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# AIDS and Conflict: Micro Evidence from Burundi<sup>1)</sup>

**Abstract:** This paper studies the relationship between civil war and HIV/AIDS in Burundi at the micro level. The case of Burundi provides interesting grounds of analysis, as seroprevalence rates are heterogeneous across the country, the serological and conflict data for Burundi are of good quality and conclusions can inform HIV/AIDS policies in Burundi and other fragile states. Ordinary least squares and instrumental variable results indicate that there is no empirical relationship between seroprevalence at the general population level and three measures of local conflict intensity within provinces. This evidence could imply that areas that are relatively more conflict affected do not need to be prioritized over others in terms of HIV/AIDS policies. Further research should focus on individual rather than geographical exposure to conflict. There are likely certain groups and individuals at risk in the general population that need special attention after conflict. Furthermore, violence changes societies, in particular gender relations, thereby indirectly feeding and possibly fueling the dynamics of the epidemic.

**Keywords:** HIV/AIDS; civil war; micro evidence; Burundi.

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## 1 Introduction

This paper studies the empirical relationship between the prevalence of HIV/AIDS and civil war at the micro level in Burundi. In the early 2000s, it was a

common assumption among donors and policy makers that conflict leads to a surge in sexually-transmitted diseases such as HIV/AIDS. Similarly, the epidemic was considered a threat to international security and state stability. The debate even entered the United Nations Security Council in 2000 (see McInnes 2006; de Waal 2010a).

Once the topic was under the limelight, the pressure to intervene mounted, while the relevant “knowledge base” on the security-HIV/AIDS nexus was “severely limited and constrained” (Barnett and Prins 2006: p. 368). At the same time, this increased attention – driven by security concerns – more generally promoted the fight against HIV/AIDS, and critiques warned that this fight should not be reduced to a “narrow national interest” thereby neglecting altruistic motives and moral obligations (Elbe 2006).

A series of transmission channels that link conflict and HIV/AIDS have been repeatedly proposed in the literature, some of them fueling and others dampening the spread of the epidemic (USIP 2001; Hankins et al. 2002; Save the Children 2002, all discussed in Spiegel 2004; Mock et al. 2004). The principal increasing factors – discussed in Section 2 – are displacement, rape, interaction of HIV/AIDS infected military and rebels with local populations, lack of infrastructure, medical services and information about the disease, as well as the proliferation of prostitution. Decreasing factors include reduced mobility due to insecurity and destroyed roads as well as better medical services in refugee camps, which was first formally pointed out by Spiegel (2004). The relevance of these factors is thought to depend on initial prevalence rates of local populations, surrounding communities or military and armed groups, the length and intensity of the conflict (Spiegel 2004), as well as the geography, scale and dynamics of conflict and its aftermath (Mock et al. 2004).

Subsequent studies and evidence caught up with policy makers and it became clear that conflict is not necessarily associated with a spread of the epidemic. The relationship is multi-faceted and complex, as well as heterogeneous across countries and time (Spiegel 2004; Spiegel et al. 2007). In fact, early evidence on the subject has been mainly anecdotal, and subsequent findings have not lend themselves to generalization as various authors have pointed out in the meantime (Anema et al. 2008; Becker et al. 2008; de Waal 2010b). The AIDS, Security and Conflict Initiative (ASCI) has put forward one of the most comprehensive studies on the subject and key findings from 29 studies are summarized in a lead paper by de Waal (2010b). Across a variety of research methods, regions and scholars, there is little evidence of direct, generalizable and significant channels between security, conflict, governance and HIV/AIDS. The study argues that the early beliefs have shaped policies and institutions much more than the realities of conflict and HIV/AIDS themselves.

While the literature on HIV/AIDS and civil war has provided foremost descriptive analysis, correlations and cross-country studies, little attention has been paid to estimating causal effects and providing micro evidence within a country. In particular, increasing rates during and decreasing rates post civil wars may be due to more general time trends, economic aid dynamics or new survey methodologies than to civil war per se.

This paper makes therefore two main contributions. First, it provides micro-level evidence at the individual level using serological and local conflict data from Burundi. The focus on the micro level is a complement to existing cross-country evidence, since conflict and seroprevalence may vary substantially within a country. Hence this relationship should not be studied only at an aggregate level. More generally, this paper fits into the rapidly expanding empirical literature of the impact of conflict on households and individuals. Verwimp et al. (2009) argue for a micro-level rather than country-level approach to study conflict and the behavior and welfare of individuals, households, groups or communities. Blattman and Miguel (2010) provide an overview of this emerging socioeconomic literature on civil war at the micro and macro levels.

Second, this paper attempts to provide causal estimates. The importance of a sound identification strategy to estimate causal effects rather than correlations (or spurious models) has been forcefully underlined in this recent civil war literature (Blattman and Miguel 2010). For example, Miguel and Roland (2011) use the plausibly exogenous distance to the 17th parallel as an exclusion restriction to study the long-run economic effects of the US bombing on districts in Vietnam. In another methodologically related study, Miguel et al. (2004) use rainfall shocks to study the causal impact of GDP on civil war.

Naive estimates may be biased due to omitted variables, or reverse causality. Conflict is likely to take place in communes that have characteristics that cannot be fully measured (Arcand and Wouabe 2009), which are also correlated with HIV/AIDS prevalence and behavior. There may also be measurable characteristics that are falsely omitted in empirical models. Even more so, the literature on this topic has often relied on simple bi-variate correlations. However the omission of key factors can confound the link between conflict and HIV/AIDS. Suppose, for instance, that remote areas of Burundi are largely untouched by conflict. If remote areas have in addition low prevalence rates (since individuals live in isolation) and one fails to capture this empirically, then simple correlations are spurious due to the confounding omitted variable (remoteness). One would probably find that conflict is positively correlated with prevalence. However this could be due to remoteness and not the impact of conflict per se. Or assume that poorer communes are more vulnerable to conflict and HIV/AIDS, and we do not adequately control for income (there is no measure of income in many prevalence surveys like the

one used in this paper), then the correlation between conflict and HIV/AIDS may be driven by the omitted income variable. Also reverse causality cannot be ruled out. Using Ugandan data from the 1990s, J. Weinstein (Unpublished manuscript) finds that HIV/AIDS prevalence can be related to violence, in this case crime rates.

My strategy to *identify* these causal effects of conflict on HIV/AIDS is based on an instrumental variable strategy that uses the exogenous presence of entry and exit points to rebel safe havens. I use these points to identify exogenous variations in conflict that then feed into HIV/AIDS outcomes. This instrumental variable strategy allows to estimate causal effects, if such points have no direct but only an indirect impact on HIV/AIDS via the conflict channel, and if these points are statistically relevant predictors of conflict in a first stage regression model.

The impact of conflict on households and communities in Burundi has been extensively studied. In terms of conflict and health, Bundervoet et al. (2009) find important impacts of civil war exposure on the health status of Burundian children. While some qualitative evidence suggests that conflict has contributed to the spread of the disease in Burundi (see for instance, Seckinelgin et al. 2008), there is no quantitative evidence to that effect. Seroprevalence rates are heterogeneous across the country and I seek to find if this is driven by local conflict intensity. It turns out that civil war has had no lasting effect on snapshot HIV/AIDS seroprevalence in Burundi in 2007. This confirms the previously mixed evidence at the macro level and rejects the common hypothesis among policy makers that conflict leads to higher seroprevalence rates in the *general* population. The work by Seckinelgin et al. (2008) indicates that indeed there are no direct, quantifiable impacts. Conflict changes a society and its gender relations, and therefore indirectly influences the nature and spread of the disease. However my paper only considers direct impacts of local conflict exposure on the epidemic at the general population level.

## 2 Transmission Channels and Impacts

Most assumptions about the channels between HIV/AIDS and conflict have been challenged and further evidence that takes into account local contexts is required according to Becker et al. (2008). Early evidence on channels has been criticized as anecdotal (Spiegel 2004). In the following discussion of the channels, I focus on the ones that are relevant to the Burundian context and the datasets at hand.

First, conflict has fueled rape and transactional sex across countries and time (for a detailed history, see Leatherman 2011). Particular large scale rape was feared to feed into prevalence rates (Hankins et al. 2002). While cases of rape

are hard to estimate, and survey evidence suffers from under- or over-reporting, upper and lower bound estimates have been used in numerical prevalence simulations: Anema et al. (2008) predict relatively small effects of large scale rape on prevalence rates in the *general* population. The authors cite work by Halperin et al. (2004) that transmission rates for one-shot sexual relationships are relatively low compared to repeated sexual intercourse, which in turn explains why rape has little impact on prevalence in the general population. Applying similar models, Supervie et al. (2010) arrive at more pronounced impacts, and in the case of Burundi, intermediate ones. More simulations by Foss et al. (2009) show that conflict and rape can have substantial impacts on prevalence rates of individuals and vulnerable sub-populations. The authors argue that the focus on general population rates misleads the debate on HIV/AIDS and violence against women.

Second, the general wisdom and early evidence put prevalence rates among soldiers above those of the general population. It was feared that risky sexual interaction between the military and civilian population may intensify during and after conflict. During World War I sexually transmitted diseases were said to be substantially higher among soldiers, which may have resulted in spill-overs into civilian populations (Selvin 1984, cited by Hankins et al. 2002). In the case of Sierra Leone, it has been argued that sexual intercourse between soldiers coming from HIV/AIDS infested countries and locals may have fueled prevalence rates (Salama et al. 1999, cited by Hankins et al. 2002). Early unsubstantiated accounts like these are rarely based on empirical models and hard evidence. Whiteside et al. (2006) show that soldiers usually do not have a higher prevalence of HIV/AIDS than civilians, underlining that prevalence is a function of demographics of the army, patterns of deployment, local prevalence, and military health policies. Moreover multi-country evidence summarized in de Waal (2010b: p. 1) rejects early, “overstated” fears. As epidemics “mature,” prevalence rates of civilians and military personnel converge. Using prevalence data from UN peacekeeping forces, Lowicki-Zucca et al. (2009) demonstrate that blue helmet missions do not contribute to augmenting local prevalence rates. In fact, peacekeepers often come from countries with relatively lower prevalence rates than the ones found in conflict countries.

State capacity, institutions, financial resources, infrastructure, foreign aid and medical services may dwindle during conflict and remain in a sorrow state thereafter. There is a lot of anecdotal evidence on this channel. Prevalence rates are feared to surge in some of Uganda’s conflict zones according to reports on the disruption of health services.<sup>1</sup> After armed conflict in Cote d’Ivoire in 2002, survey

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<sup>1</sup> See “System Disruption & HIV AIDS,” Albon, C., June 16, 2008, available at: [www.conflicthealth.com/system-disruption-hiv-aid](http://www.conflicthealth.com/system-disruption-hiv-aid), accessed on: January 1st, 2013.

evidence by Betsi et al. (2006) documents a waning health system, as well as a lack of health staff and antiretrovirals in urban areas. In some cases this may be cushioned by the interventions of non-governmental organizations and foreign donors. In the Democratic Republic of the Congo, Culbert et al. (2007) report that Doctors without Borders could maintain HIV/AIDS prevention and care activities over an extended period of instability. The reason was that “chronic conflict” unlike “frequent instability” did not impede activities. D’Awol (2008) studies HIV/AIDS policies in post-conflict Sudan and underlines that the combined impact of weak institutions and a waning health infrastructure may further deepen the vulnerability of the population to HIV/AIDS.

In contrast, Spiegel (2004) points out that conflict may reduce mobility and accessibility. Roads are often severely damaged during war, which renders access to remote communities difficult and decreases travel. This in turn can reduce the spread of the disease. Quantifying such patterns empirically and causally is no easy task, for all these factors and forces are inherently intertwined and endogenous.

Displacement and forced migration may contribute to a geographical spread of the disease. In Angola, HIV/AIDS patterns appear to have shifted considerably during the civil war. The disease is said to have wandered from the north to the center and south of the country, trailing front lines and tracking mass displacements (Santos-Ferreira et al. 1990, cited by Hankins et al. 2002). However, in an influential study Spiegel et al. (2007) provide a review of HIV/AIDS prevalence surveys and studies within communities that have been directly and indirectly affected by conflict across seven sub-Saharan African countries. The authors detect no evidence that infections rise during conflict periods, irrespective of prevalence rates prior to conflict. In particular, infection rates in urban areas remain stable. Of 12 refugee camps, nine had a lower prevalence than their immediate environment. The authors cannot document that displacement and wide-scale rape contribute to a raise in prevalence rates. Furthermore, some refugees may have better access to humanitarian assistance, medical services and infrastructure in refugee camps or in neighboring countries than in their home countries at war.

Rather than looking at various channels, some studies have estimated correlations between prevalence rates and conflict. Notably, Strand et al. (2007) report a negative spearman rank correlations between HIV/AIDS prevalence and conflict events across 14 sub-Saharan countries. Discussing these results in the context of post-conflict data from Angola, the authors argue that low prevalence in the country compared to other countries can be attributed to civil war. The paper warns that the return of soldiers and refugees may however lead to a rebound in prevalence rates. In this light, preventive measures should be taken. The paper further underlines that one has to distinguish between short and

long-term impacts on prevalence rates. Still, I would argue that simple correlations may be spurious due to general or regional time trends, as well as omitted variables – both observable and unobservable. Such observable variables include foreign aid, infrastructure, GDP and education, and they may be correlated with both conflict and HIV/AIDS, which may result in misleading implications. Iqbal and Zorn (2010) take some of these factors into account and estimate an increasing impact of conflict on prevalence rates in 43 African countries from 1997 to 2005. Differentiating between types of conflict, impacts for intra-state wars are found to be big, and for civil war, small. In a comparable study, Davenport and Loyle (2009) found little evidence of a direct impact of conflict on prevalence rates. However the study highlights indirect effects working via strained resources and state capacity, as well as flows of refugees from high prevalence countries. Of course unobservable variables may still bias the reported correlation. I move beyond correlations at the macro level by looking at the micro level using instrumental variable techniques and fixed effect models to account for unobservable confounders.

At the micro level, Smallman-Raynor and Cliff (1991) use recruitment data of the Ugandan National Liberation Army to demonstrate that the ethnic make-up of fighters is positively correlated with clinical HIV/AIDS reports across Uganda after the conflict. Again, these findings underline that military personnel can be a transmission vector during and after the war. The authors are careful not to claim any causality. Davenport and Loyle (2009) study conflict exposure and HIV/AIDS using the Rwandan Demographic and Health Survey data aggregated across 11 prefectures. Bi-variate scatter plots point to a weak and positive correlation between conflict duration and prevalence. In contrast, conflict intensity does not seem to matter. My study is considerably more disaggregated in that I use individual-level seroprevalence data coupled with conflict data across 102 Burundian communes. I can move beyond bi-variate correlations and provide OLS and instrumental variable models controlling for a large array of individual and commune characteristics, as well as province fixed effects. The latter control for all observable and unobservable characteristics of a given province.

Only few qualitative studies at the micro level have been undertaken, most relevant of which is an ethnographic study on Burundi by Seckinelgin et al. (2008, 2010). The authors trace out subtle, increasing impacts of conflict on post-conflict dynamics of HIV/AIDS, moving outside quantifiable channels. More specifically, the conflict and its resolution have re-shaped cultural practices, kinship, inheritance, marital laws and gender relations. In turn, these societal and cultural changes have influenced how men and women view, live and cope with the disease. Women have become more vulnerable to the disease.

### 3 Empirical Specification

The paper seeks to answer a series of questions on the relationship between HIV/AIDS and civil war. Most importantly, it investigates the impact of different measures of local civil war exposure on individual HIV/AIDS seroprevalence. Yet as noted there are many potential channels of how civil war can affect an individual's likelihood of infection. To investigate some of these channels, I ask the following empirical questions, using the individual data from the 2007 Burundian HIV/AIDS prevalence survey: (i) How does local conflict intensity affect the availability and knowledge of HIV/AIDS test centers? This is a binary variable that takes on a value of one if the individual knows where he can get an HIV/AIDS test in his community and zero if he does not; (ii) Does conflict impact on self-reported testing behavior? This is a binary variable that takes on a value of one if the individual reports to have done a HIV/AIDS test and zero if he does not. People in war zones might have been exposed to less information on the importance of testing or do not consider long-term health a priority; (iii) Is individual risk behavior changed by local conflict? The dependent variable takes on the value of one if people used a condom during their first sexual intercourse, (iv) I also employ a binary variable if an individual knows that condoms protect against HIV/AIDS; (v) Does local conflict increase self-reported incidences of sexual violence such as rape? Again this is a binary dependent variable.

I aim to estimate the impact of civil war as measured by communal-level conflict intensity on the probability that an individual is HIV positive and other individual measures of HIV/AIDS. Let  $i$  denote individuals,  $h$  households,  $c$  communes, and  $N$  the sample size. I estimate the following OLS model with a geographically decomposed error term as in Arcand and Wouabe (2009):

$$Y_{ihc} = x_{ihc}\alpha + w_c\beta + \lambda_c + \eta_{ihc} \quad (1)$$

where  $Y_{ihc}$  is the  $N \times 1$  outcome variable (e.g., serological status, condom use, etc.),  $x_{ihc}$  includes individual, household and commune co-variates,  $w_c$  is a measure of conflict intensity in the commune that is home to the individual  $i$ . Further,  $\lambda_c$  are commune unobservables, while  $\eta_{ihc}$  are individual- or household-level unobservables.

In the baseline results, I provide OLS estimates of equation (1). However, I cannot be sure that unobservables are correlated with the conflict measure and thus bias my estimates of  $\beta$  (a similar empirical situation is discussed in a conflict study on Angola by Arcand and Wouabe 2009). Conflict is likely to take place in communes that have characteristics that cannot be fully measured, which are also correlated with HIV/AIDS prevalence and behavior. These will show up in  $\lambda_c$ . The omission of these factors can confound the link between conflict and HIV/AIDS.



To alleviate the bias stemming from unobservables I can only include province effects, since, needless to say, commune effects would not allow identifying  $w_c$ . To remedy this, I employ an instrumental variable strategy that is outlined in the following section. The instrumental variable is used to generate exogenous variation in conflict to estimate causal links rather than correlations. I can also investigate if OLS results are robust to instrumental variable results. Instrumental variables can be used to identify exogenous variations in an otherwise endogenous system. If valid, such instruments can cut off the correlation of  $w_c$  and the error term in equation (1). Put simply, I first estimate the impact of the exogenous instrument on conflict (all co-variables are included). Then I obtain the predicted level of conflict from this first stage, and use it instead of the actual one  $w_c$  in my HIV/AIDS regression model 1. In such a two-step procedure, standard errors need to be adjusted.

Since I analyze post-conflict outcomes at the individual level, the impact of conflict is likely to be different across age groups. Therefore I report full sample as well as separate age group results (15–49; 15–20; 21–30; 31–49) for all outcome variables. In addition, there may also be an interaction of age-cohort province effects, as well as the interaction of these two factors. For instance, age groups may have differential access to information about HIV/AIDS across communes. While I do not have administrative data on the timing of, say, condom introduction/information campaigns across Burundi's provinces, I can proxy this by adding *cohort-province* dummies in the full sample regressions, rather than only separate cohort and province dummies.

Finally note that all dependent variables are binary with varying numbers of zeros. In discrete choice models with endogenous regressors, it is easier to work with the linear probability model, since then standard linear model methods can be applied, provided standard errors adjust for heteroskedasticity (Cameron and Trivedi 2005: p. 437). Another estimation issue is that positive seroprevalence tests are relatively rare in the dataset. For the baseline results I use a linear model. However, probit models and rare events logit models yield qualitatively similar results and are available upon request.

## 4 Identification Strategy

An innovation of this article compared to existing studies on HIV/AIDS and civil war is its causal identification strategy. A series of unobservable or omitted factors are both correlated with conflict intensity over the long civil war period and individual HIV/AIDS status in 2007. Ignoring these confounding factors may bias estimates as shown in the previous section.

The causal identification strategy in the light of such unobservables is based on the movement of rebels during the Burundian civil war. It exploits geographical variation in the intensity of violence experienced by different communities due to the presence of exit and entry points to and from rebel safe havens (mostly located in national parks and forests). Unlike the study by Voorst et al. (2010) on conflict and behavior in economic experiments, I cannot use the distance to the capital Bujumbura as an exclusion restriction, as it is well known that HIV/AIDS often originates or spreads more easily from urban centers and thus I cannot rule out a direct impact of the excluded instrument on my response variables. In a similar study, Arcand et al. (2011) use the distance to rebel headquarters as an exogenous variation in conflict (in this case landmine contamination).

I will compare communes within a given province that have similar health and economic potentials, but that differ in the presence of such strategic exit and entry points to rebel safe havens. Exit and entry points include, for instance, mountain passes, paths and trails. These points are plausibly assumed to serve as starting or end points for rebel attacks, but are not directly related to the response variables like seroprevalence of individuals or correlated with omitted variables. I will generate exogenous variation in the intensity of conflict using a binary dummy variable that takes on a value of one if the commune features entry/exit points to safe havens. The presence of entry and exit points should have no direct effect on HIV prevalence, but only indirectly through conflict intensity. Due to the unfortunate presence of such points, communities are relatively more exposed to conflict. This exogenous variation in conflict intensity can then be used to causally identify the resulting impact on HIV/AIDS prevalence. Figure 1 gives an example of an entry point located in the commune of Nyanza-Lac in the province of Makamba. The arrows on the map indicate how fighters entered Makamba province by boat into Nyanza-Lac or crossed the border from Tanzania. They would aim for the mountain chains Baraga and Ndenzwa where their safe havens or camps were located. The main attacking point was thus the commune of Nyanza-Lac (in particular near the fishing port of Kabonga). This was purely for strategic reasons to reach the safe haven, located there for geographical reasons. Also commune Mabanda in the same province served as an exit point for attacks on the villages of Gitara and Musenyi, however this spot was primarily used by the Burundian army to launch attacks on rebels, and hence I do not use it. As I include province dummies, I compare the impact of entry/exit points on commune level conflict within a province while controlling for a series of observable variables. The hypothesis is that Nyanza-Lac witnessed relatively more conflict than all other communes in this province due to the unfortunate presence of an entry point to a rebel safe haven. As mentioned, this entry point has plausibly no direct effect on HIV/AIDS prevalence, but only



**Figure 1:** Identification Strategy - Entry Point to Rebel Safe Haven in the Province of Makamba. Map: Based on UNHCR, Global Insight Digital Mapping © 1998 Europa Technologies Ltd.

indirectly through conflict intensity. Of course, this does not mean that other communes were unaffected by conflict.

To be valid, my instrument must satisfy the following conditions: (1) the instrument must be a significant predictor of conflict; (2) there should be no direct effect of the instrument on HIV/AIDS, but only an effect via conflict intensity.

For further robustness, I include a host of co-variates that are potentially correlated with conflict and HIV/AIDS outcomes, such as a commune’s remoteness proxied by terrain elevation, as well as the distance of communes to Bujumbura and to their respective provincial capital. Furthermore, I include population density estimates at the commune level. In addition, individual and household-level variables include a rural dummy, education level, gender, age, religion, employment, family composition, relationship status, ownership of television and radio receivers and marriage status.

The first-stage model estimates the impact of the instrument  $D_c$  on conflict:

$$W_c = X_{ihc} \gamma + D_c \pi + \nu_{ihc} \tag{2}$$

with  $\pi > 0$ . To be a valid instrument, the coefficient associated with  $D_c$  must be statistically significant.

The predicted level of conflict (now exogenous) is then used instead of the actual variable in the HIV/AIDS models in equation (1). If valid, this strategy cuts off the correlation of conflict with the error term and thus solves issues of endogeneity due to measurement error, reverse causality and omitted variables.

HIV/AIDS at the individual level may be affected by communal and individual level conflict exposure, and a combination of the two. This paper looks solely at communal conflict exposure during the civil war and HIV/AIDS outcomes in post-conflict times. In other words, this is *snapshot* geographical evidence, in that I do not consider the dynamics of this relationship for individuals over time. I want to inform policy makers in post-conflict contexts as to whether regions that had been most exposed to war also need relatively more resources to fight the disease.

However, individual conflict exposure or displacement history may also determine the HIV/AIDS status. Unfortunately, the Burundian HIV/AIDS survey does not include information on the place of residence of people before and during conflict, nor measures of individual conflict exposure and a displacement history. In other words, I cannot discuss if certain conflict-affected sub-populations (regardless of their place of residence) need special attention in terms of HIV/AIDS policies.

The question is then whether the validity of my results on communal conflict exposure is endangered by unobserved individual level conflict exposure or displacement history. For three main reasons, I argue that this is unlikely to be a problem: First, the study by Spiegel et al. (2007) provides evidence that displacement does not necessarily come with increased HIV/AIDS prevalence rates. The authors report rates for Burundian refugees in Tanzania. In 2003, three camps (host regions) had rates of 4.5% (2.0%), 1.7% (2%) and 1.6% (3.7%). In other words, rates are similar to those of hosting regions and the Burundian prevalence data used in my paper. This suggests that individual displacement history is unlikely to play a major role.

Second, while displacement was large-scale and touched many households, survey evidence from Burundi (UNFPA, 2002<sup>2</sup> as cited by Bundervoet et al. 2008) shows that displacement lasted on average less than a year and most households returned to their provinces. Also the UNHCR numbers suggest that few Burundians remain displaced. In other words, the 2007 HIV/AIDS prevalence survey is unlikely to suffer from attrition bias due to households that remain displaced. Furthermore, the time of displacement was small in relation to the overall duration of the war.

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<sup>2</sup> United Nations Population Fund Agency (2002). Situation démographique et sociale au Burundi. Résultats de l'enquête sociodémographique et de santé de la reproduction. Burundi: Département de la population.

Third, the instrument may be correlated with unobservable characteristics such as the displacement history and the likelihood of returning home. If this were the case, one would see an endogenous sorting of observed characteristics with respect to the instrument. In particular, the instrument should not be correlated with pre-determined and demographic characteristics of adults such as age, literacy or gender. I provide such placebo or falsification regressions in Section 6 that reject such patterns. To sum up, most refugees returned to their provinces and they have similar (sometimes lower) HIV/AIDS prevalence rates than the general population. The instrument does not suffer from endogenous sorting and thus is unlikely correlated with the likelihood of returning. I would therefore argue that even in the presence of massive displacement, I can estimate the impact of communal violence on post-conflict HIV/AIDS outcomes. Nevertheless, I also argue in the discussion of my results that more evidence is needed on individual rather than communal conflict exposure.

## 5 Data

This paper uses the 2007 Burundian HIV/AIDS serological and behavioral survey among the general population aged 15 to 49 years (Enquête combinée de surveillance des comportements face au VIH/SIDA et d'estimation de la séroprévalence du VIH/SIDA au Burundi chez les personnes âgées de 15 à 49 ans). The survey was undertaken by the CEFORMI (Centre de formation et de recherche en médecine et maladies infectieuses, Burundi) in the 17 provinces of Burundi, sponsored by the CNLS (Conseil national de lutte contre le SIDA, Burundi) and financially and technically supported by the World Bank, the Global Fund, the CNR (Centre national de référence, Burundi), the World Health Organization, the United Nations Population Fund Agency and UNICEF. The data collection took place in December 2007. Note that the Burundian civil war broke out in 1993 and ended in 2005 (for details, see for instance, Lemarchand 1996; Chrétien and Dupaquier 2007). This means that at the time of the serological survey, the incubation time had been sufficiently long to assess the impact of the war period on the snapshot prevalence of HIV/AIDS at the individual level. As it is standard in regression models, I work with the unweighed data. To obtain representative descriptives, the data needs to be weighed, as it is done in the accompanying reports (CEFORMI 2008): Sero-prevalence in the general population above 18 months is 2.97%, more specifically 4.59% in urban, 4.41% in semi-urban, and 2.82% in rural areas. General populations rates attain 2.81% in men and 2.91% in women. In addition to the serological component, the individual survey covers ages 15–49 years and includes the

following themes: basic individual characteristics (e.g., gender, age, education, marital status), sexual activity (regular, irregular, commercial), knowledge and use of condoms, self-reported information about sexually transmitted diseases, knowledge and opinions on HIV/AIDS, knowledge about public interventions, and infrastructure related to HIV/AIDS.

Clearly, all measures except the serological test are self-reported and might therefore suffer from over- or under-reporting due to, say, taboos and cultural beliefs. An extensive ethnographic study by Seckinelgin et al. (2008) sheds light on the inter-linkages between HIV/AIDS, gender and conflict in Burundi (as cited by deWaal 2010b). Based on qualitative interviews, the study underlines that sexual behavior of the young generation has become more liberal since the conflict despite the continued domination of men in society and sexual matters. Like in many countries, sex and, in particular, extramarital relations are taboo. As a consequence, HIV/AIDS is a sensitive topic. There is also an important gender component interrelated to conflict and the epidemic.

I account for this problem in three ways. First, I can estimate separate models by age groups and gender to alleviate inter-generational differences in behavior, reporting and conflict exposure. Further, I include age cohort-province dummies to control for cohort differences that vary across provinces. These measures should increase efficiency. Second, from a statistical point of view, measurement error in the dependent variable of a linear model (for instance, self-reported condom use) solely affects statistical efficiency, that is to say, standard errors associated with coefficients may be inflated. The good news is that my estimated coefficients are still consistent (for a discussion of measurement error, see Cameron and Trivedi 2005). Differences in sex taboos are unlikely correlated with conflict or my instrumental variable. Third, I use many different self-reported measures of HIV/AIDS related behavior, exposure and knowledge, all of which suffer from varying degrees of measurement error. And I can contrast these self-reported measures with the objective serological test. If results across outcome measures are qualitatively similar, this gives a reliable indication of the impact of conflict on HIV/AIDS. That said, under-reporting has to be kept in mind when interpreting results individually.

I merge three communal measures of conflict intensity with the HIV/AIDS survey among the general population. The first measure of conflict intensity stems from local news-based events data provided by Cyrus Samii at New York University. The latter was compiled by local research assistants and existed only in hand-written form. I have digitized and coded these records. From these local news data, I use the total number of violent events per commune from 1993 to 2003. While the data are very detailed, there is a possibility that local news underreport bad news for the government. Therefore, I use two

additional indicators. I obtained data from the United Nations High Commission for Refugees (UNHCR) in Burundi about the total number of repatriations by commune up to 2010. The data can be further disaggregated by age and gender. Background information and data are available on the Burundian UNHCR website.<sup>3</sup> According to the dataset, between 2002 and 2010 a total of 506,289 Burundians were repatriated. And by the end of 2012, the UNCHR estimates that 40,000 Burundian nationals were expected to return home. Repatriation numbers are the best proxy of communal displacement intensity. Only a relatively small number of refugees still remain outside of Burundi. My third indicator is taken from the Armed Conflict Location and Event Dataset (ACLED) that records political conflict events in civil wars in over 50 developing countries (Raleigh et al. 2010). Data and background material are publicly available at <http://www.acleddata.com> for the years 1997 to 2009. Variables include dates and locations of conflict events, types of events, actors and fatalities. Sources primarily draw on publications, media and reports from humanitarian agencies. The project is attached to the International Peace Research Institute Funding and is funded by the World Bank, the CCAPS Minerva project and the European Research Council. Since I do not know in which commune the event took place. However, I do have longitude and latitude and I calculate the number of events in a 2 km radius around the commune capital. The correlation between the local news data and the UNHCR numbers, as well as the ACLED data is 6% and 20%, respectively. Finally, the paper uses original data from interviews conducted with eyewitnesses, rebels and military in October 2010. These interviews were conducted by an experienced, local research assistant. The data collection for the identification strategy is also a joint effort with Cyrus Samii. The interviews provide insights on the dynamics of various rebel movements during the civil war and inform the identification strategy. To the best of my knowledge, there are 10 communes with main exit or entry points.<sup>4</sup> Clearly, the identification of these points is no easy task and there may be more. However, I will show that the ones identified are indeed related to measures of conflict exposure. As the current political situation in Burundi is rather tense, most interviews were conducted off-record, however sources or notes can be shared on request.

Summary statistics are presented in Table 1. When interpreting the results in this paper it is important to note that seroprevalence in the regression

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<sup>3</sup> UNHCR Burundi, 2013, available at: [www.unhcr.org/pages/49e45c056.html](http://www.unhcr.org/pages/49e45c056.html), accessed on: January 25th, 2013

<sup>4</sup> Nyanza-Lac, Muruta, Kinyinya, Giharo, Mutumbuzi, Isare, Kamenge, Muhuta, Rumonge and Kabezi.

**Table 1:** Summary Statistics—Household, Individual and Commune Variables, 6076 observations.

	Mean	Median	SD	Min	Max
<b>Conflict Variables</b>					
Violent Events in Local News	4.07	2.00	5.25	0	36
UNHCR Repatriations	6159	1337	12,380	0	65,670
ACLED Events in 2 km Radius	4.32	1.00	14.32	0.00	80
<b>Response Variables</b>					
Seroprevalence	0.03	0	0.18	0	1
Knows Where to get HIV Test	0.22	0	0.41	0	1
Has Taken HIV Test	0.18	0	0.38	0	1
Used Condom (First Intercourse)	0.06	0	0.24	0	1
Thinks Condoms Protect	0.46	0	0.50	0	1
Victim of Sexual Violence	0.03	0	0.16	0	1
<b>Commune Variables</b>					
Exit/Entry Points	0.07	0	0.26	0	1
Population	55,810	53,720	20,770	25.72	113,300
Terrain Elevation	1523	1579	383	773	2382
Latitude	-3.35	-3.31	0.40	-4.33	-2.52
Dist. to Provincial Capital	14.23	13.65	10.39	0	38.80
Dist. to Bujumbura	72.06	68.39	42.07	0.68	165.90
<b>Household and Individual Variables</b>					
Gender	0.56	1	0.50	0	1
Age	29.12	28.00	10.23	15	49
Rural Household	0.86	1	0.35	0	1
Attended School	0.54	1	0.50	0	1
Currently in School	0.16	0	0.37	0	1
Primary Degree	0.40	0	0.49	0	1
Secondary Degree	0.12	0	0.33	0	1
Higher Degree	0.01	0	0.08	0	1
Wage Labor	0.26	0	0.44	0	1
Catholic	0.64	1	0.48	0	1
Muslim	0.04	0	0.19	0	1
Protestant	0.24	0	0.43	0	1
Lives Alone	0.06	0	0.24	0	1
Lives with Parents	0.23	0	0.42	0	1
Lives with Spouse	0.54	1	0.50	0	1
Lives with Partner	0.03	0	0.18	0	1
Married	0.64	1	0.48	0	1
Radio	0.41	0	0.49	0	1
TV	0.07	0	0.25	0	1

sample is 3.2%. Prevalence rates tend to be higher for the oldest age groups: 2.2% are seropositive in the age group 15–20, 3.5% for the ages 21–30 and 3.85%



in the oldest group 31–49. 46% of individuals know that condoms can protect against HIV/AIDS. 22% of individuals know about a test center nearby, while 18% have taken a test. 3% of people have been (self-reported) victims of sexual violence.

There is considerable variance in violence across communes. Local news sources recorded on average four violent events per commune between 1991 and 2003. The distribution across communes is skewed as can be inferred from the median of 2, a large maximum value of 36 and a standard deviation of 5.3. The proxy of displacement are UNHCR repatriations. An average commune has had 6159 UNHCR repatriations up to October 2010 and 4.32 conflict events in a 2 km radius around the commune center according to the ACLED data.

## 6 Reduced Form

Before moving on to the principal results on conflict and HIV/AIDS, I need to check the validity of my identification strategy to estimate causal effects rather than correlations.

**Table 2:** Falsification Tests for Sorting—Regressions of Pre-determined Characteristics on the Exclusion Restriction, 6076 Observations in the Full Age Sample, 2470 for 31–49. All Commune Variables are Included.

Response Variables		
Exit Point	Full Age Sample 15–49	
	Age	Gender
	0.833 (0.812)	–0.032 (0.026)
	Primary Education	Catholic
	–0.010 (0.054)	–0.017 (0.065)
Exit Point	Age Sample 31–49	
	Age	Gender
	–0.595 (0.507)	–0.007 (0.036)
	Primary Education	Catholic
	–0.047 (0.050)	–0.050 (0.065)

Standard errors are clustered at the commune level (N=102) and below estimates.

As a first step, I investigate if my instrumental variable suffers from endogenous sorting of populations. For instance, people living close to entry/exit points may chose to escape to other communes, which would then bias my casual estimates. Put differently, even though geography is exogenous, displacement of local populations may turn it endogenous. If this were true, we should see a correlation of the instrument with pre-determined, observed characteristics of individuals in the survey. I provide such placebo or falsification regressions in Table 2, I regress pre-determined characteristics such as the adult's age, gender, and education on the instrument (exit and entry points to rebel safe havens) and the other commune variables. Table 2 shows that the instrument is uncorrelated with such pre-determined characteristics, both in the full sample and in the age group 31 years and older. In other words, there is no evidence of endogenous sorting with respect to the location of exit and entry points to rebel safe havens, and these points are plausibly exogenous.

Second, these exogenous points must be relevant predictors of communal conflict. Indeed they are: Exit and entry points from and to rebel safe havens are statistically significant determinants of conflict and can thus serve to generate exogenous variation in conflict intensity. Consider the estimates in Table 3.

**Table 3:** First Stage Model—Determinants of Conflict Events in Local news, Number of UNHCR Repatriations, ACLED Events in a 2 km Radius at the Commune Level, Observations, Individual, Household and Commune Variables, As Well As Age Cohort-province Dummies Included.

	Violent Events in Local News	UNHCR Repatriations (100s)	ACLED Events 2 km Radius
Exclusion Restriction:			
Exit Points	4.534 <sup>a</sup> (2.133)	122.304 <sup>a</sup> (61.343)	-4.552 (3.368)
Commune Controls:			
Population (1000s)	-0.002 (0.031)	1.614 <sup>a</sup> (0.571)	0.208 <sup>b</sup> (0.114)
Terrain Elevation (km)	-2.673 <sup>b</sup> (1.430)	-91.884 <sup>a</sup> (27.831)	-0.398 <sup>b</sup> (0.243)
Latitude	1.358 (2.387)	44.867 (96.777)	-8.769 (6.887)
Distance to Provincial Capital	-0.080 <sup>a</sup> (0.038)	2.460 <sup>a</sup> (1.029)	-0.130 <sup>b</sup> (0.074)
Distance to Bujumbura	-0.020 (0.025)	1.784 <sup>a</sup> (0.915)	0.044 (0.051)

Standard errors are clustered at the commune level (N=102) and below estimates, <sup>a</sup>p<0.05, <sup>b</sup>p<0.1.

Results for violent events recorded in local news sources are displayed in the first column. The treatment effect amounts to roughly five events more compared to other communes without such points. Results for UNHCR repatriations are similar. The impact amounts to a sizeable 12,230 additional repatriations. Do note however that the ACLED variable is not correlated with the instrument in this first stage regression model. This is not surprising since the variable is constructed differently than the UNHCR and local news data measures. First, it really only covers the late years of conflict. Second, the database gives the latitude and longitude of events with varying precision based on international news reports. I then calculate the number of events in various radii a 2 km radius around the commune center. It is reassuring that the ACLED data is strongly and positively correlated (20%) with the local news data. However, this measure is thus less precisely tied to the commune than the local news and UNHCR data. In the latter, the conflict event is precisely located by commune. Since the identification strategy is based on a commune story, UNHCR and local news data work better. However the ACLED data can still be informative for the OLS results of the impact of conflict on HIV/AIDS. Therefore, I discuss and report OLS results for all three measures.

Table 3 also presents some selected control variables at the commune level. Total population at the commune level is positively and significantly associated with the number of displaced people. Similarly, events in a 2 km radius are more frequent in more populous communes. Terrain elevation is negatively related to violence. The higher a commune is situated, the lower its conflict exposure is. This result underlines the crucial fact that altitude is an important co-variate in the case of Burundi. Distance to the provincial capital is significantly and negatively correlated with commune violence, however it does so positively with refugees. Finally, the number of refugees is significantly and positively affected by the distance to Bujumbura.

## 7 OLS and Instrumental Variable Results

In this section, I explore the empirical link between conflict and HIV/AIDS in Burundi. The ordinary least squares (OLS) estimates provide within province *correlations* that are robust to controlling for a series of individual and communal characteristics, while the instrumental variable (IV) models go beyond correlations and provide plausibly causal effects. To this end, the previous section has shown that the instruments has predictive power. I can now use the predicted (exogenized) values of conflict from the first stage in the second stage HIV/AIDS

**Table 4:** Individual Seroprevalence Model—OLS, IV Estimates of the Impact of Conflict Events, the Number of UNHCR Repatriations or ACLED events at the Commune Level, 6076 Observations in the Full Age Sample, 1721 for 15–20, 1885 for 21–30, 2470 for 31–49; 2694 Females and 3382 Males. Individual, Household and Commune Variables, As Well As Provincial Dummies are Included. P-values are in Italics, Below Estimates and Standard Errors were Clustered at the Commune Level (N=102).

Sample	Seroprevalence					
	15–49	15–20	21–30	31–49	Female	Male
Local News						
OLS	–3.5E-04 <i>0.59</i>	1.2E-03 <i>0.39</i>	5.5E-04 <i>0.67</i>	–2.7E-03 <i>0.01</i>	1.5E-03 <i>0.18</i>	2.3E-04 <i>0.50</i>
IV	5.6E-04 <i>0.80</i>	2.5E-03 <i>0.46</i>	–2.6E-03 <i>0.56</i>	5.6E-04 <i>0.90</i>	–1.2E-03 <i>0.74</i>	–5.3E-04 <i>0.89</i>
Exogeneity Test	<i>0.69</i>	<i>0.72</i>	<i>0.41</i>	<i>0.45</i>	<i>0.44</i>	<i>0.84</i>
UNHCR(100s)						
OLS	2.0E-05 <i>0.35</i>	2.3E-05 <i>0.47</i>	6.2E-05 <i>0.22</i>	–3.3E-05 <i>0.52</i>	1.4E-07 <i>1.00</i>	3.1E-05 <i>0.35</i>
IV	2.1E-05 <i>0.81</i>	1.0E-04 <i>0.45</i>	–9.4E-05 <i>0.53</i>	1.9E-05 <i>0.90</i>	–4.1E-05 <i>0.71</i>	1.8E-05 <i>0.89</i>
Exogeneity Test	<i>0.99</i>	<i>0.53</i>	<i>0.23</i>	<i>0.70</i>	<i>0.68</i>	<i>0.92</i>
ACLED						
OLS	–1.2E-04 <i>0.61</i>	–1.6E-04 <i>0.76</i>	–5.0E-04 <i>0.17</i>	6.6E-04 <i>0.15</i>	–5.9E-04 <i>0.23</i>	2.3E-04 <i>0.50</i>

models. The question is how these exogenous variations in conflict translate into HIV/AIDS outcomes and if they differ statistically from OLS correlations.

Empirical results for the OLS and linear IV models are presented in Tables 4–7. Each table includes OLS, instrumental variable estimates for the three measures of conflict at the commune level: conflict events from local news, UNHCR repatriations and conflict events recorded in the ACLED database. I provide results for the full age sample and by age cohort. For seroprevalence and sexual violence, I have also added results for men and women separately. P-values to judge statistical significance are provided below all estimates. For each model, I also supply p-values of a Hausman test of exogeneity with the null hypothesis that OLS and IV models are statistically equivalent.

In a first instance, I consider the impact of conflict on seroprevalence. There is no evidence that conflict plays a role. Consider the results for individual seroprevalence status for the full age sample in the first column of Table 4. Both IV and OLS results across three measures of conflict suggest a zero impact of conflict on the likelihood of an individual being tested positive. Are there age differences? In columns 2, 3 and 4 of the same table, I split the sample into three

age groups to quantify intergenerational differences. In the age group of 31–49 year, the OLS estimates suggest that conflict events in local news are significantly and negatively correlated with lower seroprevalence. However this result is not to be trusted in that it is not robust to employing instrumental variables and other measures of conflict. In fact, the number of UNHCR repatriations and ACLED events all remain uncorrelated. Are there gender differences? Splitting the sample by gender yields qualitatively similar (non-)results.

Moving on to Table 5, there is some, albeit very weak, evidence of a negative association between conflict and the likelihood of being a (self-reported) victim of sexual violence. This effect is only found in the age group of 21–30 years and the IV estimates for local news and UNHCR repatriations. All OLS results are inconclusive. However even if one believes the IV effects for the age group 21–30 years, magnitudes are low: four additional events in local news reports in a commune (the sample mean) decrease the likelihood of being a victim of sexual violence by 2.48% ( $4 \times -6.2E-03$ ). In addition, the early literature has posited that conflict fuels rather than dampens sexual violence, thereby increasing seroprevalence. Here

**Table 5:** Sexual Violence Model—OLS, IV Estimates of the Impact of Conflict Events, the Number of UNHCR Repatriations or ACLED events at the Commune Level, 6076 Observations in the Full Age Sample, 1721 for 15–20, 1885 for 21–30, 2470 for 31–49; 2694 Females and 3382 Males. Individual, Household and Commune Variables, As Well As Provincial Dummies are Included. P-values are in Italics, Below Estimates and Standard Errors Were Clustered at the Commune Level (N=102).

Sample	Victim of Sexual Violence					
	15–49	15–20	21–30	31–49	Female	Male
Local News						
OLS	-1.0E-03 <i>0.05</i>	1.5E-04 <i>0.85</i>	-2.4E-03 <i>0.01</i>	-8.9E-04 <i>0.36</i>	-1.2E-03 <i>0.23</i>	-9.4E-04 <i>0.12</i>
IV	-1.4E-03 <i>0.52</i>	3.3E-04 <i>0.87</i>	-6.2E-03 <i>0.05</i>	9.3E-04 <i>0.88</i>	-4.9E-03 <i>0.37</i>	1.5E-03 <i>0.28</i>
Exogeneity Test	<i>0.87</i>	<i>0.93</i>	<i>0.20</i>	<i>0.77</i>	<i>0.49</i>	<i>0.08</i>
UNHCR(100s)						
OLS	2.1E-05 <i>0.27</i>	3.4E-05 <i>0.24</i>	2.6E-05 <i>0.49</i>	1.4E-06 <i>0.97</i>	-1.6E-07 <i>1.00</i>	3.0E-05 <i>0.05</i>
IV	-5.1E-05 <i>0.54</i>	1.4E-05 <i>0.87</i>	-2.2E-04 <i>0.03</i>	3.2E-05 <i>0.89</i>	-1.7E-04 <i>0.40</i>	5.4E-05 <i>0.16</i>
Exogeneity Test	<i>0.38</i>	<i>0.78</i>	<i>0.01</i>	<i>0.89</i>	<i>0.39</i>	<i>0.48</i>
ACLED						
OLS	-9.3E-05 <i>0.52</i>	-2.7E-04 <i>0.16</i>	1.5E-04 <i>0.66</i>	-9.4E-05 <i>0.75</i>	-8.2E-05 <i>0.66</i>	-7.9E-05 <i>0.74</i>

**Table 6:** HIV/AIDS Testing and Knowledge Models—OLS, IV Estimates of the Impact of Conflict Events, the Number of UNHCR Repatriations or ACLED Events at the Commune Level, Observations in the Full Age Sample, 1721 for 15–20, 1885 for 21–30, 2470 for 31–49; 2694 Females and 3382 Males. Individual, Household and Commune Variables, As Well As Provincial Dummies are Included. P-values are in Italics, Below Estimates and Standard Errors Were Clustered at the Commune Level (N=102).

Age Sample	Knows where to do HIV Test					Got HIV Tested		
	15–49	15–20	21–30	31–49	15–49	15–20	21–30	31–49
Local News								
OLS	2.0E-03	1.5E-03	2.9E-03	1.1E-03	4.8E-04	-8.4E-04	1.9E-03	-4.2E-04
	<i>0.50</i>	<i>0.56</i>	<i>0.44</i>	<i>0.78</i>	<i>0.77</i>	<i>0.65</i>	<i>0.50</i>	<i>0.87</i>
IV	2.0E-02	2.1E-02	2.1E-02	1.7E-02	-1.2E-02	7.7E-03	-3.0E-02	-1.6E-02
	<i>0.25</i>	<i>0.04</i>	<i>0.35</i>	<i>0.44</i>	<i>0.05</i>	<i>0.31</i>	<i>0.01</i>	<i>0.04</i>
Exogeneity	<i>0.30</i>	<i>0.06</i>	<i>0.42</i>	<i>0.47</i>	<i>0.03</i>	<i>0.26</i>	<i>0.00</i>	<i>0.03</i>
UNHCR(100s)								
OLS	9.9E-05	2.5E-04	-4.9E-05	4.5E-05	1.0E-04	1.2E-04	1.4E-04	8.6E-05
	<i>0.50</i>	<i>0.05</i>	<i>0.75</i>	<i>0.81</i>	<i>0.21</i>	<i>0.20</i>	<i>0.39</i>	<i>0.42</i>
IV	7.5E-04	8.5E-04	7.6E-04	5.9E-04	-4.4E-04	3.2E-04	-1.1E-03	-5.4E-04
	<i>0.25</i>	<i>0.04</i>	<i>0.33</i>	<i>0.44</i>	<i>0.05</i>	<i>0.36</i>	<i>0.01</i>	<i>0.02</i>
Exogeneity	<i>0.30</i>	<i>0.13</i>	<i>0.29</i>	<i>0.46</i>	<i>0.01</i>	<i>0.55</i>	<i>0.00</i>	<i>0.00</i>
ACLED								
OLS	-1.1E-04	-1.3E-03	4.3E-04	8.0E-04	-4.0E-04	-8.2E-04	-5.5E-04	3.9E-04
	<i>0.92</i>	<i>0.14</i>	<i>0.64</i>	<i>0.63</i>	<i>0.31</i>	<i>0.17</i>	<i>0.54</i>	<i>0.70</i>

I find a very small negative effect for one group. I also investigated this further and regressed seroprevalence on the sexual violence variable and all controls. I do not manage to uncover any impact of violence. These results should nevertheless be taken with a grain of salt for three reasons. First, I do not know when and how often the individual was a victim of sexual violence. The event could have occurred during or after conflict. Second, I have no information about the type of violence. Third, under-reporting may affect statistical efficiency.

The link between conflict and knowledge about the presence of HIV/AIDS test centers is presented in Table 6. As can be seen in column 1, full age sample results indicate that there is no significant link between conflict and knowing where to get tested for HIV/AIDS. This pattern emerges across all measures of conflict, as well as OLS and IV models. Conversely, I detect a significant and positive correlation in the age group 15–20 years. The more displacement a commune experienced (as proxied by UNHCR repatriations), the higher the likelihood that an individual in that youngest age group knows where to get an HIV test, both in the IV and OLS models. Take the OLS estimate: 6159 refugees more in a give commune (the sample mean) increase the likelihood of testing knowledge by 1.5% ( $61.59 \times 2.5E-04$ ). The corresponding IV estimate is bigger and amounts to 5.2%. Likewise, using the IV estimates based on local news, this positive link emerges in the age group 15–20 years. Also note that the corresponding OLS estimates are downward biased. In contrast, there is no correlation with ACLED events. To sum up, conflict increases knowledge about test centers, but only in the youngest age group and effects are small. Clearly, knowledge could also be higher if there were more centers in relatively more conflict-affected communes due to boosted post-conflict investments. However, then we should see similar patterns across age groups. It is interesting to confront these results with actual (self-reported) test taking in columns 5 to 8 (of the same Table 6). Here, it is the older age groups (21–30; 31–49) that have been negatively impacted by conflict. The OLS estimates are biased to zero, while the IV estimates for older age groups suggest that there is a negative correlation of conflict and having taken an HIV/AIDS test. Take the IV results in column 7 and 8: an additional four conflict events (the sample mean) in local news decrease the likelihood that an individual has taken a test by 12% in the age group 21 to 30, and by 6.4% in the oldest age group. An additional 6159 refugees amount to a negative impact of 6.7% and 3.3% in these age groups. The results on knowledge and testing lend themselves to a straightforward interpretation. The older age groups suffered from the longest conflict exposure and correspondingly had fewer chances to get tested during their youth in conflict times. Post-conflict, the younger age groups in conflict-affected areas have caught up or even overcompensated in terms of knowledge.

**Table 7:** Condom Use and HIV/AIDS Knowledge Models—OLS, IV Estimates of the Impact of Conflict Events, the Number of UNHCR Repatriations or ACLED Events at the Commune Level, 6076 Observations in the Full Age Sample, 1721 for 15–20, 1885 for 21–30, 2470 for 31–49; 2694 Females and 3382 Males. Individual, Household and Commune Variables, As Well As Provincial Dummies are Included. P-values are in Italics, Below Estimates and Standard Errors were Clustered at the Commune Level (N=102).

Age Sample	Used Condom (First Sex)				Thinks Condoms Protect			
	15–49	15–20	21–30	31–49	15–49	15–20	21–30	31–49
Local News								
OLS	-1.3E-03 <i>0.16</i>	-1.7E-03 <i>0.10</i>	-3.3E-03 <i>0.21</i>	2.4E-03 <i>0.08</i>	-3.5E-04 <i>0.91</i>	-2.4E-03 <i>0.57</i>	2.4E-03 <i>0.54</i>	2.8E-04 <i>0.95</i>
IV	-5.9E-03 <i>0.08</i>	6.6E-03 <i>0.22</i>	-1.5E-02 <i>0.00</i>	-6.5E-03 <i>0.21</i>	-9.0E-03 <i>0.46</i>	1.1E-03 <i>0.94</i>	-2.6E-02 <i>0.08</i>	2.8E-03 <i>0.85</i>
Exogeneity	<i>0.15</i>	<i>0.09</i>	<i>0.02</i>	<i>0.09</i>	<i>0.44</i>	<i>0.79</i>	<i>0.05</i>	<i>0.86</i>
UNHCR(100s)								
OLS	-9.3E-05 <i>0.02</i>	9.7E-05 <i>0.19</i>	-1.9E-04 <i>0.00</i>	-1.2E-04 <i>0.05</i>	2.4E-06 <i>0.99</i>	4.0E-04 <i>0.07</i>	-4.1E-05 <i>0.69</i>	-1.9E-04 <i>0.29</i>
IV	-2.2E-04 <i>0.10</i>	2.7E-04 <i>0.17</i>	-5.5E-04 <i>0.01</i>	-2.2E-04 <i>0.30</i>	-3.3E-04 <i>0.45</i>	4.5E-05 <i>0.93</i>	-9.5E-04 <i>0.08</i>	9.6E-05 <i>0.86</i>
Exogeneity	<i>0.29</i>	<i>0.30</i>	<i>0.04</i>	<i>0.63</i>	<i>0.40</i>	<i>0.47</i>	<i>0.08</i>	<i>0.55</i>
ACLED								
OLS	5.5E-04 <i>0.07</i>	1.1E-04 <i>0.69</i>	1.2E-03 <i>0.13</i>	-1.2E-04 <i>0.80</i>	-1.5E-03 <i>0.09</i>	-1.4E-03 <i>0.18</i>	-1.5E-03 <i>0.10</i>	-2.7E-03 <i>0.01</i>



I find a similar pattern when it comes to the use and knowledge of condoms, as illustrated in columns 1–4 of Table 7. Conflict-exposed individuals in the mid-age group (21–30) have a lower likelihood of having used a condom during their first sexual intercourse. This tendency is found both in the OLS and IV estimates for the local news and UNHCR conflict measure, but not with the ACLED measure. Conversely, there is only mixed evidence for the youngest and oldest age groups across models and estimates. Full sample results in column 1 are thus driven by the mid-age group. Knowledge about the protective power of condoms against HIV/AIDS in relationship to conflict are tested out in columns 5–6 (of the same Table 7). Again, I only find significance in the age sample 21–30. It is worth noting that results are comparable across the three measures of conflict: IV estimates for UNHCR, local news and the OLS results with the ACLED data point to a negative correlation between conflict and knowledge about condoms use and HIV/AIDS. Take the IV estimate using the local news data in column 7: four more conflict events (the sample mean) decrease the likelihood of condom knowledge by 10%. The coefficient associated with the ACLED conflict proxy (significant at confidence level of 10%) translates four conflict events (the sample mean) into a 0.6% decrease. However the latter is probably biased to zero. To sum up, both knowledge about condoms and their use during first sex tends to be lower in relatively more conflict-affected age groups and communes. This is also in line with findings from HIV/AIDS tests, which decrease with conflict exposure.

## 8 Discussion

The results should be interpreted with caution. While there is weak evidence that conflict exposure reduces knowledge about condoms and preventive behavior (condom use during first sex and HIV/AIDS testing), such patterns are mostly confined to the age group of 21–30 in 2007. Recall that conflict was most intense from early 1993 to 2005. Hence this age group was sexually active in times of conflict. Nevertheless, I cannot find an impact on actual seroprevalence across all age groups. Put differently, some small effect on knowledge and behavior is probably insufficient to translate into a spread of the disease – at least in the first years after the conflict. Simulation models in the spirit of Anema et al. (2008) and Supervie et al. (2010) could be used to simulate impacts on prevalence rates in the general population.

It is also worth repeating that prevalence rates tend to be slightly lower for the youngest age groups: 2.2% are seropositive in the age group 15–20, 3.5% for the ages 21–30 and 3.85% in the oldest group 31–49. However, it would be naive to

attribute such age trends to age-specific conflict exposure, since there are general time trends at work that are unrelated to conflict. Also Spiegel et al. (2007) show that prevalence rates in conflict countries often follow time trends in surrounding countries that have been untouched by conflict.

More generally, the non-results in this paper can be explained by the fact that commonly identified channels between the disease and conflict might be less central than previously thought. Take the Burundian army for instance: according to the Director of health at the country's Defense Ministry, "a survey made in 2011 in the country's military barracks shows that the HIV prevalence in the army is a bit above the country's 2%."<sup>5</sup> While we do not have data on rebel groups, this suggests that military personnel in Burundi has similar prevalence rates and knowledge about the disease as to those of the general population.

When it comes to the displacement channels, there is no correlation of HIV/AIDS with the number of refugees as proxied by UNHCR repatriations in a given commune. This corroborates findings by Spiegel et al. (2007) that three Burundian refugee camps (in Tanzanian host regions) had rates similar to those of hosting regions and the Burundian prevalence data used in my paper. Overall, this suggests that displacement has had no discernible influence on the status of the disease after conflict. It is however necessary to signal a caveat. Due to the lack of data on individual conflict exposure or displacement history, I cannot discuss if certain conflict-affected sub-populations (regardless of their place of residence) need special attention in terms of HIV/AIDS policies. This is however an interesting area of study for future research, provided data are available. In particular, there may be important interactions between local community and individual conflict exposure.

My quantitative results can inform policymakers about direct impacts at the general population level. Yet an over-interpretation of these results for the general population may taint policies and disrupt aid allocation for sub-populations in need. This paper cannot provide evidence against the argument by Foss et al. (2009) that conflict may heighten the vulnerability of individuals and sub-populations, even if we find no global impacts. A sole focus on general population rates may thus be misleading. In particular, individual rather than geographical conflict exposure could be key.

Conflict is not easily measured and the impact of conflict on HIV/AIDS may be more nuanced and manifold than what a quantitative approach may capture. It is hard to map out conflict exposure into changes in prevalence rates in a

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<sup>5</sup> See "Burundi's military lags behind in fight against HIV/AIDS," *China Daily*, November 1, 2012, Xinhua, available at: [www.chinadaily.com.cn/xinhua/2012-01-11/content\\_928566.html](http://www.chinadaily.com.cn/xinhua/2012-01-11/content_928566.html), accessed on: January 25th, 2013.

cross section. And while my three measures of conflict yield similar results, they remain crude. Conflict events are heterogeneous and may not reflect realities on the ground. In fact, the Burundian civil war has been marked by long periods of low intensity interrupted by brief episodes of brutality. My conflict measures may fail to detect more subtle forms of violence. Conversely, high intensity conflict over extended periods could lead to more visible impacts on HIV/AIDS outcomes. Likewise, conflict comes with “social traumas” that feed into HIV/AIDS dynamics (deWaal 2010). My paper finds not direct impacts and is in this sense in line with the ethnographic study by Seckinelgin et al. (2008, 2010) on Burundi. The latter argue that conflict and its resolution have profoundly transformed the Burundian society. And these societal and cultural changes eventually influence the dynamics of the disease.

How do results in this paper fit into the empirical literature on health and conflict at the micro level? Recent studies on the causal impact of conflict on households have provided more generalizable results than mine and the HIV/AIDS literature in general. In particular, child health as measured by child anthropometrics take lasting hits (see Akresh et al. (2012) for the Ethiopia-Eritrea war, Bundervoet et al. (2009) and Verwimp (2011) for Burundi). These papers indicate that both communal and individual conflict exposure affect the livelihood and health of households. In other words, HIV/AIDS may be less of a short-term priority after conflict than more immediate issues of nutrition, schooling and income generation. The adverse impacts of the war on the latter may heighten the vulnerability of certain individuals to HIV/AIDS.

## 9 Conclusion

Results in this paper indicate that at least for the case of Burundi there is no empirical relationship between seroprevalence at the *general* population level and three measures of local conflict intensity within provinces. This evidence implies that relatively more conflict-affected areas do not necessarily need to be prioritized over others in terms of HIV/AIDS policies. Further research should focus on individual (i.e., lifecycle) rather than geographical (i.e., communal) exposure to conflict. There are likely certain groups and individuals at risk in the general population that need special attention during and after conflict. Furthermore, conflict changes societies, in particular gender relations, thereby indirectly feeding into and possibly fueling the dynamics of the epidemic.

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