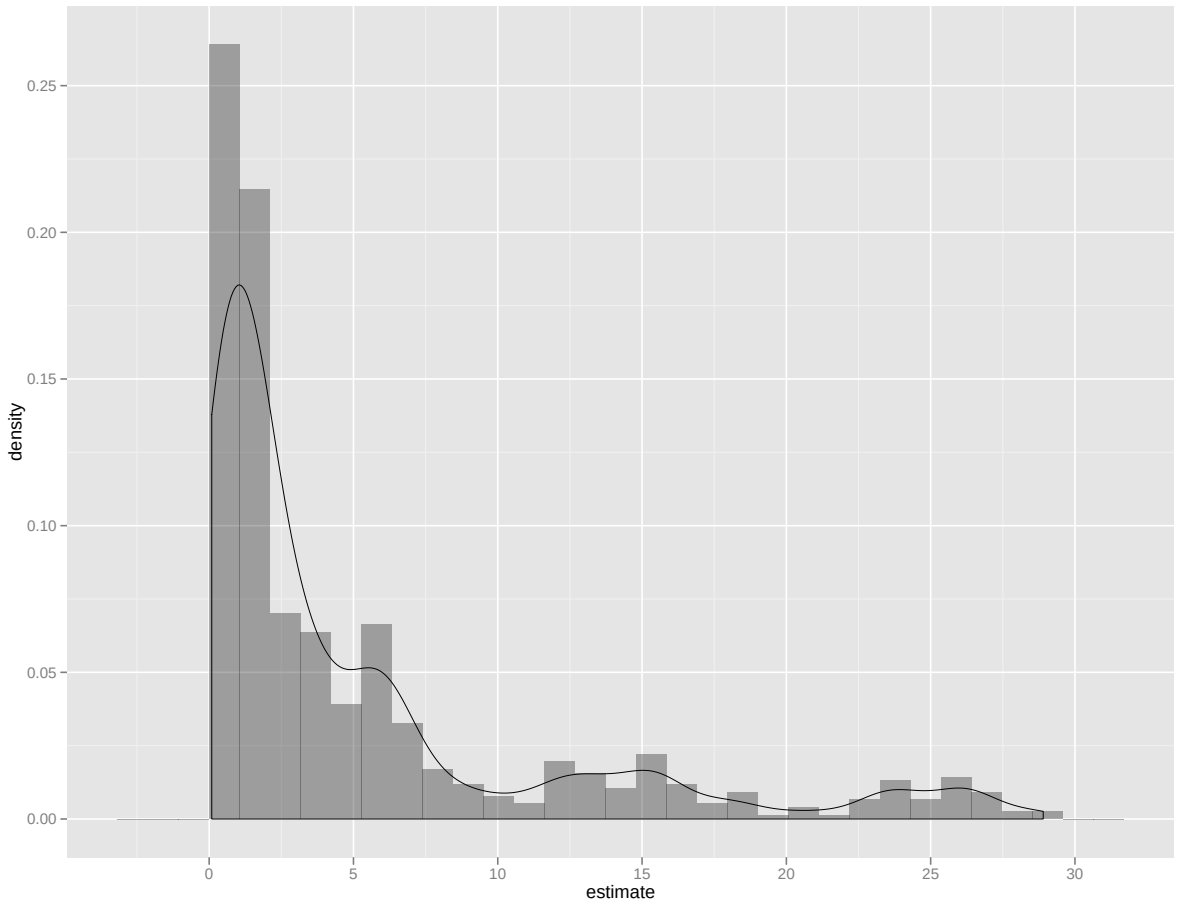

Figure 2: Histogram and kernel density of HIV prevalence, Sub-Saharan Africa

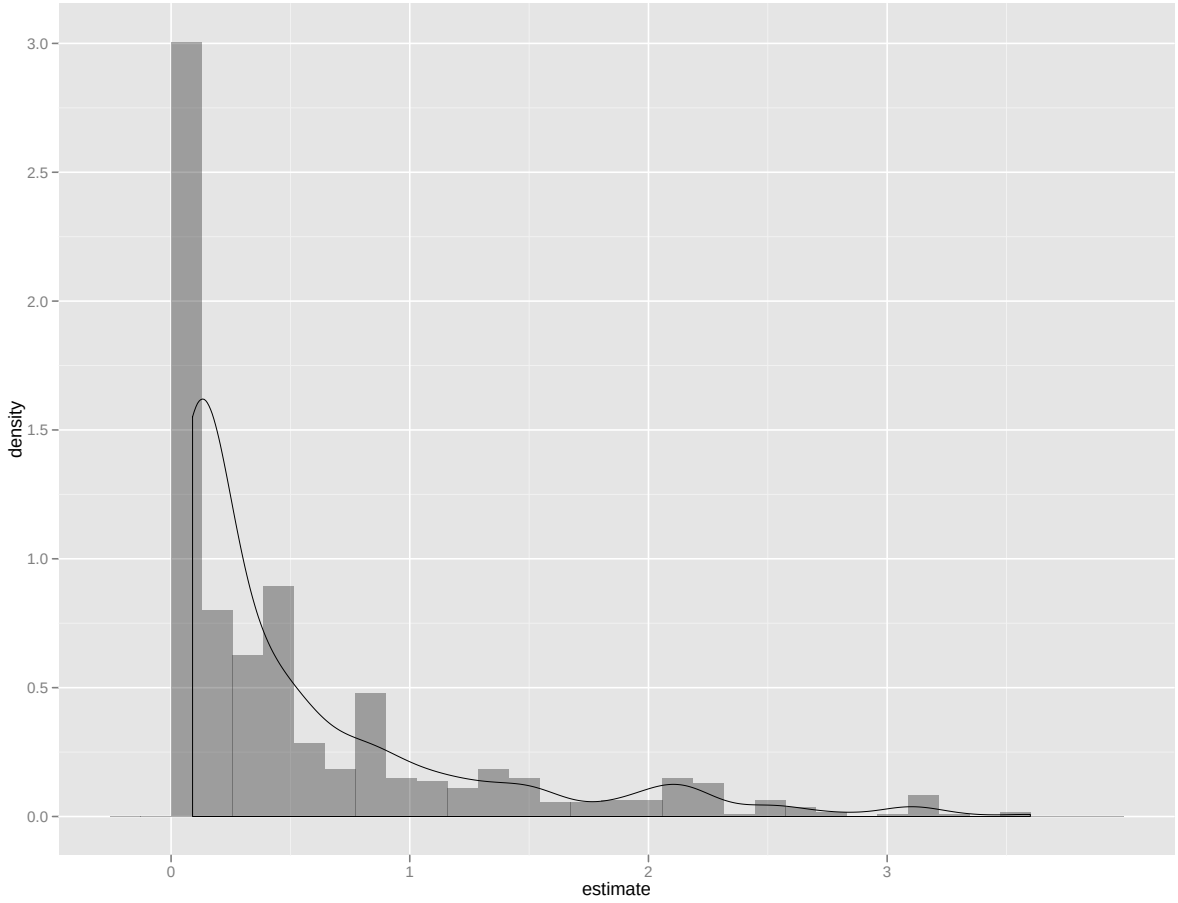

Figure 3: Histogram and kernel density of HIV prevalence, by country, other developing countries

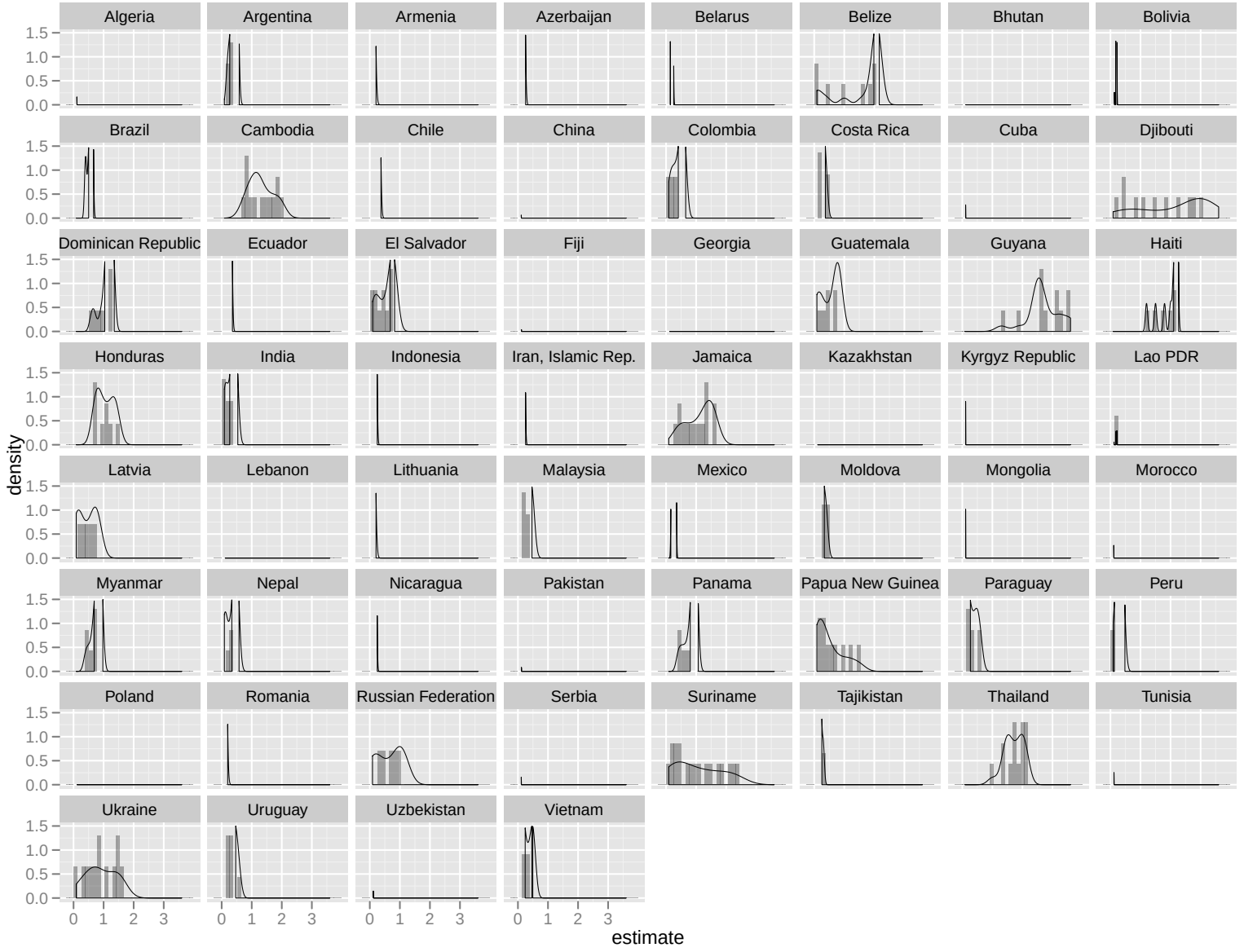

Figure 4: Histogram and kernel density of HIV prevalence, by country, other developing nations

Table 1: MEVP scores for some representative conflicts in the data under consideration

Country	**Years**	**War type**	**MEVP Score**
Afghanistan	1978–2001	Civil	7
Angola	1975–2002	Civil	6
Bosnia-Herzegovina	1992–1995	Ethnic	6
Rwanda	1990–1998	Ethnic	3 (10 in genocide)
Iran-Iraq War	1980–1988	International	6
Eritrean Indep.	1998–2000	International	5

Table 2: Multivariate regression results, Sub-Saharan African countries

Response Variable: HIV Prevalence								
					Models			
Independent Variables	**Ia–P, RSE**	**Ib–P, PCSE**	**Ic–FE, RSE**	**Id–FE, PCSE**	**IIa–P, RSE**	**IIb–P, PCSE**	**IIc–FE, RSE**	**IId–FE, PCSE**
	(1) m1AfrOLSrse *b/se*	(2) m1AfrOLSpcse *b/se*	(3) m1AfrFErse *b/se*	(4) m1AfrFEpcse *b/se*	(5) m2AfrOLSrse *b/se*	(6) m2AfrOLSpcse *b/se*	(7) m2AfrFErse *b/se*	(8) m2AfrFEpcse *b/se*
L3.Civil War	−0.911***	−0.911***	−0.388*	−0.388***	−0.562***	−0.562***	−0.209	−0.209†
	(0.076)	(0.083)	(0.175)	(0.084)	(0.129)	(0.104)	(0.130)	(0.108)
L3.Ethnic War	−0.498***	−0.498***	−0.406	−0.406***	−0.295*	−0.295***	−0.284	−0.284*
	(0.099)	(0.074)	(0.396)	(0.109)	(0.137)	(0.063)	(0.457)	(0.112)
L3.Int'l War	−0.734***	−0.734†	−0.041	−0.041	0.185	0.185	0.071†	0.071*
	(0.074)	(0.394)	(0.072)	(0.053)	(0.122)	(0.136)	(0.038)	(0.030)
Polity Score	0.091	0.091**	0.250**	0.250***	0.047	0.047†	−0.016	−0.016
	(0.059)	(0.030)	(0.078)	(0.038)	(0.080)	(0.026)	(0.026)	(0.014)
Log Off. Dev. Asst.					−0.527	−0.527**	−0.476*	−0.476**
					(0.324)	(0.161)	(0.221)	(0.145)
Log GDP/capita					−1.506†	−1.506**	1.064*	1.064*
					(0.872)	(0.568)	(0.515)	(0.519)
Refs. in country					0.005*	0.005**	−0.001†	−0.001***
(000s)					(0.002)	(0.002)	(0.001)	(0.000)
Log Health Care					4.773***	4.773***	−0.251	−0.251*
Exp./capita (USD curr.)					(0.881)	(0.601)	(0.327)	(0.128)
Observations	678	678	678	678	449	449	449	449
Countries		41	41	41		41	41	41
R^2	0.056	0.056	0.818	0.818	0.301	0.301	0.966	0.966
Root Mean Sq. Err.	6.577	6.577	2.889	2.979	5.951	5.951	1.317	1.382
Rho			0.805				0.964	

Table entries are linear regression coefficients, with standard errors in parentheses. Constants have been omitted for presentation. P: Pooled (OLS/GLS) model; FE: Fixed Effects estimator. RSE: Robust Standard Error; PCSE: Panel-Corrected Standard Error.
† p<0.10, * p<0.05, ** p<0.01, *** p<0.001

Table 3: Multivariate regression results, all developing countries

Response Variable: HIV Prevalence								
				Models				
Independent Variables	**Ia–P, RSE**	**Ib–P, PCSE**	**Ic–FE, RSE**	**Id–FE, PCSE**	**IIa–P, RSE**	**IIb–P, PCSE**	**IIc–FE, RSE**	**IId–FE, PCSE**
	(1) m1DevOLSrse *b/se*	(2) m1DevOLSpcse *b/se*	(3) m1DevFErse *b/se*	(4) m1DevFEpcse *b/se*	(5) m2DevOLSrse *b/se*	(6) m2DevOLSpcse *b/se*	(7) m2DevFErse *b/se*	(8) m2DevFEpcse *b/se*
L3.Civil War	−0.692***	−0.692***	−0.259	−0.259**	−0.514***	−0.514***	−0.152	−0.152*
	(0.063)	(0.097)	(0.161)	(0.081)	(0.089)	(0.057)	(0.094)	(0.064)
L3.Ethnic War	−0.268***	−0.268***	−0.286	−0.286**	−0.116	−0.116**	−0.177	−0.177**
	(0.052)	(0.041)	(0.268)	(0.091)	(0.079)	(0.037)	(0.258)	(0.061)
L3.Int'l War	−0.394**	−0.394	−0.061	−0.061*	−0.492***	−0.492*	0.042†	0.042**
	(0.123)	(0.249)	(0.041)	(0.028)	(0.079)	(0.249)	(0.022)	(0.016)
Polity Score	0.045†	0.045**	0.173**	0.173***	−0.028	−0.028**	−0.006	−0.006
	(0.026)	(0.015)	(0.056)	(0.023)	(0.037)	(0.011)	(0.015)	(0.007)
Sub-Sahara Africa	5.057***	5.057***			7.447***	7.447***		
dummy	(0.292)	(0.161)			(0.475)	(0.194)		
Log Off. Dev. Asst.					0.095	0.095	−0.239*	−0.239***
					(0.153)	(0.093)	(0.110)	(0.067)
Log GDP/capita					−0.562	−0.562*	0.564†	0.564*
					(0.518)	(0.222)	(0.315)	(0.263)
Refs. in country					0.000	0.000	−0.000	−0.000
(000s)					(0.000)	(0.000)	(0.000)	(0.000)
Log Health Care					2.048***	2.048***	−0.172	−0.172*
Exp./capita ($ curr.)					(0.512)	(0.128)	(0.246)	(0.087)
Observations	1393	1393	1393	1393	938	938	938	938
Countries		98	98	98		97	97	97
R^2	0.227	0.227	0.850	0.850	0.337	0.337	0.973	0.973
Root Mean Sq. Err.	4.647	4.647	2.048	2.123	4.572	4.572	0.926	0.978
Rho			0.835				0.971	

Table entries are linear regression coefficients, with standard errors in parentheses. Constants have been omitted for presentation. P: Pooled (OLS/GLS) model; FE: Fixed Effects estimator. RSE: Robust Standard Error; PCSE: Panel-Corrected Standard Error.
† p<0.10, * p<0.05, ** p<0.01, *** p<0.001

Table 4: First differenced regression results

Response Variable: Year-on-year difference in HIV Prevalence				
	Models			
	Sub-Saharan Africa		Developing countries	
Independent Variables	Pooled, PCSE	Fixed, PCSE	Pooled, PCSE	Fixed, PCSE
D.Civil War	−0.022	−0.035∗	−0.008	−0.014
	(0.021)	(0.017)	(0.019)	(0.014)
D.Ethnic War	−0.074	−0.048∗	−0.045	−0.042
	(0.071)	(0.019)	(0.050)	(0.028)
D.Int'l War	0.006	0.004	0.006	0.005
	(0.023)	(0.009)	(0.021)	(0.010)
D.Polity Score	−0.007	−0.001	−0.002	0.001
	(0.010)	(0.005)	(0.005)	(0.004)
D.Log GDP/capita	−0.212	−0.447∗	−0.094	−0.236
	(0.235)	(0.216)	(0.208)	(0.171)
D.Log Off. Dev.	−0.096+	−0.055	−0.051	−0.033
Asst.	(0.056)	(0.047)	(0.045)	(0.035)
D.Refs. in country	0.000	−0.000+	−0.000	−0.000
(000s)	(0.000)	(0.000)	(0.000)	(0.000)
D.Log Health Care	−0.082	−0.112+	−0.049	−0.057
Exp./capita ($ curr.)	(0.073)	(0.067)	(0.069)	(0.066)
Sub-Sahara Africa			0.119∗	
dummy			(0.055)	
Constant	0.155∗∗∗		0.030∗∗	
	(0.039)		(0.011)	
Observations	338	338	678	678
Intercept				
Countries	41	41	91	91
R-Sq.	0.0129	0.588	0.0281	0.577
Root Mean Sq. Err.	0.575	0.409	0.408	0.296

Table entries are linear regression coefficients, with standard errors in parentheses. Constants have been omitted for presentation. P: Pooled (OLS/GLS) model; FE: Fixed Effects estimator. RSE: Robust Standard Error; PCSE: Panel-Corrected Standard Error. † p<0.10, * p<0.05, ** p<0.01, *** p<0.001