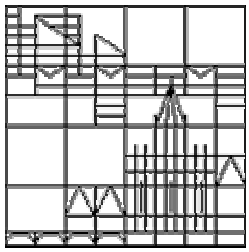
University of Konstanz

Department of Public Policy and Management



"The Killing of the Fittest –

A Quantitative Analysis of HIV/AIDS and Conflict"

Presented to the University of Konstanz,
Department of Public Policy and Management,
In Partial Fulfillment to the Requirements for the MA Degree in Politics and Management
(Diplomarbeit)

By

Sophia Benz

Konstanz, January 31st of 2005

1. Supervisor: Prof. Dr. G. Schneider
2. Supervisor: Prof. Dr. I. de Soysa

Sophia Benz
Stadelhofgasse 11
78462 Konstanz
Matr.-nr: 01/454274
Email: Sophia.Benz@uni-konstanz.de

Contents:

List of Graphs, Tables and Figures

List of Abbreviations

AI	Amnesty International
AIDS	Acquired Immunodeficiency Syndrome
ARRM	AIDS Reduction Risk Model
AVEGA	Association of Genocide Widows
BBC	British Broadcasting Company
CIA	Central Intelligence Agency
ECOMOG	Economic Community Cease-Fire Monitoring Group
GDP	Gross Domestic Product
GNI	Gross National Income
GNP	Gross National Product
HBM	Health Belief Model
HDI	Human Development Index
HIV	Human Immunodeficiency Virus
ICAD	Inter Agency Coalition on AIDS and Development
ICG	International Crisis Group
IOM	International Organization for Migration
ISAC	Inter Agency Standing Committee Task Force on HIV/AIDS in Emergency Settings
Max.	Maximum
Min.	Minimum
N	Sample Size
NGO	Non-Governmental Organization
No.	Number
Obs.	Observations
OLS	Ordinary Least Squares
PPP	Purchasing Power Parity
SD	Standard Deviation
SIPRI	Stockholm International Peace Research Institute
STD	Sexually transmitted Diseases
STI	Sexually Transmitted Infections
U.K.	United Kingdom
U.N.	United Nations
UNAIDS	United Nations Programme on HIV/AIDS
UNDP	United Nations Development Programme
UNECA	United Nations Economic Commission for Africa
UNHCR	United Nations High Commissioner for Refugees
UNICEF	United Nations Children's Fund
UNIFEM	United Nations Fund for Women
UNOMOZ	United Nations Operation in Mozambique
UNPD	United Nations Population Division
U.S.	United States
USAIDS	United States Agency for International Development
USIP	United States Institute for Peace
WDI	World Development Indicators
WHO	World Health Organization

1. Introduction

Discussions regarding HIV/AIDS as well as HIV/AIDS-related research have expanded beyond general health issues and now include analyses of economic and developmental challenges. Increasingly, the epidemic is recognized as a cause and a symptom of underdevelopment and poverty.[1] In January 2000, the U.N. Security Council, for the first time ever, debated a health issue and added another aspect to the fight against the epidemic: U.N. Resolution 1308 (July 2000) highlights the potential threats that the epidemic poses for international security, particularly in conflict and peacekeeping settings. Integrated and horizontal development approaches, therefore, signify a start by addressing refugee populations, military or peacekeeping personnel, and women and children affected by conflict.[2]

Research increasingly discusses and examines the linkage between public health, infectious diseases or HIV/AIDS and war and conflict experience.[3] Partly, research focuses on Sub-Saharan Africa, where both HIV-prevalence and conflict involvement are generally very high. Some recent approaches started to address the transmission of HIV and other sexually transmitted diseases (STDs) in war settings in Central and South Asia (e.g. Afghanistan).[4] Health conditions of refugees in stable camp settings and internally displaced people are also explored in various case studies and reports.[5] However, large-sample, quantitative analysis by political scientists remains limited.

[1] See Stillwaggon (2001); Fredland (1998); ICG: HIV/AIDS as a security issue (2001).
[2] E.g. UNAIDS/UNIFEM joined initiative in Sierra Leone or the UNAIDS/UNHCR strategic plan on HIV/AIDS for refugees for 2002-2004 (see http://www.unaids.org/security/Issues/conflict.html, 16.6.2003); U.S. General Accounting Office Report: HIV/AIDS and U.N. peacekeeping: United Nations faces challenges in responding to the impact of HIV/AIDS on peacekeeping operations (2001); UNAIDS: Fact Sheet 1: HIV/AIDS and security (2002); UNAIDS: Fact Sheet 4: HIV/AIDS and peacekeeping (2002); UNAIDS: Together we can: leadership in a world of AIDS (2001).
[3] See e.g. Davis et al. (2002); Ghobarah et al. (2003); Davis et al. (2003); Hankins et al. (2002); Murray et al. (2002); Levy et al. (1997); Goyer (2001); Van der Heijden (1997); Schönteich (1999); Machel (2001); Fleshman (2001); Burkhalter (2002); Guha-Sapir et al. (2002); Leaning (2003); Connolly et al. (2002); Smith (2002).
[4] See e.g. Hankins et al. (2002) or Subramanian (2002).
[5] See e.g. Cossa et al. (1994), Rey et al. (1995), Amowitz et al. (2002), Mabey et al. (1997), Santos-Ferreira et al. (1990), Carballo & Frajzngier (2001), Carballo & Nerukar (2001), ICAD: HIV/AIDS and displaced people (2001), Sharma (2003), Hsu (2003), Nanayakkara (2003), Salama (1999).

Therefore, this paper examines decisive mechanisms linking the often complex and indirect relationship between HIV/AIDS and conflict, and presents a quantitative analysis that addresses the question of how conflict involvement impacts HIV-prevalence rates. In contrast to most previous studies, the simple multiple linear regression analysis applied here is based on a nearly complete sample of 197 countries.[6]

In addition, whereas previous research has focused heavily on the effects of (civil) war as the most severe form of violent conflict on public health outcomes, the empirics in this paper include all types of conflict (wars, intermediate and minor conflicts) as key explanatory variables. In addition, this study distinguishes between the type of conflict involvement (intense in terms of battle deaths or extensive in terms of duration). Thus, the question as to how significant the type and duration of conflict involvement affects the explanation of HIV-prevalence rates can be addressed. The specification of the dependent variable (formally `public health outcome´ and now `HIV-prevalence´) as well as the generalization of the key explanatory variable (previously `war involvement´ and in this study `all type of conflict involvement´) increases the number of possible falsifying instances. All over, this serves to enlarge the content domain of the applied theory.[7]

To measure the dependent variable, I use HIV-prevalence data, which mitigates the problem of choosing appropriate lag times associated with data on AIDS-deaths.[8] The latest UNAIDS/WHO report on the global AIDS epidemic (released on July 15th of 2004) also provides additional estimate ranges ("plausibility bounds") for the total number of infections. This analysis uses these improved data to run regression analyses along with the low values of HIV-prevalence rates. This allows one to discern whether the coefficients of the conflict variable remain significant and positive, given the uncertainties of the data.

This analysis goes on to model a non-linear relationship between HIV-prevalence and the duration of conflict involvement; both very short and very extensive conflict involvement seem to be correlated with low HIV-prevalence. The corresponding explanation for the latter fact draws on the isolating effects of enduring conflict involvement, which limits people's exposure to the outside world and thus to carriers of the HIV-virus.

[6] A list of the 197 countries included in the final sample is given in Annex F.
[7] See Schnell et al. (1999).
[8] For a discussion of data problems (concerning HIV-data and AIDS-death data) see page 98-101 of this paper.

A non-linear relationship is also assumed between the peace duration since last conflict involvement and HIV-prevalence rates. I hypothesize that in the short run, effects of processes related to peace and subsequent development, such as increasing levels of urbanization and income inequality result in increasing HIV-prevalence rates. However, in the long run development allows for more rapid control and effective responses and associates with lower HIV-prevalence.

Bivariate, multivariate and robust regression results from this analysis support the main hypothesis that countries' prior conflict involvement significantly correlates with higher HIV-prevalence in 2003. Effects of the corresponding conflict measures are much stronger compared to the effects of other control variables. This holds true even when low HIV-estimates are used. In addition, countries' own conflict involvement exerts a much stronger effect on HIV-prevalence compared to the much weaker and negative effect found for neighboring war involvement on HIV-prevalence in bordering countries. Preliminary support is also found for the assumed non-linear relationships between the peace time since last conflict involvement or the duration of conflict involvement and HIV-prevalence. Based on these and other results, I conclude that it is rather extensive conflict involvement (in terms of duration) than intense conflict involvement (in terms of battle deaths) which has a significant, non-linear effect on HIV-prevalence. Finally, it is rather the duration of conflict involvement instead of the type of conflicts involved (wars, intermediate or minor conflicts) which is important in regard to HIV.

Significant results and strong effects on HIV-prevalence are also revealed for the lagged HIV-variable, which indicates temporal dependence in HIV-data. As expected, the level of education correlates negatively and significantly with HIV-prevalence, which also holds true for the cultural variable measuring the proportion of Muslim or Jewish population. The positive effect of income inequality on HIV-prevalence also stays highly significant even when effects of mediating variables are controlled for. In addition, findings reveal a positive impact of high levels of urbanization on HIV-prevalence. However, pace of urbanization seems to be correlated with lower HIV-prevalence rates. Contrary to expectations, results indicate that the higher the proportion of young adults in the total population is, the lower HIV-prevalence levels are. This might be due to a problem of reverse causation. A similar argument explains the rather weak and often insignificant effect of health spending on HIV-prevalence. Finally, the variable measuring population density does not add to the explanatory power of the model and stays insignificant.

These results mostly support prior large-sample quantitative research, e.g. in regards to the insignificant effects found for population density [9] and health spending on HIV-prevalence.[10] Prior quantitative analyses exploring the socio-economic determinants of the HIV/AIDS epidemic also conclude that low levels of wealth as well as high levels of income inequality are strongly correlated with high adult HIV-prevalence rates or AIDS-cases.[11] The cultural variable has also been shown to be negatively correlated with HIV-prevalence.[12]

In regard to the effect of conflict on HIV-prevalence, prior results from large-sample quantitative research are controversial. In contrast to this study, Ghobarah et al. (2003) do not find an impact of a country's own civil war involvement in raising own HIV/AIDS rates. Instead, they find a significant and very strong effect of neighboring war involvement on HIV-prevalence in bordering countries. Findings from Davis et al. (2003), however, support the hypothesis of a positive effect of domestic and international conflicts on HIV-prevalence. They use Spatial Error Models, which estimate HIV-prevalence in one country dependent on HIV-prevalence in neighboring countries. Interestingly, they find that conflict involvement is positively correlated with HIV-prevalence, but reduces HIV-growth rates. The same holds true for development, which shows a significant and positive effect on HIV-prevalence in some of their models but is negatively correlated with HIV-growth rates. A study by Mahal (2001) explores the socio-economic determinants of HIV/AIDS. The analysis does not include a conflict variable, but rather an indicator measuring the size of the armed forces relative to the urban population. Contrary to Over (1998), Mahal (2001) does not find a significant association for this variable with HIV-prevalence. Davis and Kuritsky (2002) examine the relationship between violent conflict and routinely used health outcome indicators in Sub-Saharan African countries from 1980 to 1997. HIV-prevalence is not the dependent variable, but included as a control variable. Findings indicate that countries which have experienced conflict have worse health outcome indicators than countries without conflict experience. Smallman-Raynor et al. (1991) and Santos-Ferreira et al. (1990) also apply quantitative methods and link the geographical distribution of AIDS cases at the district or provincial level to the association between war and disease. However, both studies remain limited to a single case (Uganda and Angola).

[9] See also Davis et al. (2003).
[10] See Over (1998: 49).
[11] See Over (1998:46-47); DeHoltgrave et al. (2003); Mahal (2001).
[12] See Bonnel (2000), Mahal (2001).

In summary, findings from prior studies are hardly comparable and often inconsistent. In addition, most research lacks a sound theoretical basis explaining the relationship between the two macro-variables conflict and HIV-prevalence. Therefore, the first part of this paper describes the theoretical background of the analysis which draws on a social epidemiological approach (the Jaipur Paradigm). The Jaipur Paradigm explains how socio-economic forces determine differences in the progress of HIV/AIDS epidemics. I add a micro-foundation in order to explain how these macro-level factors interact with individual HIV risk behavior. Finally, the Jaipur Paradigm is enlarged by a conflict dimension and the specific mechanisms linking HIV/AIDS and conflict involvement are discussed.

The second part of this paper presents the quantitative analysis, which empirically tests whether previous conflict experience (between 1995 and 2002) has had an observable impact on national HIV-prevalence by the end of 2003. The following discusses the results from the bivariate analysis as well as outcomes of the multiple and robust regression analyses. I conclude with some policy implications and a summary on data limitations as well as limitations of this study.

2. Theoretical Background

2.1. An Introduction to the Jaipur Paradigm

Early attempts to explain the spread of STDs, including HIV/AIDS, relied heavily on behavioral and genetic explanations.[13] However, it is plausible that sexual behavior alone, or a "hyper sexualized African culture," cannot explain HIV-prevalence rates between 25%-38% of the adult population in some African countries, which are over fifty times that of the U.S., eighty times that of France and thousand times that of Cuba.[14] Contrary to behavioral explanations, data show that Americans and Europeans are in the lead when it comes to the number of sexual partners and frequency of sexual intercourse, which is associated with a higher risk for HIV-infection.[15] Both, rich and poor countries are characterized by high rates of unprotected, multi-partnered sexual activity. These similarities in sexual behavior between Western and African countries indicate that additional decisive factors explain high HIV-prevalence in African countries.

Empirical evidence comes from Buvé et al. (2001), who find that differences in risky sexual behavior are outweighed by differences in factors influencing HIV-transmission probability. These factors are the so called "biological co-factors" of transmission. The authors conclude, that differences in HIV-prevalence in four Sub-Saharan African cities in Benin, Cameroon, Kenya and Zambia cannot be explained by differences in sexual behavior. In particular, the higher incidence and prevalence of treatable bacterial STDs, which generally increase the risk for HIV-transmission due to lymphocyte activation and immunosuppression, [16] is identified as an important biological co-factor of HIV-transmission in Sub-Saharan Africa. According to Mabey et al. (1997), a high prevalence of STDs might explain why there is a heterosexual HIV/AIDS epidemic in Sub-Saharan Africa but not in Western Europe.[17]

---

[13] See Darrow et al. (1986).
[14] See Stillwaggon (2001).
[15] See Government of South Africa: South African Presidential Aids Advisory Panel Report (2001: 72).
[16] "There is scientific evidence that a person with an untreated sexually transmitted infection (STI), particularly involving ulcers or discharge, is on average, six to 10 times more likely to pass on or acquire HIV during sex. The presence of an STI means that there is more chance of broken skin or membranes allowing the virus to enter or leave the body. The very same cells that the virus is seeking to infect will be concentrated at the site of the STI because these cells are fighting the infection. According to current thinking, the risk for becoming HIV-infected from a single exposure is increased 10 to 300-fold in the presence of a genital ulcer caused by syphilis, chancroid or genital herpes" (UNAIDS, Questions & Answers II: basic facts about the AIDS epidemic and its impact 2004).
[17] See Buvé et al. (2001: 130); see also Mabey et al. (1997: 18); or Shell (2000: 14).

Approaches focusing only on the afore mentioned two factors (behavior and the prevalence of biological co-factors (e.g. STDs)) to determine HIV-risk have been referred to as "Biomedical Individualism" or "Behavioral Lifestyle Approaches." According to these theories, disease occurrence is based on individual lifestyle choices. Population disease patterns are the sums of these individual choices. It follows, that individuals can voluntarily alter their ways of living, which would theoretically reduce their risk for HIV-infection.[18]

However, explanations relying only on these two factors do not account for considerable cross-country variation in HIV-prevalence, e.g. within the same region. Even the addition of other co-factors, such as differences in the date of introduction of the virus (the stage of the epidemic) cannot fully account for differences in national prevalence levels. For instance, HIV/AIDS epidemics in South Africa and Thailand both began in the early 1990s. However, by 1999, HIV-prevalence in the general adult population of South Africa was 19.9%, whereas Thailand faced prevalence rates of only 2.2%.[19] In addition, "identifying risks at the individual level, even multiple risks, does not sufficiently explain interactions and pathways at that level, nor does it incorporate the social forces that influence risks to individuals" (Berkman et al. 2000: 6).

Already in 1949, Sidney Kark published a study, which put emphasis on the role of a third category of factors ("social forces") in explaining differences in the spread of sexually transmitted infections (STIs). He argues that structural conditions or societal-level factors are the driving force behind the epidemic spread of Syphilis in Africa. According to him, social conditions in regions, specifically population mobility, provide fertile ground for the spread of STIs.[20] It took decades for Public Health researchers to incorporate this central idea of socio-economic and cultural contexts into their attempt to systematically explain the spread of HIV/AIDS.

A group of 30 students and professors met for a training workshop at the "Indian Institute of Health Management and Research" in Jaipur in 1995. The purpose of the meeting was to explain differences in the profiles of local HIV/AIDS epidemics in order to understand the heterogeneity of prevalence rates across countries and regions.

[18] See Fee et al. (1993: 1481); Zierler et al. (1997: 406, 407).
[19] See Buvé et al. (2002: 2013).
[20] See Kark (1946).

The scientists developed the so called "Jaipur Paradigm," which serves as an econometric model to illustrate the interaction between HIV/AIDS and society. Its central premise is that with respect to HIV/AIDS, societies differ in their susceptibility and vulnerability to the disease. Susceptibility refers to the level of risk for HIV-infection in a particular social environment, defined as those aspects of a society which make it more or less likely that an epidemic will develop. In contrast, vulnerability refers to those aspects of a society which influence the likelihood that an epidemic will have a serious impact on social and economic organizations.[21] Before I will explain, how conflict involvement as a macro-level factor influences societies' susceptibility and vulnerability to the epidemic, the following section of this paper introduces and discusses the Paradigm.

According to the Jaipur Paradigm, societies' susceptibility and vulnerability to HIV/AIDS is determined by only two factors: their level of wealth and the degree of social cohesion. Though income inequality is mentioned as another important factor influencing the susceptibility to HIV-infection, it is not included as an additional variable to the model. It is argued that the level of income inequality is already observed in the level of social cohesion as income inequality is negatively correlated with social cohesion.[22]

Social cohesion refers to the degree of control within a society exerted by religious, moral or political leadership. It measures how cohesively societies operate as social, ethnic, linguistic and cultural units.[23] The concept refers to (1) the absence of latent social conflict (in form of income inequality, racial/ethnic tensions, disparity in political participation or other forms of polarization) and (2) to the presence of strong social bonds or high levels of "social capital."[24] Thus, cohesive societies are those that are richly endowed with stocks of social capital, which is associated with informal sociability, social trust, civic involvement in public affairs, high levels of volunteerism and the existence of a vivid community organizational life.[25]
The authors of the Jaipur Paradigm themselves note that social cohesion may be an expression of cultural homogeneity, it may be the product of good governance and a strong civil society, it may be related to a prescriptive religious culture, or it may be the result of a controlling authoritarian political system or military dictatorship.[26]

[21] See Barnett et al. (2000: 1099).
[22] See Barnett et al. (2000: 1100).
[23] See Shell (2000: 8).
[24] See Kawachi & Berkmann (2000: 175).
[25] See Putnam's measure of social capital as described by DeHoltgrave et al. (2003: 62).
[26] See Barnett et al. (2000: 1100).

The linkage between social cohesion / capital and HIV/AIDS might also not be immediately clear. However, Dr. Jonathan Mann (former General Director of the WHO) links at least one dimension of the social cohesion concept (a strong civil society, which protects human rights) to less vulnerability to HIV-infection.[27] Elsewhere it has been argued that separatism, fragmentation, and marginalization associate with decreased capacity for action, mainly because socially isolated individuals tend to live in areas with low social capital.[28] Health information and innovative behaviors (e.g. condom use) might also diffuse more rapidly in communities that are cohesive and in which members trust each other.[29] At the individual level, social capital may influence health behaviors by establishing social identities and societal norms, which are collectively negotiated and which promote healthy behaviors, trust and respect. Members of cohesive and trusting communities are more likely to experience high levels of perceived self-efficacy and empowerment, which increases the likelihood that they will engage in health-protective behaviors.[30] Social capital may also lead to the development of and accessibility to healthcare services.[31] In summary, social capital facilitates collective action and eventually leads to policies which are more likely to benefit and protect all citizens.[32]

Evidence comes from a study conducted by Holtgrave et al. (2003), which examines poverty, income inequality and social capital as predictors of state-level AIDS rates in the U.S. Social capital is found to be the strongest predictor of both STD and AIDS rates. Findings indicate a negative correlation between social capital and AIDS rates and the authors conclude that membership in social organizations protects against risky sexual behaviors.

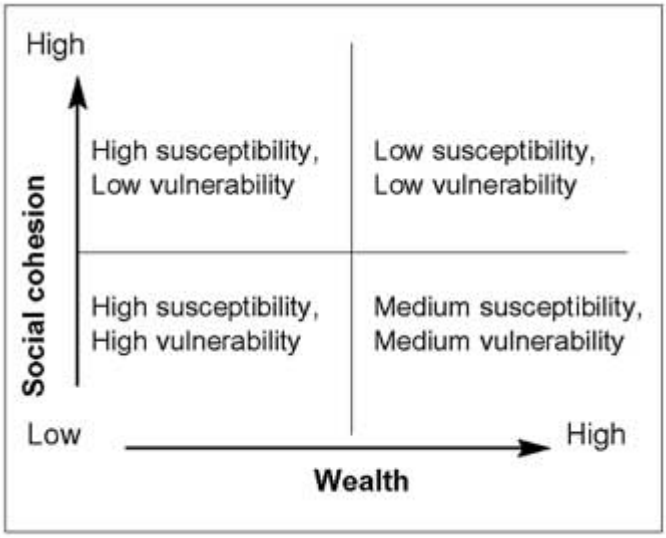
However, a study from South Africa which examines only the civic participation aspect of social capital, suggests that the type of organization to which one belongs makes a difference. Membership in social groups with high levels of social drinking increase HIV-risk, whereas membership in organizations such as churches, sport clubs and youth groups are associated with a decreased risk for HIV.[33] These findings indicate that community networks and relationships are not always positive in their effect on sexual health.

[27] See Mann (1999).
[28] See DeHoltgrave et al. (2003: 62).
[29] See Kawachi & Berkman (2000: 184).
[30] See Campbell et al. (2002: 51); Kawachi & Berkman (2000: 185).
[31] See Kawachi & Berkman (2000: 184-185); Deneke et al. (1991).
[32] See Poundstone et al. (2004: 26); DeHoltgrave et al. (2003: 62); Kawachi & Berkman (2000: 179-180, 185).
[33] See Campbell et al. (2002).

Nevertheless, there is support for the Jaipur Paradigm in the existing HIV/AIDS literature and the fact that levels of social cohesion, social capital, wealth or income inequality correlate with HIV/AIDS.[34]

The following graph illustrates the categorization of societies along the two axes of the Paradigm (level of wealth and level of social cohesion):

Graph1: The Two Axes of the Jaipur Paradigm



(Barnett et al. 2000: 1100).

The graph shows that poor countries with little social cohesion generally face high levels of susceptibility to HIV-infection and high levels of vulnerability to its impact. These countries often experience civil war or economic collapse, which has been the case in Rwanda, Cambodia and Haiti. The Paradigm predicts that it may take time for the epidemic to develop, but it will eventually reach very high levels, like in most parts of Sub-Saharan Africa. Countries that lack social cohesion and an equal distribution of wealth face difficulty in effectively responding to the epidemic. Thus, HIV-prevalence levels are also predicted to remain high.

In contrast, in poor countries with high levels of social cohesion (e.g. societies with strong religious cultures or good governance) HIV-rates are unlikely to rapidly increase. Country examples are Iran, Senegal, Cuba and India. These countries have all seen low epidemic growth rates and a plateau of low HIV-prevalence. Although they face high poverty-related susceptibility they are able to mobilize fast and effective responses due to high levels of social cohesion.

[34] See Over (1998), Mahal (2001), DeHoltgrave et al. (2003), Kawachi (2000).

Unified national planning, a public environment conducive to the social inclusion of people with HIV and those most at risk, strong and active civil society organizations and low levels of income inequality are all critical factors that assist in effective interventions.[35]

Relatively rich countries with low levels of social cohesion are predicted to experience an epidemic, which will take off very rapidly (due to low levels of social cohesion) and reach extremely high levels. Examples are Botswana, Namibia, Cote d´ Ivoire, the Dominican Republic and South Africa with high rates of labor-migration. However, as soon as these countries mobilize the financial means for an appropriate response, they will experience a rapid decline in HIV-prevalence. Thus, they face medium levels of susceptibility to HIV-infection and medium levels of vulnerability to the impact of AIDS.

In societies with both, high levels of income and social cohesion the epidemic only effects a "relatively" small number of people. In addition, these societies suffer less from HIV/AIDS as they are able to mobilize fast and effective responses due to high levels of wealth and social cohesion. Therefore, they face low levels of susceptibility and vulnerability to HIV/AIDS. Examples are wealthy Western nations, which are expected to experience slow epidemic growth, a low peak and slow decline.

The notion that a national level of HIV-prevalence depends on a societies' susceptibility as well as its vulnerability is crucial which is why this study relies on the Jaipur Paradigm as its theoretical basis. In summary, the Paradigm explains differences in the progress of HIV/AIDS epidemics through variations in countries' socio-economic susceptibility and vulnerability, defined as variations in their level of wealth and social cohesion. Thus, the Jaipur Paradigm applies a so called "Social Epidemiology Perspective," which considers social conditions as fundamental causes of disease.[36] Social epidemiology examines how people are exposed to risky or protective factors and under what social conditions individual risk factors are related to disease. According to this branch of epidemiology, the prospects for the health of every society are determined by social, political and economic forces that shape the environment.

[35] See Gorbach et al. (2002: 36).
[36] Definition: "Epidemiology is the study of the distribution and determinants of disease in human populations, with the implicit final goal of disease prevention…Thus, epidemiological studies aim to describe the characteristics of a disease, discover its cause, and eventually interrupt the causal chain to make possible prevention of the disease" (Flam et al. 1986:62).

It is expected that social trends or shifts (such as migration, political instability or changes in values, norms and social relations) constantly influence living conditions, which are themselves either positively or negatively related to the health of societies, regions or communities.[37]

As the conceptualization of the epidemic determines the appropriately deemed response, the Jaipur Paradigm clearly underpins the importance of addressing underlying socio-economic factors in HIV-prevention. Most of the responses to the pandemic have focused on its biomedical and behavioral determinants. However, according to the Paradigm, distributing condoms, promoting abstinence and condom use or administering STD treatment without understanding how that particular society works is an ill-conceived strategy. "[The Jaipur Paradigm] provides justification for interventions at the socio-economic level and adds weight to the view that governments have a substantial responsibility in this area …This means addressing issues of equality, human rights and the construction of `civil society´"(Barnett et al. 2000: 1101).

2.1.1. The Jaipur Paradigm: Critique and Application

The concept of social cohesion within the Jaipur Paradigm remains rather unclear, multidimensional and difficult to measure. Examples from countries with high levels of social cohesion are authoritarian governments, such as North Korea, Cuba or the countries of the former East Bloc. Although these countries show low levels of social cohesion on the civil society dimension (due to little experience with non-governmental organizations, historically repressed social activism and restricted public discussion of socially sensitive topics, such as sex and drug consumption),[38] social cohesion can nevertheless be enforced by fundamentalist, religious societies or militaristic regimes. In this manner, relatively low levels of social cohesion on one dimension (e.g. the civil society dimension) might be offset by high values on other dimensions (e.g. by high levels of cultural or social homogeneity). This is how countries with authoritarian governments can reach high levels of social cohesion, while social cohesion is also expected to be high in democratic countries practicing good governance.[39]

[37] See Poundstone et al. (2004: 22); Berkman et al. (2000: 6); Badura et al. (1991: 4); Macintyre et al. (2000); for more information on the historical framework of Social Epidemiology see Berkman et al. (2000).
[38] See Gorbach et al. (2002: 42).
[39] See Manning et al. (2002: 14, 15).

However, social cohesion is not just a nebulous but also a dynamic concept which changes over time as societies constantly undergo political, economic and social change. The authors mention that the applicability and usefulness of the concepts will have to be tested in detailed case studies, which will result in a refinement of the components that define social vulnerability and social susceptibility to HIV.[40] However, testing the Jaipur Paradigm in detailed case studies would require time-series data on the development of national HIV-prevalence rates, which are not available by now.

In addition, the grouping of societies into countries with four strict different epidemic profiles (as given in Graph1) is originally based on only six national case studies (U.K., Botswana, South Africa, Uganda, India and Ukraine). The conceptualization also ignores distinct social patterns of vulnerability and susceptibility of within countries and of regions. The factor of mobility which most likely plays a role in the spread of the HIV-virus remains completely disregarded.

Most importantly, a full theoretical explanation of differences between epidemic profiles amongst societies needs to be a holistic approach. This means that cultural, socio-economic, political as well as epidemiological factors need to be included, while taking into account that each person's probability of infection is determined by individual (sexual) behavior.[41] The Jaipur Paradigm, however, only implicitly assumes that individual HIV risk behavior is determined by socio-economic variables. More specifically, it is implicitly assumed that the level of wealth and social cohesion is somehow causally related to HIV-transmission. Increased vulnerability to HIV-infection, however, does not necessarily and directly translate into higher rates of HIV-infection. Interpreting a population-based association (between socio-economic variables and national HIV-prevalence) and inferring a causal effect on individual-level processes (the risk for HIV-transmission) is problematic. Indeed, it seems questionable that an exclusively macro-level intervention, such as improving social cohesion, would lower HIV-prevalence independently of individual-level behavior changes.[42] A better explanation for the interaction of societal vulnerability and susceptibility at the macro-level with biological and/or behavioral risk factors at the individual micro-level is needed.

[40] See Barnett et al. (2000: 1101).
[41] See Barnett et al. (2000: 1098).
[42] See Myer (2002).

With this goal in mind, the following section of this paper tries to add the missing "micro-foundation" to the Jaipur Paradigm by relying on Social Psychological Approaches explaining individual health risk behavior. Only in this manner, the later discussed mechanisms linking conflict and HIV-prevalence can be fully understood.

2.2. Explaining Individual Health Risk Behavior: The Health Belief Model [43]

The Health Belief Model (HBM) is a social psychological model, which serves to explain and predict individual health behavior. The model focuses on the attitudes and beliefs of individuals and puts emphasis on the role of perceptions (perceptions of vulnerability to infection, perceptions of the seriousness of disease threat, etc.). The model was originally introduced in the 1950s by psychologists working in the U.S. Public Health Service (Hochbaum, Rosenstock, Leventhal and Kegeles). Since then, the HBM has been adapted to explain a variety of long- and short-term health behaviors, including sexual risk behaviors and the transmission of HIV/AIDS.

The model assumes that two major factors influence the likelihood that an individual will adopt less risky behavior or a recommended preventive health action: First the individual must feel personally threatened by, or susceptible to the disease with serious or severe consequences. Second the individual must believe that the benefits of taking the preventive action outweigh the perceived barriers to (and/or costs of) preventive action. Thus, a person intends to act less risky and to adopt a recommended preventive health action if he or she feels that a negative health condition (i.e. HIV) is avoidable. In addition, a person intents to act less risky if he or she has a positive expectation that the negative health condition can be avoided by taking the specific recommended action (i.e. using condoms will be effective at preventing HIV), and if he or she feels that successful health action can be taken.

[43] See Bloor (1995: 88-94); U.S. National Institute of Health, National Cancer Institute: Theory at a glance: a guide for health promotion practice (2003); or http://www.tcw.utwente.nl/theorieenoverzicht/Theory%20Clusters/Health%20Communication/Health_Belief_Model.doc/, 29.10.2004; or http://www.etr.org/recapp/theories/hbm/Resources.htm, 29.10.2004; or http://www.comminit.com/hivaids/change_theories.html, 29.10.2004.

This focus on rational risk perception is not without its problems. It is debatable as to whether those, who believe they are at risk for HIV, will be more likely to protect themselves by avoiding risky behavior. For instance, evidence from a survey of Rwandan refugees indicates that better knowledge of AIDS and its modes of transmission do not necessarily translate into changes in behavior (e.g. condom use).[44] Similarly, although condom demand has increased, condom use patterns have remained substantively unchanged in South Africa. Condoms are used in only 14% of first sexual acts, despite the fact that there is a high level of awareness in many South African communities as to the sexual transmission of HIV.[45]

Pamela DeCarlo (1999) summarizes that an intellectual understanding of risk (knowledge) alone is often not a strong enough incentive to change behavior. Where risk is a motive for change it might not always be powerful enough to promote it. Again, this points to social, economic, structural or political factors, which also influence the susceptibility or vulnerability to HIV-infection. UNAIDS agrees and defines vulnerability in the context of AIDS as arising from "circumstances that are beyond the direct control of the people involved. Such circumstances include poverty, low social status, inequality, gender discrimination, discrimination, marginalization, and criminalization. Among other things, these circumstances also reduce or deny a person's access to HIV information, services, means of prevention and support" (UNAIDS, Questions & Answers II: Basic facts about the AIDS epidemic and its impact (2004)). Thus, the HBM includes an additional category of variables (demographic, socio-psychological and structural factors) that are "beyond the direct control of the people" while determining risk perception and individual health behavior. These factors (social environments) influence behavior by shaping norms, enforcing patterns of social control, providing environmental opportunities to engage in certain behavior and by producing stress. Risky behavior might then become an effective coping strategy for stressful experiences.[46] According to the HBM, environments also constrain individual choices; in this case, the transfer of intentions into actual behavior might be hindered because of external barriers.

Table 1 on the following page again presents the key variables of the HBM.

[44] See Benjamin (2001).
[45] See Government of South Africa: South African Presidential Aids Advisory Panel Report (2001: 75).
[46] See Berkman et al. (2000: 7, 9).

Table 1: Key Variables of the Health Belief Model [47]

KEY VARIABLES	DEFINITIONS
"Perceived threat"	Consists of two factors: (1) the "perceived susceptibility" (one's subjective perception of the risk for contracting a health condition) and (2) the "perceived severity" (feelings concerning the seriousness of contracting an illness or of leaving it untreated, including an evaluations of medical, clinical as well as social consequences)
"Perceived benefits"	One's belief in the efficacy of the advised action to reduce the risk or the seriousness of the impact
"Perceived costs"	The potential negative consequences that may result from taking particular health actions, including physical, psychological and financial demands
"Perceived self-efficacy"	This addition to the HBM is based on the Self Efficacy Theory of Badura.[48] Perceived self-efficacy refers to one's confidence in the ability to successfully perform an action
"Other variables"	Diverse demographic, socio-psychological and structural variables that affect an individual's perceptions and thus indirectly influence health-related behavior
"External motivators, cues to action"	Events, either bodily or environmental (e.g. social support systems, media publicity) that motivate people to take action
"External barriers"	Factors that hinder the transfer of intentions into actual behavior

2.3. Explaining HIV Risk Behavior: The AIDS Risk Reduction Model [49]

The AIDS Risk Reduction Model (ARRM), introduced in 1990, provides a framework for explaining and predicting the behavior change efforts of individuals with respects to the sexual transmission of HIV. The three-stage ARRM incorporates several variables from other behavior change theories, including the afore mentioned Health Belief Model and the Efficacy Theory.

The first stage of the ARRM consists of recognizing and labeling one's behavior as high risk. The hypothesized factors that influence this stage are the individual's knowledge of sexual activities associated with HIV-transmission, the belief that one is personally susceptible to contracting HIV and the belief that having AIDS is undesirable (social norms). In addition, external barriers, such as aversive emotional states (e.g. high levels of distress), may facilitate or hinder the labeling of one's behaviors as risky.

[47] See U.S. National Institute of Health, National Cancer Institute: Theory at a glance: a guide for health promotion practice (2003).
[48] See http://www.ticino.com/usr/opadlina/new/ph/epi-ges.htm, 29.10.2004.
[49] See http://www.comminit.com/hivaids/change_theories.html, 29.10.2004.

The second stage involves the commitment to reduce high-risk sexual behavior. This phase is influenced by the perceived costs and benefits of behavior change, the question of whether the changes will affect personal enjoyment especially with regard to sex, the perceived response efficacy (that is the question of whether the changes will successfully reduce the risk for HIV-infection), perceived self-efficacy as well as social factors (such as group norms and social support).

The third stage is the "taking action stage," which is broken down into three phases: 1) information seeking; 2) obtaining remedies and 3) enacting solutions. Depending on the individual, phases may occur concurrently or may be skipped. The hypothesized influences on the "taking action" stage are prior experiences with problems and solutions, informal or formal help (e.g. from social networks), the individual's level of self-esteem, the resource requirements of acquiring help, the individual's ability to communicate verbally with the sexual partner and the sexual partner's beliefs and behaviors.

The consideration of the beliefs and behaviors of sexual partners is an important stage. This serves to capture the social character of sexual risk behavior, which self-evidently involves more than one individual. Therefore, the ARRM puts emphasis on the way in which individual risk behavior is constrained by power relationships. In general, and in the case of the HBM, internal and external factors may motivate individual movement across the three stages of the ARRM. Those factors can be socio-psychological factors, demographic, structural and political factors.

Both, the Health Belief Model and the AIDS Risk Reduction Model include an important component of reasoned action. However, both approaches leave room for external (macro-) factors to influence actions and the cost/benefit calculations of individuals. According to the models, risky individual behavior cannot be explained without taking into account external motivators, external barriers and other variables affecting the processes of perception building, commitment making and the transfer of intentions into actual behavior. These external variables, among them the macro-variables of the Jaipur Paradigm, become decisive in linking macro-environments to individual health risk behavior. Later on, it will be discussed in more depth as to how the level of wealth, income inequality and other control variables included in this analysis affect individual's HIV risk behavior and, eventually, national HIV-prevalence levels.

The main focus of this analysis, however, is on the nexus between HIV/AIDS and another, external macro-variable constraining individual risk behavior, namely conflict experience. In the following, it is argued that conflict experience affects other macro-variables, which have been identified as "distal determinants" of the HIV/AIDS epidemic.[50] Conflict involvement changes individual's micro-environments, which eventually affects individual risk behavior.

For this reason, the following discusses decisive mechanisms which explain how "conflict involvement" affects individual HIV risk behavior and, therefore, interacts with societies' susceptibility and vulnerability to HIV-infection. The overall purpose is to answer why the populations of war-affected people are particularly at risk for HIV-infection. The first part of this paper then concludes with the presentation of the final theoretical model.

3. Mechanisms Linking Conflict and HIV-Prevalence

3.1. The Effects of Conflict on Infrastructure

As mentioned earlier, perceptions of susceptibility to infection are dependent on individuals' knowledge of HIV, sexual risk activities and measures to reduce the risk for transmission. Thus, the act of recognizing and labeling one's behavior as high risk heavily depends on the individual's level of awareness. However, conflict involvement at the macro-level directly affects the level of awareness of individuals and, therefore, individual risk behavior through the disruption of public education and prevention campaigns.[51] Thus, conflict involvement acts as an external barrier to perception building.

In addition, conflicts overwhelm health care services. People, no matter how well-informed, are often left with few options to protect themselves at a time when they are especially vulnerable. Empirical evidence comes from Liberia[52] and Sierra Leone[53]. In the latter case, 62% of rural health units stopped functioning during civil war.

[50] See Barnett et al. (2000).
[51] See Renaud (2001: 11) or Bellamy (2001), who states that levels of awareness of HIV/AIDS - and how to prevent it – are generally very low especially in conflict-affected countries.
[52] See U.N. Press Release AFR/640 OP/865 (9.6.2003); Schowengerdt et al. (1998); Connolly et al. (2002).
[53] See UNICEF: HIV/AIDS and children affected by armed conflict (2002).

Elsewhere (e.g. in Uganda and Mozambique) health services have been specifically targeted by one side during civil war so as to weaken the opposition.[54] Hoeffler (1999) states that during war, both opponents and governments target physical infrastructure as a part of their strategy. Among the main targets are hospitals.[55] Additionally, it is found that on average only 30% of the population in 12 war-affected Sub-Saharan African countries had access to clean water during conflict periods and only 20% had access to sanitation facilities. People in urban areas experienced even worse conditions. During the conflict in Djibouti, access to safe water and access to sanitation facilities was limited to 42% and 24%, respectively of the rural, and 86% and 66% of the urban population.[56] Similarly, decades of conflict severely weakened health care infrastructure in Afghanistan.[57] This limits individual's access to health care and treatment, which results in increases in the spread of infectious diseases, including HIV/AIDS.

For example, HIV-positive mothers often do not receive antiretroviral drugs before and after birth to reduce their viral load, which has been identified as a very predictive criterion for transmission of HIV to the infant.[58] Although mother-to-child transmission of the virus most often occurs at delivery, infants of HIV-positive mothers face the additional risk for infection through breast milk feeding.[59] Especially during conflict, breast milk substitutes or funds to purchase baby feeding formula are often not available. In addition, access to clean water, which makes it safer and more practicable to bottle-feed, cannot be assured.[60] These examples illustrate how limited access to health care due to damaged or under-funded infrastructure affects individual risk behavior (e.g. breast-feeding) which can lead to increases in the risk for HIV-transmission.

Reduced access to prevention and treatment during conflict also makes it unlikely that conflict experienced individuals have "prior experiences with solutions" (e.g. formula feeding or condom use), which, according to the ARRM, motivates people to take action and adopt less risky behavior.

[54] See Van der Heijden (1997); Ghobarah et al. (2003: 192-193).
[55] See Hoeffler (1999: 5); see also Carballo & Frajzngier (2001: 8, 14).
[56] See Hoeffler (1999: 15, 16).
[57] See Hankins et al. (2002: 2248).
[58] See Government of South Africa: South African Presidential Aids Advisory Panel Report (2001: 32, 33); Dabis et al. (2000).
[59] See Dabis et al. (2000); Johnson (2003). A randomized clinical trial conducted between 1992 and 1998 in four antenatal clinics in Nairobi randomly assigned mothers to formula- or breast-feeding. Results reveal that HIV-free survival was significantly higher in formula fed infants (see Mbori-Ngacha et al. 2001). UNAIDS also warns that breast-feeding increases the risk for mother-to-child transmission by 10-15% (see UNAIDS: Questions & Answers II: basic facts about the AIDS epidemic and its impact: 2004). Others report even much higher increases in risk for HIV-transmission for breast-fed infants (see Fowler et al. 1999: 781).
[60] See Government of South Africa: South African Presidential Aids Advisory Panel Report (2001: 72).

Another important fact to consider is that during war, government spending is more likely to be directed towards the purchase of weapons and ammunition rather than toward the provision of appropriate health care to soldiers and the general population.[61] Collier et al. (2004) find that during war, military spending rises by around 1.8 % of GDP and that during the first decade of the post-conflict period, governments tend to maintain it at much higher levels than pre-conflict.[62] Others agree with the conclusion that the displacement of social spending toward the military tends to continue after conflicts have ended.[63]

In cases where salaries of medical and health care personnel are not paid and basic supplies are not available this may lead to the departure of qualified staff members during and after conflict. In Mozambique, scarcity of health care personnel between 1976 and 1992 was also related to conflict experience as qualified staff had fled to urban centers or been kidnapped during internal armed conflict.[64]

In summary, HIV-prevention and treatment systems are severely weakened or completely disrupted in countries affected by conflict. Along with the disruption of schooling during conflict and reduced government spending on education and HIV/AIDS awareness programs, the overall result is a higher prevalence of STDs and HIV during and following conflict. This is exacerbated by changes in private spending priorities and the overall lack of resources for acquiring help, which also becomes a major constraint to actual behavior change.

Additionally, armed conflict damages agricultural and economic infrastructures. Property, such as livestock, land and cattle herds are sometimes even systematically destroyed. Village food stores and seed stocks are attacked and plundered and anti-personnel mines prevent farmers from returning to their fields. This adversely affects agricultural production in conflict times where import of food is limited or even non-existent. The consequences are food shortages, malnutrition and starvation. As a result, communities develop deficiencies of micronutrients, which cause a deterioration of the immune system. This facilitates HIV-infection and hampers recovery.[65]

[61] See Goyer (2001: 16); Guha-Sapir et al. (2002: 19).
[62] See Collier et al. (2004: 132).
[63] See Van der Heijden (1997); Ghobarah et al. (2003: 192-193).
[64] See Cossa et al. (1994: 117).
[65] See Guha-Sapir et al. (2002: 16).

Others argue that especially with regard to mother-to-child transmission of HIV/AIDS vitamin deficiency becomes an important risk factor.[66] It can thus be expected that war-related malnutrition associates with an increase in infectious diseases, including those which are sexually transmitted.

The breakdown of government infrastructure poses another serious problem. For instance, the breakdown of law and order may correlate with an increasing incidence of rape committed by soldiers and civilians during wartime.[67] Countries in conflict often do not have the mechanisms to report abuse or arrest offenders, which undermines the safeguards of legislation against violence or discrimination. As prosecution becomes unlikely, clan-based militia, e.g. the so called "moryan raiders" in Somalia, are more able to rape women with impunity.[68] Many peace agreements include amnesty for crimes committed by members of military forces, which further hinders the prosecution of war-related rape in post conflict situations.[69]

The breakdown of law and order and associated increasing access to injectable drugs has also been cited as an important factor contributing to the spread of HIV in conflict settings.[70] Today, increasing drug trade in combination with politically unstable (post-conflict) situations partly accounts for rapidly increasing levels of HIV/AIDS infections in Eastern Europe and parts of Asia.[71] An increase in drug injection becomes especially likely when conflict disrupts supply routes of drugs that are usually ingested, sniffed or smoked. This results in the introduction of drugs that are more likely to be injected. For instance, studies reveal, that the war in Afghanistan was associated with increased needle sharing among injecting drug users in neighboring Pakistan, which might be a result of the disruption of regular heroin trafficking from Afghanistan.[72] Along with the shortage of sterile injecting equipment during wartime the overall result is increasing HIV-prevalence among the group of injecting drug addicts.

[66] See Government of South Africa: South African Presidential Aids Advisory Panel Report (2001: 72).
[67] See Smith (2002: 4).
[68] See Human Rights Watch: Rape in Somalia (1995).
[69] See Salama et al. (1999: 1569) in the case of Sierra Leone.
[70] See Hankins et al. (2001).
[71] See U.N.: A more secure world: our shared responsibility (2004: 15-16).
[72] See Hankins et al. (2001: 2247-2249).

3.2. Uprooted Populations and HIV/AIDS

Military conflict brings economic and social dislocation, including the forced movement of refugees across borders and internally displaced people, who remain within their country of origin. UNAIDS refers to data provided by the Inter Agency Standing Committee Task Force on HIV/AIDS in Emergency Settings (IASC) and states that various conflicts resulted in over 42 million refugees and internally displaced people worldwide in 2003.[73] While many flee the general violence of war, most seek refuge because they are specifically targeted by armed forces.

These people are often demoralized, dispirited and desperate, which makes for an ideal breeding ground for the HIV/AIDS epidemic. Uprooted populations experience the loss of a sense of personal control and coping, which normally counteracts the impact of stress and moderates the relationship between stress and health.[74] Perceptions of self-efficacy are low as war-affected populations feel helpless and desperate, which can undermine benefits of preventive education.[75] In addition, language problems compound feelings of isolation and social exclusion. These feelings are amplified by the fact that dense and reciprocal social relationships, including their sexual component, are destroyed or disrupted. Families become separated, members are killed and social support systems and norms break down. All of these factors would otherwise have been potential "external motivators" for low-risk behavior. In situations of psychosocial pressures, loneliness and lack of emotional support, beliefs about sexual health and behavior may be forced to change. Carballo & Frajzngier (2001) note that "…attitudes concerning sex and sexual behaviour may become more flexible as women and men search for emotional support and care" (Carballo & Frajzngier 2001: 9).

In the absence of formative social structures and the constraining and guiding influence of family and community, especially adolescents are more likely to engage in risky, self-destructive behavior. Feelings of uncertainty that result from conflict, the need to feel emotional closeness or simple boredom can all lead young people to ignore the threat of HIV.[76]

[73] See UNAIDS/WHO: Report on the global AIDS epidemic (2004: 175).
[74] See House et al. (1991: 155).
[75] See DeCarlo (1999).
[76] Smith (2002: 9); Renaud (2001: 9); Carballo & Frajzngier (2001: 9, 10).

Therefore it is not surprising that children in refugee camps have been found to begin to experiment with sex and drug use earlier and tend to be ignorant of the risks of HIV-infection.[77] Renaud (2001) cites an UNHCR official, who reports from adolescent refugees in Tanzania, saying that "they are drunk by noon and have either raped or had sex by 2pm."[78]

In summary, high HIV risk behavior might become a manifestation of social marginalization and psychosocial stress of war-affected populations in general.[79] At the individual level, conflict related levels of distress affect individual risk behavior. This is also in accordance with the ARRM, whereby high levels of distress have been described as external factors that hinder the labeling of one's behaviors as risky. Consequently, practical and psychological obstacles to adopt less risky behavior with regard to HIV increase enormously during wartime.

In addition, constant psycho-social stress itself has been shown to be associated with immunological abnormalities. Recent research points out that the degree of stress is determined by the desirability, magnitude, unpredictability, time-clustering and uncontrollability of the negative event.[80] Where the negative event constitutes conflict experience, violence or uprooting, all these factors are certainly pronounced.

Amnesty International (AI) reports from Rwanda where women and girls suffered or witnessed acts of indescribable brutality, including the murder of family members and loved ones during the genocide and war. Men and children were anguished by the physical and psychological assaults on their female family members. This study, conducted by AVEGA (an association for genocide widows) in 1999, reveals that over 80.9% of people surveyed were found to be profoundly traumatized.[81] Not surprisingly, over 90% of internally displaced women, interviewed by Amowitz et al. (2002) in Sierra Leone, reported being extremely worried about possible sexual assault committed by combatants on themselves or their family members.[82] Anxiety and depression decrease the lymphocyte count, which eventually lowers natural T-cell activity and has been found to increase the risk for HIV-transmission.[83]

[77] See U.N.: Fact Sheet: global crisis - global action: AIDS as a security issue (2001: 2); UNAIDS Point of View: refugees and AIDS (1997: 2); UNAIDS Technical Update: refugees and AIDS (1997: 4).
[78] See Renaud (2001: 8, 9).
[79] See Carballo & Nerukar (2001: 558); Smith (2002); Strathdee et al. (2002); Hankins et al. (2002).
[80] See House et al. (1991: 150); see also Ursin (1991).
[81] See AI: Rwanda: marked for death: rape survivors living with HIV/AIDS in Rwanda (2004: 7).
[82] See Amowitz et al. (2002: 518).
[83] See Government of South Africa: South African Presidential Aids Advisory Panel Report (2001: 28).

In line with this biological explanation, Mabey et al. (1997) emphasize extreme psychological stress to explain a high prevalence of STDs in Rwandan male refugees in Tanzanian camps.[84]

Generally, displaced populations searching for help in camps tend to find poor health services, inadequate sanitation and limited access to medicine. Health conditions in camps are critical, especially during the early phase of rapid influx of many persons.[85] This partly explains the death rates among refugees arriving in Ethiopia, Kenya, Nepal, Malawi, and Zimbabwe between 1990 and 1993, which were approximately five to twelve times the baseline death rate in their countries of origin. Death rates among internally displaced people in northern Iraq, Somalia and the Sudan ranged from 12 to 25 times the baseline crude death rate for the non-displaced population. Among the most commonly reported causes of death were infectious diseases.[86]

In addition, preventing the spread of HIV has not been a priority of early interventions in the past, because it is not perceived as an immediate threat to life. Often, HIV is regarded as a development issue rather than a concern for agencies responding to emergencies.[87] In particular, internally displaced people often fall between the cracks of international AIDS programs. They do not have access to the refugee aid of the international community due to their dubious legal position[88] while at the same time they are excluded from their own governments' HIV/AIDS programs. Similarly, refugees have been systematically excluded from their host countries´ HIV/AIDS National Strategic Plans.[89] Lack of basic and culturally appropriate information or education materials in local languages is mentioned as another constraint on effective HIV-treatment and prevention.[90] This leaves refugees and internally displaced people without full access to prevention options, including quality STD diagnosis and treatment as well as affordable access to condoms.[91]

[84] See Mabey (1997: 20).
[85] See Carballo & Frajzngier (2001: 5).
[86] See Toole et al. (1993: 600, 603); see also Toole (2000: 204).
[87] See Spiegel (2004: 331); Smith (2002: 1-2); McGinn et al. (2001); UNAIDS Technical Update: refugees and AIDS (1997: 3); http://hivinsite.ucsf.edu/InSite?page=kb-08-01-08#S2X, 10.3.2004; UNAIDS/WHO: Report on the global AIDS epidemic (2004: 181).
[88] See Benjamin (2001), Toole et al. (1993: 601), Toole (2000: 206-208).
[89] See Spiegel et al. (2004: 23), Spiegel (2004: 327).
[90] See Spiegel et al. (2004: 22), Smith (2002: 10).
[91] See McGinn (2000).

Another serious danger for the spread of the HIV-virus lies in transfusions of HIV-infected blood, which in refugee camps and emergency situations are often needed in large numbers. Renaud (2001) informs that only 57% of the blood is tested for HIV in countries ranked medium or low on the UNDP Human Development Index (HDI).[92] In conflict situations, where regular transfusion systems have broken down, it becomes even more difficult to ensure the safety of blood.[93] In addition, the military has been considered an excellent blood donor population in the past. Given the fact that HIV-infection within the group of military personnel is very high and that blood donor transfusion services started testing the donor blood very late this is very concerning.[94]

However, people in conflict settings face other more immediate threats to life, which override concerns about becoming infected with HIV.[95] Clearly, perception of risk is not a strong motivator when AIDS is just one risk of many and when the effects of HIV are not immediately felt. This tends to bias perceptions of the threat posed by HIV which, according to the HBM, affects individual risk behavior.

More immediate threats to refugee life also include extreme poverty, hunger and desperation. This can force women and girls into prostitution, as a means of obtaining food or shelter, safe transit through the war zone or papers and privileges for themselves and their families. In particular, women traveling alone may have little choice but to sell sex for survival. Often they establish (short-term) partnerships in transit or at their destinations in order to gain access to protection.[96] In addition, refugee camps are also known to attract prostitutes from surrounding communities to cater to male refugees.[97] For example, Hutu refugee camps in Rwanda's neighboring countries were notorious for prostitution and sexual promiscuity.[98] The increasing adoption of prostitution as a short-term strategy or coping mechanism to secure basic human necessities for survival in conflict times forces changes in gender roles which results in even greater powerlessness and increased vulnerability of women to HIV.[99]

[92] See Renaud (2001: 7).
[93] See UNAIDS Point of View: refugees and AIDS (1997: 2); UNAIDS Technical Update: refugees and AIDS (1997: 4).
[94] See Shell (2000: 13).
[95] See Shell (2000: 12).
[96] See Haour-Knipe (2003: 2).
[97] See Fourie et al. (2001: 7); Smith (2002: 7).
[98] See McKinley (1998).
[99] See Benjamin (2001).

In return and along with male boredom, depression and substance abuse in refugee camps, the risk for sexual violence increases. Rape often occurs within and around the camps and becomes an additional factor linking refugee existence to HIV-infection.[100]

In the case of Tanzania Benjamin (2001) reports that Rwandan refugee women and girls were raped when they visited the latrine or fetched water. UNAIDS finds that HIV-prevalence among Rwandan people, who said they had spent the conflict years in refugee camps was 8.5%; two-fifths of the victims surveyed were teenagers. Most of the infected had fled from rural areas where pre-conflict HIV-prevalence was much lower (about 1.3%), which suggests a six-fold increase in HIV-infection among refugees in the camps. Again, UNAIDS attributes high HIV-prevalence to the high incidence of rape. "Among women who had been raped 17% were HIV-positive, compared with 11% of those who had not" (UNAIDS/WHO: AIDS in the military 1998: 12).

McGinn (2000) refers to a population-based survey of 339 Burundian refugee women in another Tanzanian District, where more than one in four women reported being raped since the start of the conflict three years earlier. Two-thirds of the rapes occurred in or near the camp. It is reported that survivors identified the perpetrators as other refugees in 59% of cases, local Burundian residents in 24% of cases, and local Tanzanians, soldiers and police in the remaining incidents.[101] The author concludes that the evidence suggests that refugee women experience rape and other forms of sexual violence at least as often as, and probably more often than, women in settled populations.[102]

Similarly, a recently released study by Lynn Amowitz et al. (2002) refers to rape within the context of the civil war in Sierra Leone. In order to assess the prevalence and impact of war-related sexual violence and other human rights abuses among internally displaced persons, a cross-sectional randomized survey was conducted over a 4-week period in 2001.

[100] See Bellamy (2001); UNHCR/ Save the Children-UK: note for implementing and operational partners on sexual violence and exploitation: the experience of refugee children in Liberia, Guinea and Sierra Leone (2002); Benjamin (2001).
[101] See McGinn (2000: 178).
[102] See McGinn (2000: 179). The author also reminds that most research focuses on refugees living in stable camp settings and that the situation may differ in an emergency phase, for those not living in camps or internally displaced people. Having in mind the mentioned risk factors contributing to high HIV-prevalence among refugee population, excluding those not living in camps and internally displaced people from current research probably results in an underestimation and underreporting of the prevalence of rape and HIV in displaced populations.

Structured interviews and questionnaires of internally displaced Sierra Leone women, living in three internally displaced people camps and one town, were conducted and a total of 991 women provided information on 9166 household members. The outcome reveals that 9% of 991 respondents and 8% of 5001 female household members reported war-related sexual assaults. Indeed, the prevalence of war-related sexual assault, committed within a period of only three years, was equivalent to the lifetime prevalence of non-war-related sexual assault. Based on their findings the authors estimate that approximately 50.000 to 64.000 Sierra Leonean internally displaced women may have suffered war-related sexual assaults.[103]

In summary, displaced populations face an increased likelihood of prostitution and sexual violence, which broadens and accelerates the spread of HIV.[104] "Conditions of refugee life are particular conducive to sexual violence, both in the early stages of a complex emergency, when rape is used by armies as a weapon of war, and later in the stable phase, when violence perpetrated by intimate partners or acquaintances may become more prevalent" (McGinn 2000: 178-179). Female refugees from Sierra Leone and Rwanda agree and believe that the long conflict engendered a culture of violence that permeates their societies. They recognize that war erodes traditional practices that otherwise promote respect and gender balance in societies.[105] Thus, the decline of social and traditional values and norms clearly affects risk behavior of civilians as well as soldiers.

Given the afore mentioned mechanisms (e.g. high levels of psycho-social stress, rape, prostitution or poor health conditions in camps) it is reasonable to expect displaced populations to be at higher risk for HIV. There is growing evidence from war zones, e.g. in Myanmar, Rwanda, Bosnia and Sierra Leone that war and forced migration are linked to the spread of HIV/AIDS.[106] Particularly for Eastern and Southern Africa various studies have shown a much higher HIV-prevalence in people with a record of international mobility or migration.

[103] See Amowitz et al. (2002: 520); see also Physicians for Human Rights (2002).
[104] See U.N.: Fact Sheet: global crisis - global action: AIDS as a security issue (2001: 2); violent sexual intercourse increases the risk for HIV-transmission, which is supported e.g. by a study conducted in rural Uganda. Findings indicate that compared to HIV-negative women, young HIV-positive women were approximately 8 times more likely to have been subjected to unwanted sex through rape (see Quigley et al. 2000: 422).
[105] See Benjamin (2001).
[106] See ICG: Myanmar: the HIV/AIDS crisis (2002: 3); UNAIDS: Fact Sheet 2: HIV/AIDS and conflict (2002: 2).

Studies carried out in health care centers in West Africa confirm this correlation between international mobility and vulnerability to HIV. In Senegal 70% of HIV-infected patients in the main health care facility were found to have a migration background in Central and West Africa.[107] Another author concludes that risk for HIV-infection is six times higher in refugee camps than outside.[108]

In addition, the location of the refugees rather than refugee status itself was found to be important in the examination of the geographical spread of HIV. For instance, the proximity to a war zone or major city is said to account for variation in HIV-prevalence rates among refugee populations in Angola[109] and Swaziland.[110]

However, the focus of this analysis is not the geographic distribution of HIV/AIDS cases. I also do not examine the direction of transmission, which depends on the relative prevalence levels in the areas of origin and destination and is, therefore, not inevitably from refugees to local residents. Instead, I focus on overall increases in national HIV-prevalence and argue that war-related mobility and mixing of populations with different HIV-rates and awareness levels increases HIV-prevalence overall.[111]

In particular, the mixing together of high-prevalence urban populations and low-prevalence rural populations in refugee camps additionally contributes to the spread of the epidemic.[112] This has been the case in Tanzanian camps hosting Rwandan refugees from urban areas (where HIV-prevalence levels were about 35%) and from rural areas (where HIV-prevalence was about 5%). Sexual contact between high- and low-prevalence populations quickly spreads the virus among both the refugees and the surrounding population (which in this case also had a low HIV-prevalence of 7%).[113] Exposure to the military further promotes transmission, which becomes the third decisive mechanism linking HIV/AIDS and conflict involvement.

[107] See UNECA: Lessons Africa has learnt in 15 years of responding to HIV/AIDS (2000).
[108] See Gardiner (2001: 2).
[109] See Santos-Ferreira et al. (1990).
[110] See VanRensburg et al. (1995).
[111] See also McGinn et al. (2001); or http://hivinsite.ucsf.edu/InSite?page=kb-08-01-08#S2X, 10.3.2004.
[112] See UNAIDS/WHO: Report on the global AIDS epidemic (2004: 175).
[113] See Smith (2002: 20); see also Carballo & Frajzngier (2001: 18).

3.3. The Trojan Horse Hypothesis: AIDS and the Military

3.3.1. HIV-Prevalence in the Military

As already partly discussed, mechanisms linking conflict and HIV/AIDS are numerous. Most critical, however, are extremely high infection rates of STDs and HIV among the armed forces. Even during peacetime, soldiers have STI rates two to five times greater than those of civilian populations. Those rates can soar to up to 50 times higher during armed conflict.[114]

Where data is made public, this leads to figures such as 20% to 40% HIV-positive soldiers in Sub-Saharan Africa countries, with rates of 50% to 60% in a few cases where the virus has been present for over 10 years and average adult prevalence is already very high.[115] In South Africa, for example, 60% to 70% of military personnel are infected; at a military police base in northern KwaZulu-Natal, a 90% infection rate was found.[116] Others mention infection rates among the military in Malawi of 75% and in Zimbabwe of 80% - approximately three times as high as average rates among the adult population.[117] Even in peaceful Botswana, one in three members of the military have been tested HIV-positive;[118] estimates of HIV-prevalence among the military in Angola and the Democratic Republic of Congo range between 40% and 60%.[119] VanBeelen (2003) notes that 50-60% of all beds in the Kenyan Armed Forces Memorial Hospital are occupied by military personnel with HIV/AIDS-related infections and that at least 6-10 soldiers die each week as a result of AIDS.[120] Data from a rural blood bank in Mozambique revealed that 39% of military blood donors were HIV-positive, compared to 15% of non-military donors. Almost 75% of these HIV-positive soldiers have been tested positive for syphilis as well.[121] The proportion of reported HIV-cases in the military is also comparatively high in El Salvador[122] as well as Cambodia and Thailand.[123] Although HIV-prevalence levels in the military forces are still low, HIV-prevalence in military recruits also alarmingly increases in Vietnam.[124]

[114] See UNAIDS: AIDS and the military (1998: 3).
[115] See U.N.: Fact Sheet: global crisis - global action: AIDS as a security issue (2001: 2).
[116] See Kirk (2000).
[117] See ICAD: HIV/AIDS and displaced people (2001:2).
[118] See UNAIDS/WHO: Report on the global AIDS epidemic (2004: 176).
[119] See VanBeelen (2003: 6).
[120] See VanBeelen (2003: 7).
[121] See VanBeelen (2003: 6).
[122] See Wollants et al. (1995: 129).
[123] See Carballo & Frajzngier (2001: 19).
[124] See Gorbach et al. (2002: 41).

The government of Ethiopia found that only 5.5% of tested military personnel were HIV-positive, which is less than the national prevalence of 10.6%. However, Renaud (2002) points to the hidden factor that most soldiers had been screened prior to entry into the army and those with HIV had been rejected. In addition, the length of stay in the military averaged at only 10 months, meaning that the HIV-positive conscripts had become infected very fast.[125]

These data indicate wide country specific ranges, but overall significantly higher HIV-prevalence rates within the military. Accurate figures on infection rates of various rebel groups and militias do not even exist but are also expected to be high. In summary, Over (1998) finds that for the average developing country, reducing the military size from 30% to 12% of the urban population would reduce sero-prevalence among low-risk urban adults by about one percentage point.[126]

High susceptibility of military forces to HIV-infection has been attributed to their work environment,[127] their relatively low levels of maturity combined with high levels of testosterone,[128] their high levels of sexual activity[129] and the military's professional ethos, which tends to excuse or even encourages risk-taking on and off the battle field, also in regard to sexual behavior.[130] In addition, aggressiveness may lead soldiers to engage in coercive sex or risky behavior, such as sex with multiple partners or purchased sex. "During times of conflict this is abetted by high levels of alcohol and drug consumption by soldiers" (Fourie et al. 2001: 7). Trauma caused by conflict experience, brutalization by day-to-day experience of violence, frustration, apathy, loneliness and dispiritedness have also been identified to lead to an increase in sexual violence, particularly against women.[131] This is supported by the U.N. and the finding that young men who staff regular armed forces and other armed groups are main perpetrators of sexual abuse and exploitation in settings of war and conflict.[132] Thus, sexual violence and rape committed by soldiers becomes an additional important factor linking HIV/AIDS and conflict.

[125] See Renaud (2001: 15).
[126] See Over (1998: 48).
[127] E.g. deployment to unsettled areas with exposure to socially disrupted settings where STIs may abound, a high possibility of infection through wounds and contaminated blood, etc. (see Goyer 2001: 13).
[128] See Fourie et al. (2001: 7).
[129] See VanBeelen (2003: 6).
[130] See UNAIDS: AIDS and the military (1998).
[131] See ICAD: HIV/AIDS and displaced people (2001); Hankins et al. (2002: 2246); Smith (2002: 11).
[132] See U.N. Fact Sheet: global crisis - global action: AIDS as a security issue (2001: 2).

3.3.2. Rape and Sexual Violence Committed by Soldiers

Indeed, Amowitz et al. (2002) find that war-related rape of internally displaced women in Sierra Leone was widespread and mostly committed by combatants.[133] Case study evidence from Sierra Leone also comes from the French agency Médecins Sans Frontières, which reports that of 1,862 women abducted during the Revolutionary United Front's offensive on Freetown in January 1999, 55% had been repeatedly raped.[134]

Donovan (2002) even states, that nearly all the women and adolescent girls who survived the Rwandan genocide are now living with the traumatizing memory of a brutal sexual attack that they had suffered or witnessed firsthand.[135]

Swiss et al. (1998) report on women's experience of violence, including rape and sexual coercion, committed by a soldier or fighter during the Liberian civil war from 1989 through 1994. Almost half of the women interviewed reported experiencing at least one act of physical or sexual violence by a soldier or fighter. Attempted acts of physical or sexual violence by soldiers or fighters were reported in 15% of the cases. An additional 42% reported witnessing a soldier or fighter kill or rape someone else. However, only women from the capital city of Monrovia, which was under the protection of peacekeeping forces after August 1990, participated in this study. Figures also exclude sexual violence committed by civilians. Thus, these figures probably even underestimate the prevalence of rape and sexual coercion committed by soldiers and fighters as well as the overall prevalence of rape during the war.[136]

A comparison of sexual violence levels before and after "conflict involvement" is available in the case of East Timor. There, sexual violence levels committed by non-family members during and after the violent crisis that followed East Timor's 1999 vote for independence were analyzed. Results reveal that sexual violence levels during the crisis where 57.1% higher.[137] Often, however, sexual violence and forced marriage continue to be perpetrated by members of the military forces, security forces and unpaid militias after conflicts end.

[133] See Amowitz et al. (2002: 520).
[134] See Astill (2000).
[135] See Donovan (2002: 17).
[136] See Swiss et al. (1998: 628).
[137] See Ward et al. (2004: 27).

Although baseline studies on sexual violence levels from before and after the conflict or war experience do not exist, anecdotal evidence from Rwanda also suggests that domestic and sexual violence have increased significantly since the genocide in 1994.[138] There, economic and social vulnerability of women and girls continue to leave them exposed to sexual violence. Generalized trauma exacerbates sexual and domestic violence.[139]

In many cases rape has even been systematically used by regular and state armies as a weapon against civilians during wartime.[140] Systematic mass rape has been undertaken during war-fare for example in Uganda, Bosnia, Mozambique, Liberia, Sierra Leone, Sri Lanka, Rwanda, Burma, Somalia and the Democratic Republic of Congo.[141] Among the objectives for systematic rape are spreading terror and fear, inflicting psychological damage, disabling the enemy by destroying the bonds of family and society or even ethnic cleansing. In addition, systematic rape of women is often aimed at male combatants who are out of reach while fighting. "Through a single act of rape, the assailant can humiliate and demoralize, and thereby "attack", the women's male relatives who are unable to protect her … Enforced pregnancy is used as a form of ethnic cleansing, because the women is forced to bear a child that has been "ethnically cleansed" by the blood of the rapist" (Shanks et al. 2000: 1153).

After the genocide in Rwanda approximately 2000-5000 children were born to women who were raped, in most by more than one man or repeatedly over the course of several weeks or months.[142] Systematic sexual molestation, mutilation and rape of women and girls during the genocide were integral to the plan to annihilate the Tutsi population. "Among the weapons of choice calculated to destroy while inflicting maximum pain and suffering was HIV. Eyewitnesses recounted later that marauders carrying the virus described their intentions to their victims: they were going to rape and infect them as an ultimate punishment that would guarantee long-suffering and tormented deaths" (Donovan 2002: 17).

[138] See AI: Rwanda: marked for death: rape survivors living with HIV/AIDS in Rwanda (2004: 2, 3).
[139] See AI: Rwanda: marked for death: rape survivors living with HIV/AIDS in Rwanda (2004: 9).
[140] See Elliott (1999); Swiss (1999); or McGinn (2000) for an overview on case studies regarding sexual violence in conflict settings.
[141] See ICAD: HIV/AIDS and displaced people (2001: 2); Salama et al. (1999); Human Rights Watch: Rape in Somalia (1995); Shanks et al. (2000: 1153); Swiss et al. (1993); Swiss et al. (1998); Smith (2002: 8); Wax (2003).
[142] See Donovan (2002: 17).

Based on the information given, sexual violence levels are expected to increase during and even after conflict involvement. As HIV-prevalence within the armed forces is generally high, it is not surprising that there exists country evidence linking HIV/AIDS and war-related rape. In Kingali, the capital of Rwanda, HIV-prevalence among pregnant women from rural areas rose up to 24% in 1995, which is said to be a result of rape and displacement during the 1994 genocide.[143] The U.N. estimate that between 250,000 and 500,000 rapes were committed, gang rape was common, and there were many incidences of women being abducted and held for long periods as sex slaves. Of the women who survived these attacks 70% are estimated to have been infected with HIV.[144] Burma is mentioned as another example, where soldiers' practice of raping ethnic minority women in conflict areas has attributed to the spread of HIV.[145] In addition, HIV-infection is also strongly associated with the movements of military personnel and rape in Liberia[146] as well as in Nigeria and Uganda.[147] Using regression analysis, Smallman-Raynor et al. (1991) confirm the association of war and HIV/AIDS for the later case. They show that the distribution and spread of HIV-infection in Uganda during the 1980s and 1990s is linked to the pattern of military recruitment some ten years earlier. The researchers conclude that the association between war and disease accounts for much of the geographic distribution of AIDS cases in the country.

At the individual level, sexual violence or rape completely denies the victim control over sexual behavior. Verbal communication with the sexual partner or negotiating safer sex becomes impossible. Where sex is enforced by violence the risk behavior of one individual is characterized by constraint, not free choice, and clearly determined by the partner's sexual beliefs and behaviors. For this reason and from the perspective of the victim, war-related sexual violence clearly acts as an "external constraint" on self-determined, individual HIV risk behavior. More generally, war-related poverty, powerlessness, lack of respect for human rights and marginalization all drive discrimination and exploitation, creating risk situations in which people have very little individual choice.[148]

[143] See http://www.unaids.org/security/Issues/conflict.html, 16.6.2003.
[144] See AI: Rwanda: marked for death: rape survivors living with HIV/AIDS in Rwanda (2004: 3, 6).
[145] See Burkhalter (2002: 3).
[146] UNAIDS/WHO: AIDS: Epidemic Update (2002: 35).
[147] See Fleshman (2001); Van der Heijden (1997).
[148] See Haour-Knipe et al. (2003: 2).

3.3.3. Prostitution

A related problem is the growth of local sex industries in response to demand from military bases. As military service often includes lengthy periods spent away from home, personnel are looking for ways to relieving loneliness, stress and the build-up of sexual tensions. It is also mentioned that the military culture uses the purchasing of sex as part of bonding among soldiers.[149] In addition, military personnel bring a degree of steady income into impoverished rural areas where men are off fighting or have fled. This can translate into an increase in the number of women who turn to prostitution to survive during and after conflict. Therefore, prostitution is often discussed as another leading factor linking HIV/AIDS and conflict.[150]

The U.N. warn, that even in peacetime, the likelihood of unsafe commercial sex appears to rise in the presence of military bases and units.[151] Soldiers on deployment regularly have sexual contacts with prostitutes as well as the local population. For example, UNAIDS estimates that approximately 45% of Dutch navy and marines personnel on peacekeeping duty in Cambodia had sexual contact with sex workers or other members of the local population during a five-month tour.[152] Similar type of risk behavior of soldiers in Sierra Leone, with an estimated HIV-prevalence of 60-70%, might partly explain increasing HIV-prevalence in prostitutes. "Studies carried out in Sierra Leone in 1995 …revealed that female sex workers in Freetown had HIV-infection rates of 26.7 %. By 1997, with much of the country embroiled in fighting, rates had soared to 70.6 %" (UNAIDS/WHO: AIDS Epidemic Update 2002: 35).
Country evidence also comes from Thailand, where soldiers have been found to be more likely than other men to visit prostitutes, and less likely to always use a condom.[153] The potential for the spread of the HIV-virus from the prostitutes over soldiers into the population of non-prostitutes is considerable, because most of the men visiting prostitutes also reported having non-prostitute partners. Of those, who reported having both types of partners, most had unprotected intercourse with prostitutes and with non-prostitutes.[154] In this manner, female sex workers and their male (military) clients act as core or bridging groups and play an important role in HIV/STI transmission to the general population.[155]

[149] See VanBeelen (2003:6).
[150] See Hankins et al. (2002); Goyer (2001:16); Astill (2000); Machel (1996: 34, 35); UNAIDS/WHO: AIDS Epidemic Update (2002: 35); UNAIDS: AIDS and the military (1998).
[151] See U.N.: Fact Sheet: global crisis - global action: AIDS as a security issue (2001: 2).
[152] See UNAIDS: AIDS and the military (1998).
[153] See VanLandingham et al. (1993).
[154] See VanLandingham et al. (1993: 311).
[155] See Lowndes et al. (2002) for evidence from Benin.

Machel (1996) finds that even children become victims of prostitution following the arrival of peacekeeping forces. "In Mozambique, after the signing of the peace treaty in 1992, soldiers of the United Nations Operation in Mozambique (UNOMOZ) recruited girls aged 12 to 18 years into prostitution …In 6 out of 12 country studies on sexual exploitation of children in situations of armed conflict…the arrival of peacekeeping troops has been associated with a rapid rise in child prostitution" (Machel 1996: 34, 35).

Elliott (1996) interviews child prostitutes in war-torn Liberia, who had their first sexual experience between the ages of 10-13 years. Many of the girls had been raped during their time as child prostitutes or have been involved into risky sexual practices. She refers to secondary sources, which indicate that foreign troops take advantage of the extreme poverty of women and girls in the local setting and exploit the situation for sex. Child prostitutes are preferred to adult prostitutes as they are less demanding with little or no responsibility. Young girls often receive food items and material rewards, whereas adult prostitutes demand money rather than goods in return for sexual relations with military personnel. In addition, the usage of young girls for sex is also seen as a means of avoiding HIV-infection.[156]

Female child soldiers, who are forced to be "wives" or sex slaves to one or several boys or men in their unit in Sierra Leone, Liberia or the Democratic Republic of Congo are particularly vulnerable to STIs, HIV, and unwanted pregnancy through rape and sexual exploitation.[157]

In summary, the described mechanisms illustrate how the HIV-virus uses combatants as `Trojan Horses´ to spread itself among the civilian population surrounding military bases and to enter low-prevalence areas. In accordance with Dr. Robert Shell, I, therefore, call the hypothesis, that HIV is spread by troops, the `Trojan Horse Hypothesis.´[158] Soldier's mobility during warfare results in a mix of people with high- and low-risk behavior that otherwise might not mix. Thus, armies moving between high- and low-prevalence countries become primary vectors for transmitting the disease and serve as a `bridge population.´[159]

[156] See Elliott (1996).
[157] See Renaud (2001: 6).
[158] See Shell (2000: 12); or Shell (2002).
[159] This is not a new phenomenon. The military has played a significant role in the spread of other infectious diseases in the past, such as the Influenza Epidemic of 1918/1919 or the spread of Smallpox and Syphilis in the fifteenth and sixteenth century (see Shell 2000: 9).

3.3.4. Demobilization of Soldiers and Resettlement of Uprooted Populations

As has been argued earlier, the presence of armies recruited from high-prevalence countries has an immense impact on domestic HIV-prevalence rates in areas of conflict, as they contribute to the spread of the virus through the afore mentioned mechanisms (e.g. rape and prostitution). In this manner, even peacekeeping forces can become main agents for spreading HIV. Soldiers recruited from high-prevalence countries make up 11% of the total U.N. peacekeeping force.[160]

However, the other way around, soldiers recruited from low-prevalence countries (e.g. Angola) but posted to areas where HIV-prevalence is high (e.g. Congo) might also spread the disease in their communities when returning home. Often, they engage in unprotected sex with prostitutes while being on duty but either have wives or steady girlfriends back home. In this manner, HIV-positive soldiers transmit the virus to monogamous women who would otherwise not be at risk. Again, peacekeepers are no exception. In the case of Nigeria, Alban et al. (2000) refer to a statement of General A. Adefolalu (Head of the Army Medical Corps) given at the Third All African Congress of Armed Forces and Police Medical Services in Pretoria in 1999. Mr. Adefolalu reports that Nigerian peacekeepers, starting their mission in the late 1990, showed a HIV-prevalence rate of 7% after one year of employment. Within three more years of employment the prevalence rate rose to 15%. Of those Nigerian peacekeepers returning from Sierra Leone and Liberia, 11% were HIV-positive as compared to 5% HIV-prevalence in the overall adult population.[161]

Robert Shell (2000) also refers to the role of returning veterans and exiles in unwittingly spreading the virus throughout South Africa from 1976 through 1994. The author informs that, after apartheid ended, thousands of former members of liberation groups were incorporated into the South African National Defense Forces without testing them for HIV. Many of them had been living in frontline camps in countries with higher HIV-prevalence. Along with members of the old homeland armies, they were distributed to military camps all over the country.[162] Cuban veterans returning from Angola are given as another example of demobilized soldiers, who contributed to the spread of the national HIV/AIDS epidemic.[163]

---

[160] For example, HIV-infection in Sierra Leone was 2.99% in 1999. Troops sent to Sierra Leone as part of peacekeeping missions have been drawn from countries such as Kenya, where 1 in 4 adults are HIV-positive (See Goyer 2001: 16); see also ICG: HIV/AIDS as a security issue (2001: 22-23).
[161] See Alban et al. (2000).
[162] See Shell (2000: 7, 12).
[163] See Shell (2002: 7, 8).

These examples illustrate that concerns are justified and that the demobilization of soldiers might indeed accelerate the spread of the virus; particularly, as most of the demobilized soldiers return to rural areas with generally low prevalence levels. Considering the high HIV-prevalence in the armed forces as well as the determination that a soldier's sero-status is a major consideration in being demobilized or mustered out, there is urgent need to recognize this serious public health threat. Still, this is not done for example in Ethiopia. After the peace treaty with Eritrea in December 2000, 55.000 troops were sent home without any HIV-testing or counseling. They attended HIV-education for only two hours.[164]

Similar logic applies to the resettlement of refugees and internally displaced people. Mozambique serves as an example for increased risk for STDs during repatriation and resettlement in post-war situations. There, seventeen years of civil war caused massive population movements. Between 1992 and 1995 an estimated 1.7 million refugees returned from Malawi, Zimbabwe, Tanzania, Zambia and Swaziland. Along with demobilized soldiers and internally displaced people they were resettled. Four early studies, conducted between 1987 and 1992, reveal that HIV-prevalence in displaced populations was much higher (3.2%-4.6%) than the 1.2% prevalence in the general population. In addition, displaced people showed lower awareness of condoms. War in Mozambique severely destroyed health infrastructure, which additionally might have contribute to increasing HIV-prevalence during the early post-war period.[165] Similarly, approximately four million internally displaced people will eventually return or have already returned to their place of origin in Angola. In addition, over 450,000 refugees returning from high HIV-prevalence host countries, such as Zambia and Namibia, will be repatriated.[166] The same concerns relate to the return of large numbers of internally displaced people and ex-combatants to their homes in Sierra Leone. Therefore, the story does not end with armed forces in action or refugees in camps but continues within the demobilization and resettlement phase.

In summary, war-affected populations (whether internally displaced people, refugees, civilians or soldiers) are disproportionately at risk for STDs, including HIV-infection, as conflict creates perfect conditions for interacting epidemics. "It may be said that conflict increases the spread of STDs and HIV at least through displacement and military presence, which are the inevitable result of war" (McGinn 2000: 178).

[164] See Renaud (2001: 6).
[165] See DeHulsters et al. (2003: 77); see also UNAIDS/WHO: Epidemiological Fact Sheet - 2004 Update: Mozambique (2004).
[166] See Williams et al. (2002); Spiegel (2004: 22).

The breakdown of health systems as well as their under-funding, the disruption of schooling, reduced government spending on education and HIV-awareness and the movement of armies during wartime can be added to mass displacement, refugee existence, violence, rape, powerlessness of women, poverty and increased prostitution to explain high HIV-prevalence rates in countries with conflict experience. Conflict increases the spread of STIs and facilitates HIV-infection through various routes, including sexual contact, rape, injecting drug use, contaminated blood transfusions and occupational injuries.[167]

Therefore, "conflict involvement" is added as a key explanatory variable to the theoretical model of this analysis. The final model, which explains the linkage between conflict and HIV-prevalence is then based on the earlier described Jaipur Paradigm (Social Epidemiology) enlarged by a conflict dimension as well as a micro-foundation (provided by the HBM and ARRM).

4. The Final Theoretical Model

The following basic thoughts constitute the theoretical model of the applied analysis. The model distinguishes between macro- and micro-effects and puts the earlier mentioned mechanisms into a more systematic approach which explains the linkage between conflict involvement and HIV-prevalence.

First, the determinants of the epidemic are considered to be of four types, namely

(1) macro-environmental factors (e.g. wealth, income distribution, culture or conflict),
(2) micro-environmental factors (e.g. access to health care or level of social control),
(3) behavioral factors (e.g. sexual mixing patterns and condom use) and
(4) biomedical co-factors (such as the stage of the epidemic, male circumcision,[168] existing virus subtypes and the presence of other STDs).[169]

[167] See Hankins et al. (2002: 2245).
[168] "Research has identified plausible biological explanations for a connection between HIV infection and lack of circumcision. The tissue of the internal foreskin absorbs HIV up to nine times more efficiently than female cervical tissue, mainly because it contains Langerhans and other HIV "target cells" in much greater quantities than the cervix or other genital tissue (including other parts of the penis). In addition, the internal foreskin has a mucosal surface, as opposed to the more hardened skinlike surface of the external foreskin. This mucosal surface is particularly susceptible to tears and abrasions, and, consequently, infection by STDs and HIV.

Second, factors at different levels of analysis interact in such a way that changes at the macro-level affect micro-environments of individuals which results in behavioral changes at the micro-level.

Third, I argue that conflict experience fits into the Jaipur Paradigm as it results in the breakdown of all social cohesion, which is considered to be an advantage in fighting the disease. Income levels are also affected by conflict experience, which is the second decisive macro-variable explaining susceptibility to HIV-infection and vulnerability to its impact.

Finally, conflict experience impacts societies' susceptibility and vulnerability to HIV/AIDS independently of its effect through wealth, social cohesion or income distribution. For example and as already discussed, conflict experience directly affects the mobility of populations as it is associated with moving soldiers and often results in mass displacement of refugees and internally displaced people. Also at the macro-level, conflict leads to the breakdown, damage and under-funding of health care and education systems. Rapid changes in social values and the breakdown of institutions have been mentioned as well as changes in women's status e.g. as they become refugees.

Again, these war-related changes at the macro-level, along with war-related increases in disparity in income and overall decreasing levels of wealth, can explain the following changes in the micro-environment: a decrease in social control, increases in instability of sexual partnerships, an increase in commercial sex work, a decrease in access to health care and deteriorating health care systems, lack of preventive health education, increasing levels of violence and powerlessness of women as well as an increase in drug trade and individual depression. In particular, individuals from migrating or refugee backgrounds, or other persons in the path of war, are unlikely to see risk for HIV as a significant additional treat to their lives.

All this contributes to changes in individual risk behavior, such as increased drug and alcohol use, rape, changing sexual mixing patterns, an increased number of different sexual partners or less condom use.

These epidemiological, geographic, and biological findings provide very strong – though not conclusive – evidence that male circumcision significantly lowers the risk for HIV-infection"(UNAIDS, Questions & Answers II: Basic facts about the AIDS epidemic and its impact 2004). A quantitative analysis on male circumcision and risk for HIV- infection in Sub-Saharan Africa was conducted by Auvert et al. (2001) and a meta-analysis of 27 studies also concludes that male circumcision is associated with a significantly reduced risk for HIV-infection among men in Sub-Saharan Africa (Weiss et al. 2000); see also Buvé et al. (2001); Lowndes et al. (2002), Quigley (2000).

[169] See Mannings et al. (2002: 13).

The deduced final outcome, which is observable at the macro-level is a decrease in the overall health status of individuals and an increase in national STDs and HIV-infection rates.

A very clear example, illustrating the afore mentioned, comes from Rwanda, where the massacres in 1994 resulted in a massive number of war related male deaths, leaving behind thousands of widows. Women, whose families have been scattered or killed, try to piece their lives together. Their strong desire to replace children lost in the massacres and war leads them to share a single male sexual partner to have children. Polygamy is being practiced, which increases the risk for HIV-infection.[170] Thus, conflict involvement and war related male deaths (at the macro-level) results in changes in social and sexual norms (micro-environment), which affects individual risk behavior and sexual mixing patterns (at the micro-level).

Finally, conflict involvement directly affects biological co-factors of transmission, particularly when it comes to malnutrition, the presence of other STDs and the existence or emergence of virus subtypes. For instance, as military forces are moving, various subtypes of the HIV-virus are getting recombined. This lead to the occurrence of the so called "Congo-HIV-Mélange" in the Democratic Republic of Congo, where fighting fractions were backed by military forces from Uganda, Rwanda, Zambia, Namibia, Zimbabwe and Angola. Congolese rebels have also been moving freely in Tanzania, Burundi, Sudan and the Central African Republic.[171] In general, biological co-factors are always related to the socio-economic, structural and cultural environment. For example, the presence of other STDs is determined by access to health care, whereas male circumcision is largely governed by traditional customs and religious beliefs.[172]

According to this, differences in HIV-prevalence can be explained by a complex interplay of (sexual) risk behavior and biological co-factors that affect the probability of HIV-transmission. However, (sexual) risk behavior itself is determined by cultural and socio-economic context variables.

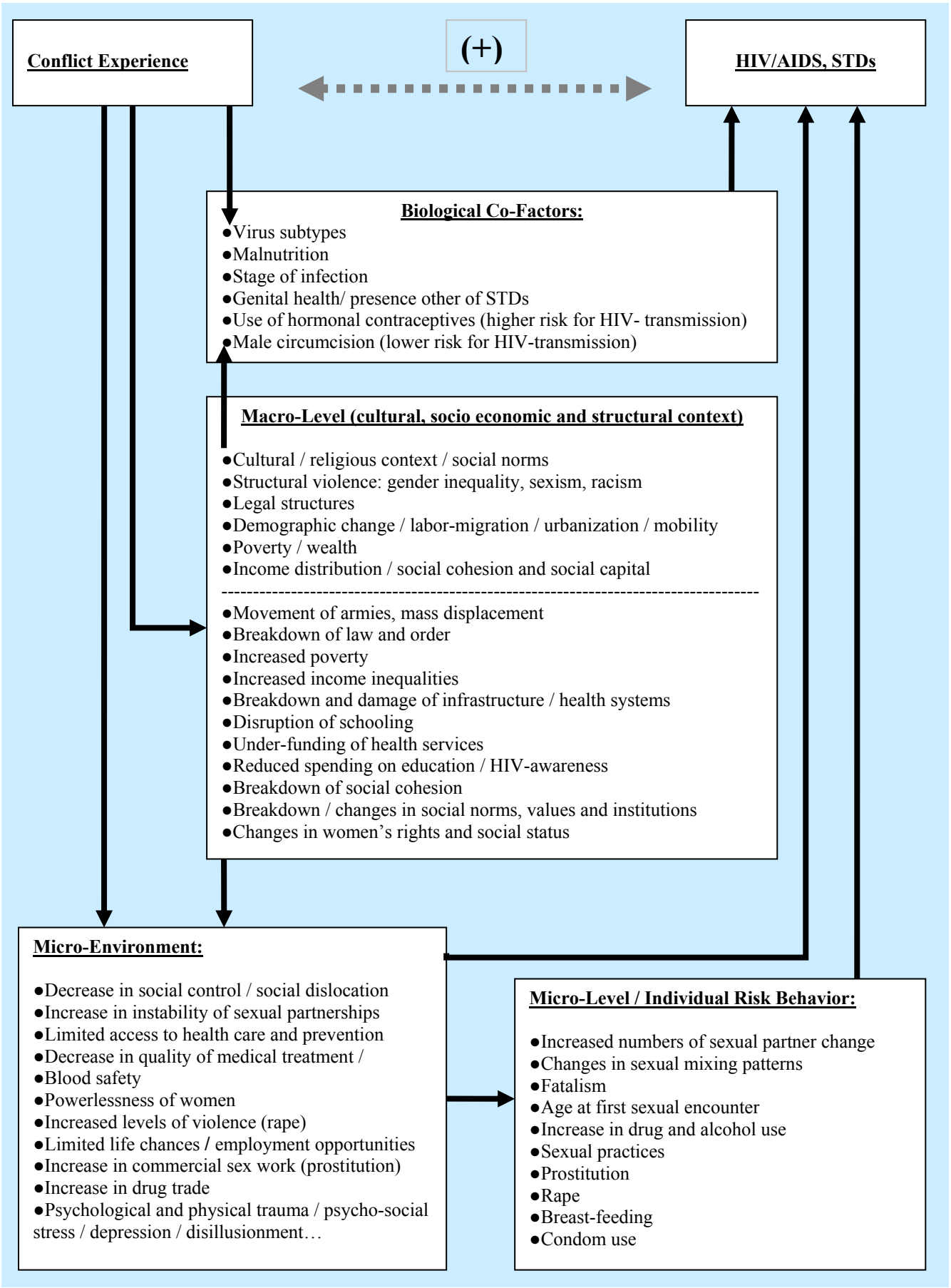
[170] See McKinley (1998); Smith (2002: 8).
[171] See http://www.smh.com.au/news/specials/intl/aids/aids16.html, 1.6.2001.
[172] See Over (1998: 39).

In summary, the theoretical model of this analysis combines a "biological individualism perspective" with the "social epidemiology perspective" of the Jaipur Paradigm, enlarged by a conflict dimension. In accordance with Krieger (2001) and others, I call this an "ecosocial approach."[173] The micro-foundation or underlying causal model expects that variations in macro-level conditions (e.g. social, cultural, political and economic factors) lead to differences in lifestyle, individual health risk behavior and exposure to risk factors. This results in differences in overall health outcomes and HIV-prevalence. Therefore, variations in macro-level conditions help to predict the level of HIV-infection in a given country.

The following graph again summarizes the final theoretical model, which explains the linkage between conflict involvement and HIV-prevalence levels.

[173] See Krieger (2001); Zierler et al. (1997: 409); Poundstone et al. (2004).

Graph 2: The Final Theoretical Model Explaining the Linkage Between Conflict Involvement and HIV-prevalence



5. Main Hypothesis and Choice of Quantitative Analysis

According to the afore mentioned, this study empirically tests the hypothesis that prior conflict involvement (between 1995 and 2002) leads to a difference in countries' HIV-prevalence rates. Thus, the Null-Hypothesis states that conflict involvement between 1995 and 2002 does not result in a significant difference in 2003 HIV-prevalence.

This main hypothesis is based on the following temporal logic: end of 2003 HIV-prevalence rates, which are the latest and sole HIV-data available for such a large-sample study, can only be brought into connection with previous conflict experience. In order to account for reverse causation all other independent variables are also measured prior to the dependent variable (HIV-prevalence).

I specifically refer to conflicts experienced between 1995 and the end of 2002 in order to account for the disease progress from HIV-infection over illness (AIDS) to death. Estimates for the incubation period [the time interval between initial exposure to infection and appearance of the first symptom or sign of disease (AIDS)] range from 5-15 years, with the shorter period found in the developing world. The time between the onset of AIDS and death is about three years.[174] For this reason, restricting the lag time to an overall period of eight years from infection to death is necessary in order to include those infected in 1995 (or later), who remain alive and are HIV-positive in 2003. A lag time of more than eight years would fail to include those people who became HIV-infected during the study period, but are already dead in 2003 and, therefore, not captured by 2003 HIV-data.

Two types of statistical analyses provide the basis for the test. First, a statistical test of significance is performed on the difference of means in HIV-prevalence of 197 countries. Second, a multivariate linear regression analysis tests the impact of conflict involvement on HIV-prevalence while controlling for factors known and expected to contribute to HIV-prevalence.

.

[174] See http://www.worldbank.org/aids-econ/toolkit/intro.htm, http://www.ifst.org/aids.htm, 25.6.2004; or http://www.rho.org/html/hiv_aids_overview.htm, 25.6.2004; or Johnson (2003).

6. Measurement [175]

6.1. Measurement of the Dependent Variable (HIV-Prevalence)

The HIV-data are taken from the latest UNAIDS/WHO report on the global AIDS epidemic, released on July 15th of 2004. On the advice given by the UNAIDS Reference Group on HIV/AIDS Estimates, Modeling and Projections, new software has been developed to model the course of HIV/AIDS epidemics and their impact. Along with changes in methodology and assumptions, this has led to overall improved quality and accuracy in the end of 2003 estimates. In addition, estimates are presented with ranges (low and high estimates), called 'plausibility bounds.'[176] This is supposed to reflect the extent of uncertainty in estimates, which depends mainly on the type of epidemic and the quality, coverage and consistency of a country's surveillance system. This range provides an additional test to the robustness of the coefficients of the conflict variable.

The three measures of the dependent variable are **`HIV-prevalence rates´ (y1), `low estimates of HIV-prevalence rates´ (y2) and `high estimates of HIV-prevalence rates´ (y3).** These data refer to adult HIV-prevalence rates as the percentage of the whole adult population (15-49 years old) estimated to be HIV-positive at the end of 2003. UNAIDS/WHO estimates include all people infected with HIV who were alive at the end of 2003 – regardless of whether they have developed symptoms of AIDS.[177]

The age range (15-49 years old) captures those who are in their most sexually active years. UNAIDS/WHO state that - while the risk for HIV-infection continues beyond 50 years - the vast majority of people who will become infected are likely to have done so by this age. Still, it is necessary to point out that the data used exclude HIV-positive adults over the age of 49, who might have become infected during the study period. However, most of the developing countries with particularly high HIV-rates (where this could have resulted in the exclusion of a large number of people) have life expectancies lower than 49 years.

[175] Complete information on measurement and data sources is given in Annex B.
[176] For more information on the estimation and application of the plausibility bounds see Grassly et al. (2004) or Walker & Grassly & Garnett & Stanecki & Ghys (2004).
[177] With respect to the interpretation of prevalence rates it is important to note that current rates do not take into account those who have already died from AIDS. Thus, the cumulative figure is much higher. Therefore, the likelihood that a person, who is not infected with HIV/AIDS, will acquire the virus is much higher than the current prevalence rates.

Thus, I conclude that this age cut should not be too problematic as the total number of adults over the age of 49, who are HIV-positive and still alive, is expected to be very low. Additionally, HIV-positive children aged 0-14 are also not covered by the data. However, most of them are likely to have acquired the virus via mother-to-child-transmission. As the mothers' infection might be associated with conflict related factors, such as rape or prostitution, and as mothers are most likely over the age of 15 and, therefore, already captured by the data, the exclusion of children is a means of avoiding double counting.

Table 2 provides a brief overview of the most important summary statistics of the dependent variable.[178] It is immediately clear that HIV-prevalence rates are distributed in a heavily positive skewed manner. HIV-prevalence is rather low in the majority of countries while some countries face extremely high HIV-rates at the end of 2003. The lowest rates can be found in Asian and Western European countries, whereas Sub-Saharan African countries show an average value (between 6.9-8.3%) markedly above other regional averages. The highest worldwide HIV-prevalence (y1) of 38.8% is found in the adult population of Swaziland.

Table 2: Summary Statistics, HIV-Prevalence Data

	MEAN	MEDIAN	MIN.	MAX.	SD	SKEWNESS	N
HIV-prevalence (y1)	2.54	0.4	0.1	38.8	5.56	4.14	197
Low Estimates of HIV-Prevalence (y2)	1.90	0.2	0.01	37.2	5.02	4.72	197
High Estimates of HIV-Prevalence (y3)	3.65	0.7	0.2	40.4	6.44	3.18	197

[178] Summary statistics of all variables included in the final analyses are given in Annex A.

Generally, the U.N. distinguish between three different epidemic states.[179] In countries with low-level epidemics (first state), HIV-infection may have existed for many years, but has never spread to significant levels in any sub-population. Infection is largely confined to individuals with high-risk behavior. However, networks of risk are rather diffuse (with low levels of partner exchange or sharing of drug injecting equipment). Often, the virus has been introduced only very recently. In this first epidemic state, HIV-prevalence has not consistently exceeded five percent in any defined risk group.

In contrast, in countries facing concentrated epidemics (second state), HIV has spread rapidly in a defined sub-population, but is not well-established in the general population. As a numerical proxy, HIV-prevalence is consistently over five percent in at least one defined subpopulation, but below one percent in pregnant women in urban areas.

In countries facing generalized epidemics (third state), HIV is firmly established in the general population of sexually active adults with HIV-prevalence levels consistently over one percent in pregnant women. Heterosexual transmission is always the dominant mode for the spread of HIV in generalized epidemics. As the epidemic matures, most of the people whose behavior puts them at risk for infection will already be infected. Thus, it is expected that new infections in risk groups may decline or level off due to the saturation of the susceptible hosts.[180]

The differentiation of the three epidemic states is mentioned in order to make clear that the numerical cut-off point criteria of "only" one percent of HIV-prevalence in pregnant women seems to be low. However, this already defines a generalized HIV/AIDS epidemic. At least 54 of the countries in the final sample (N=197) have been defined as countries facing generalized epidemics in 2002.[181] At the end of 2003, already 72 out of 197 countries in the sample are estimated to face HIV-prevalence levels of over 1% of not just pregnant women but the whole adult population. There, small shifts in general population prevalence translate into a massive burden for health services, particularly in large countries. That is simply explained by the sheer numbers of HIV-positive people in the general population.

[179] See UNAIDS/WHO Working Group on Global HIV/AIDS/STI Surveillance: guidelines for second generation HIV surveillance (2000: 24).
[180] See Levin et al. (2001).
[181] Garcia-Calleja et al. (2004) examine 132 countries, which are part of this analysis we well. Out of these, 54 have been defined as countries facing "generalized epidemics", 30 as facing "centralized epidemics", and 48 as facing "low epidemics".

6.2. Measurement of the Key Explanatory Variable (Conflict Involvement)

The key explanatory variable, `conflict involvement´, proxies many of the previously mentioned variables associated with high HIV-prevalence rates (such as increasing prostitution, rape, refugee flows, military funding substituting for spending on education and health and mobility of soldiers). As mentioned earlier, conflicts taking place before 1995 cannot be linked to people still alive and tested HIV-positive at the end of 2003. Thus, I exclude them from my analysis.

To measure `conflict involvement´, I refer to data compiled by Gleditsch et al. (2004). I use the dataset on armed conflicts 1946–2001, the dataset on unclear cases of armed conflicts 1946-2001 as well as the dataset on armed conflicts active in 2002.[182] From these three datasets, I created the following indicators:

`Conflict involvement´ (x1) equals the total number of all types of conflicts (wars, intermediate and minor conflicts)[183] a country was involved in between 1995 and 2002.

`Conflict involvement, extended sample´ (x10) equals the total number of all types of conflicts a country was involved in between 1995 and 2002, including unclear cases of conflict involvement.[184]

---

[182] All conflict data used is based on version 2.1 of the dataset on `Armed Conflict 1946–2001´ as first described by Gleditsch et al. (2002). Data has been downloaded from the dataset homepage (see http://www.prio.no/cwp/armedconflict, 15.8.2004).

[183] Definitions: `Armed conflict´ refers to `a contested incompatibility that concerns government and/or territory where the use of armed force between two parties, of which at least one is the government of a state, results in at least 25 battle-related deaths´ (Gleditsch & Strand & Wilhelmsen 2004: 3). Armed conflict is categorized into `Minor Armed Conflicts´ with at least 25 battle-related deaths per year and fewer than 1000 battle-related deaths during the course of conflict, `Intermediate Armed Conflicts´ with at least 25 battle-related deaths per year and an accumulated total of at least 1,000 deaths, but fewer than 1,000 per year, and `Wars´ with at least 1,000 battle-related deaths per year (see Gleditsch & Strand & Wilhelmsen 2004: 4). Changes in intensity (e.g. from intermediate conflict to war in the following year) have been coded as two separate conflicts. In cases of uncertainty of intensity (e.g. minor conflict in Guinea 2000-2001, but possibly intermediate conflict in 2001) the presumed increase of intensity of warfare has been taken into account (Guinea has been coded as a country experiencing a minor conflict in 2000 and intermediate conflict in 2001). A state was found to be "involved" in a conflict if its government, opposition or both were fighting on the countries own territory or if fighting groups were participating in external conflicts. To include this last type of involvement seems important as AIDS is often spread by soldiers returning from or fighting in external conflicts. Participation in international or multilateral forces (ECOMOG in Sierra Leone or the Multilateral Coalition against Terror) is not included due to problems in categorization and lack of data.

[184] "Unclear cases" of conflict involvement are cases with insufficient information concerning at least one of the three components of the armed conflict definition: a) the number of battle-deaths, b) the identity or level of organization of a party or c) the type of incompatibility. Cases that have been completely rejected on the grounds that they definitely do not meet the criteria of armed conflict are not included in the unclear cases list. Based on this information and as fighting definitely took place, unclear cases qualify for being included in the sample.

`War involvement´ (x2) equals the total number of wars a country was involved in between 1995 and 2002.

`Involvement in intermediate armed conflicts´ (x3) equals the total number of intermediate armed conflicts a country was involved in between 1995 and 2002.

`Involvement in minor armed conflicts´ (x4) equals the total number of minor armed conflicts a country was involved in between 1995 and 2002.

`Years spent in conflict´ (x5) equals the total number of years a country spent in conflict between 1995 and 2002 (each country's involvement in at least one conflict (minor armed conflict, intermediate armed conflict or war) in each year was coded as a one, non-involvement as a zero).

`Years spent in conflict, extended sample´ (x11) equals the total number of years a country spent in conflict between 1995 and 2002, including unclear cases of conflict involvement.

Using these different indicators increases the validity of measurement and enables one to find out whether the type of conflict involvement (war, intermediate or minor) or the duration of conflict involvement matters in regard to the explanation of HIV-prevalence rates. Whenever possible, I will use measurement based on the extended sample, which includes more cases of conflict involvement.

Additionally, it might be of importance to examine how long ago a country was involved in conflict. In order to measure the impact of the peace time since last conflict involvement on HIV-prevalence I refer to the following two indicators:

`Peace duration´ (x9) equals the total number of years since last conflict involvement (whether this has been a war, intermediate or minor conflict).

`Peace duration, extended sample´ (x12) equals the total number of years since last conflict involvement, including unclear cases of conflict involvement.

However, a distinction between the type of conflicts involved cannot be made. Thus, using the extended conflict sample, which includes unclear cases, is not possible for measure x2, x3 and x4.

In accordance with Gregson et al. (2001), I test whether there exists a non-linear, inverse U-curve relationship between the peace duration after conflict involvement and HIV-prevalence. The corresponding explanation suggests that HIV/AIDS can be described as an "epidemic of development" and that vulnerability particularly occurs during the often fragile post-conflict period.

As already discussed, demobilization of soldiers and repatriation of refugees in post-conflict settings might be related to increases in HIV-prevalence, particularly in countries which have experienced decades of war-related isolation.
In addition, the emergence of peace will likely lead to increases in trade, migration and travel.[185] Thus, I argue that with peace also comes economic development, which is associated with processes, such as formal sector employment, urbanization, intensive short-term labor-migration and better transport infrastructure. The last three of these developments particularly intensify mobility and interactions between populations, which, in return, facilitates rapid and extensive transmission of HIV-infection from urban to rural populations. In addition, formal sector employment initially results in disparity in income and education. As wealth becomes concentrated in major cities and urban areas, this in return even increases rural-urban labor-migration, mobility of populations and urbanization levels. For instance, the recent spread of HIV-infection in South-East Asia has been linked to improved roads and transport infrastructure as well as economic growth coupled with economic disparity and high levels of rural-urban migration.[186]

Again, these changes at the macro-level affect the micro-environment of individuals and eventually HIV risk behavior. Most importantly, urbanization changes sexual mixing patterns. The rate of sexual partners increases as opportunities for sexual networking increase in cities. There, traditional value systems have less influence and social norms and values change rapidly. For example, economic change often increasingly "commodifies" sexuality, whether through advertising or prostitution.[187] Examples would include Vietnam or China, where increasing economic development and transition to market economy came along with an expansion in social openness, increasing drug use and the emergence of prostitution in big cities.[188]

[185] See Williams et al. (2002).
[186] See Hsu et al. (2003: 12).
[187] See Altmann (1999: 563).
[188] See Gorbach et al. (2002: 39, 40).

Altmann (1999) summarizes that the rapid spread of HIV/AIDS in the past two decades is closely related to the forces of "development." It is likely that the HIV-virus was spread beyond its original home through urbanization and population shifts. Evidence of this comes from Tanzania, Rwanda, Zimbabwe and Zaire where the initial spread of HIV and the initial cases of AIDS were identified in the populations with relatively higher socio-economic status (income and education) and populations who were involved in trade.[189] Thus, scientists criticize policies of international bodies, which promote faster economic development in combination with cut backs in resources available for public health and education campaigns. It is argued that structural adjustment, as promoted by the World Bank during the 1980's, added to the conditions that made people vulnerable to HIV-infection.[190]

Although the information given describes mechanisms how post-conflict development may tend to increase susceptibility to major HIV/AIDS epidemics at the population level, other aspects associated with enduring peace and development give hope. Due to more highly educated populations and rising awareness, extensive and better quality primary health care services, more developed health infrastructures and greater resources that could be deployed for HIV-control, it might be expected that increasing development allows for more rapid control and effective responses.[191]

In addition, public health situations in refugee and internally displaced population camps also improve with enduring peace. There is even evidence that refugees and internally displaced persons in 52 Asian and African post-emergency phase camps, which existed for four or more years, had better reproductive health outcomes than their respective host country and country-of-origin populations.[192] This result is attributed to improved access to preventive and curative health care services, availability of food, clean water and sanitation. All but one of the camps offered condoms to residents and most of the camps had gender-based violence programs in place.[193] Generally, logistics systems become more robust, allowing providers to expand the range of products and to improve routine efficiency of programs.[194] Although there might exist a selection bias (as several post-emergency camps could not be included in the sample due to logistical constraints or lack of government or NGO authorization) refugee populations in post-conflict camp settings stabilize with progressing peace.

[189] See South African Presidential Aids Advisory Panel Report (2001: 42); Jones (1998: 62).
[190] See Altmann (1999: 565, 574); Poundstone (2004: 29); At least in the short-term, structural adjustment programs have been associated with intensified rural-urban migration, increasing inequality, urban poverty and the intensified marginalization of women (see Jones 1998: 61-63).
[191] See Gregson et al. (2001).
[192] See Hynes et al. (2002); Spiegel (2004: 22).
[193] See Hynes et al. (2002: 601, 602).
[194] See Crystal et al. (2004: 19).

Therefore, peace and subsequent development are expected to be positively correlated with HIV-prevalence only in the short run; whereas in the long run, enduring peace and development should result in decreasing growth rates of HIV-infection and lower HIV-prevalence.

Finally, war involvement of neighboring countries has been found to affect HIV-prevalence rates in bordering nations. Ghobarah et al. (2003) even state that the impact of neighboring civil wars on HIV/AIDS is "immediately apparent" and "enormous."[195] In the case of Angola, Santos-Ferreira et al. (1990) also conclude that "penetration of HIV-infection in Angola is taking place from neighboring countries and spreading over and from war zones" (Santos-Ferreira et al. 1990: 785). This points to international spill-over effects related to mechanisms, such as refugee flows. In addition, very recent research by Collier et al. (2004) finds that civil war in one country reduces the economic growth rates of neighboring countries by around 0.9 percentage points. Associated increases in poverty might, therefore, link neighboring conflict involvement to health outcomes in bordering nations. The authors also find that military spending by one country - due to civil war - increases the average military spending of neighboring countries.[196] As far as these developments associate with reduced health expenditures, an "arms race" might influence health outcomes in bordering countries.

In order to test the hypothesis of a positive and significant impact of neighboring war involvement on HIV-prevalence in bordering nations, I use the following two measures of neighboring war involvement. Both account for the number of neighboring nations:[197]

`Average number of wars in neighboring countries´ (x17) divides the total number of wars in neighboring countries between 1995 and 2002 through the number of neighboring countries.

`Proportion of neighboring countries experiencing war´ (x31) divides the total number of neighbors experiencing war between 1995 and 2002 through the number of neighboring countries.

[195] See Ghobarah et al. (2003: 200).
[196] See Collier et al. (2004: 134).
[197] Data on the number of neighboring countries is given in the CIA World Factbook (2003). Neighboring countries are defined as countries sharing a land boundary. Guantanamo Bay (leased by the U.S.) and Vatican City have not been coded as extra countries; West Bank and Gaza has been coded as one country; Islands have been assigned a value of 0 for both measures of neighboring conflict involvement.

Of the 197 cases in the sample, 58 have experienced conflict; whereas 139 countries were not involved in any form of conflict between 1995 and 2002. When using the extended sample, which includes unclear cases of conflict involvement, 64 countries have experienced conflict compared to 133 with no conflict involvement. Angola has the highest number with 13 different conflicts and seven different wars. Myanmar has experienced the maximum number of intermediate conflicts (5) whereas Ethiopia has been involved in most minor conflicts (6). Twenty six countries in the sample have seen their most recent conflict involvement in 2002.

Although the average duration of conflict involvement for the whole sample is only 15 months out of the eight year period, Algeria, Angola, Burundi, Colombia, India, Israel, Myanmar, Pakistan, the Philippines, Somalia, Sudan, Turkey and Uganda have been involved in at least one conflict every single year.

Eritrea, Djibouti, Canada, Kenya, Central African Republic, Angola, Democratic Republic of Congo, Botswana and Burundi border nations of which 66% or more have been involved in war between 1995 and 2002. The following countries are surrounded by neighbors who have experienced, on average, at least one war in the eight year period: Angola, Botswana, Burundi, Central African Republic, Democratic Republic of Congo, Republic of Congo, Kenya, Namibia, Rwanda, South Africa, Sudan, Tanzania, Uganda, and Zambia.

7. Bivariate Descriptive Analysis of Conflict and HIV-Data

The average HIV-prevalence rate (y1) for countries with no conflict experience is 2.12% (median 0.3) as compared to an average HIV-prevalence of 3.53% (median 0.85) for countries that have been involved in conflict. Using low (y2) or high (y3) HIV-estimates as well as the extended sample including unclear cases of conflict involvement shows a similar pattern. This is illustrated by the figures in Table 3 on the following page.

Table 3: Descriptive Analysis, Conflict and HIV-Data

CONFLICT INVOLVEMENT	HIV-PREVALENCE (Y1), MEAN	LOW ESTIMATES OF HIV-PREVALENCE (Y2), MEAN	HIGH ESTIMATES OF HIV-PREVALENCE (Y3), MEAN
No	2.12 (0.3 Median)	1.60 (0.2 Median)	3.03 (0.5 Median)
Yes	3.53 (0.85 Median)	2.60 (0.45 Median)	5.12 (1.6 Median)
No (including unclear cases)	2.19 (0.3 Median)	1.67 (0.2 Median)	3.10 (0.5 Median)
Yes (including unclear cases)	3.26 (0.65 Median)	2.37 (0.4 Median)	4.78 (1.15 Median)

Countries surrounded by neighbors which have experienced, on average, one or more wars between 1995 and 2003 show a very high mean HIV-prevalence rate of 7.16%. Compared to this, the mean HIV-prevalence is less than 2% in countries in peaceful environments (with an average number of wars in neighboring countries<1).

The 34 countries in the sample with a medium duration of conflict involvement (between one and five years) exhibit a much higher average HIV-prevalence rate at the end of 2003 (4.37% (y1), 3.33% (y2), and 6.03% (y3)) as compared to the 139 countries with a short duration of conflict involvement of less than one year (2.12% (y1), 1.60% (y2), and 3.03% (y3)). Surprisingly, the 24 countries, which have experienced very extensive conflict involvement (six to eight years), show a similarly low average HIV-prevalence rate (2.35% (y1), 1.56% (y2), and 3.84% (y3)).

In summary, the descriptive analysis indicates that within the classification of conflict experiencing countries, those with a medium duration of conflict involvement exhibit a higher average HIV-prevalence rate. Cases contributing most to a particularly high average HIV-prevalence within this subgroup are Zimbabwe (with four years of conflict experience and an average HIV-prevalence of 24.6%), Namibia (five years of conflict involvement, 21.3% HIV-prevalence), Lesotho (one year of conflict involvement, 28.9% HIV-prevalence) and the Central African Republic (two years of conflict involvement, 13.5% HIV-prevalence).

In general, countries with conflict involvement show a higher mean HIV-prevalence rate when compared to those without any conflict involvement. The same holds true for countries surrounded by neighbors with high war involvement.

8. Tests on Mean Differences

The first statistical analysis of this paper tests the expectation that the following countries will show particularly high mean HIV-prevalence rates at the end of 2003: (1) countries which experienced conflict, (2) countries which experienced at least one war, (3) countries surrounded by neighbors which have been involved in at least one war on average and (4) countries with a medium duration of conflict experience.

Conflict involvement, war experience and neighboring war involvement of more than one war on average between 1995 and 2002 have been coded as a one. No conflict, no war experience and war experience in neighboring countries of less than one war on average have been coded as a zero. For each of the conflict measures tests of statistical significance are performed on the difference of means where the Null-Hypotheses (H_0) state that the mean(0) – the mean(1)=diff = 0 and the Hypotheses (H_a) state that the mean(0) – the mean(1)= diff< 0.

In addition, countries with a medium duration of conflict involvement are compared to the group of countries with a very short or very long duration of conflict involvement. The Null-Hypothesis (H_0) in that case states that the mean (short or long duration of conflict experience) – the mean (medium duration of conflict experience) =diff =0 and the Hypothesis (H_a) states that the mean (short or long duration of conflict experience) – the mean (medium duration of conflict experience) =diff <0. Differences in the mean HIV-prevalence of the groups compared are expected to be statistical significant.

A t-test is a common, but in this case not appropriate, method for comparing mean differences. The two-sample t-test on mean differences is a parametric test which assumes that data are normally distributed and both samples have the same standard deviation (SD). These assumptions are violated in the case of data with a skewed distribution, where means and medians differ.

As already mentioned, the distribution of the HIV-prevalence rates is heavily positively skewed. In addition, a look at the mean HIV-prevalence and its standard deviation for countries with and without conflict involvement reveals that the mean is less than 1.7 * SD for countries with conflict whereas the mean is more than 2.5 * SD for countries without conflict involvement. The standard deviation is larger when the mean is larger. Thus, assuming normality, equal standard deviations and using a t-test on these data should be avoided.

There are two solutions to this problem. First, a non parametric method can be applied, in which no assumptions are made about the basic distribution of the data. For two independent samples, the appropriate procedure is the Mann Whitney Test, which is equivalent to the Wilcox Rank Sum Test. There, the two samples are combined and the scores are ordered according to their rank. The test measures the number of times that scores of one group outrank scores of the other group. The Null-Hypothesis states that both samples are drawn from populations with the same distribution. If this is true, there will be no tendency for ranks from one sample to be larger than those from the other sample. For skewed data, comparing means may not be appropriate, so medians can be used to give estimates of the effect of conflict involvement on median HIV-prevalence. The corresponding test performs a non-parametric k-sample test on the equality of medians where the Null-Hypothesis states that the two samples are drawn from populations with the same median.[198]

However, analysis based on ranks has the disadvantage of losing information. This is avoided by the second solution which transforms the data, so that the assumptions of the t-test are satisfied and a t-test on mean differences can be applied. Taking natural logarithms (logarithmic transformation) of positively skewed data or applying Box-Cox transformation are often useful strategies to make non-normal data resemble normal data.

Table 4 on the following page presents summary statistics on transformed and non-transformed HIV-data by groups of comparison. Obviously, standard deviations of the two samples of transformed data are much closer than standard deviations of the two samples of non-transformed data.

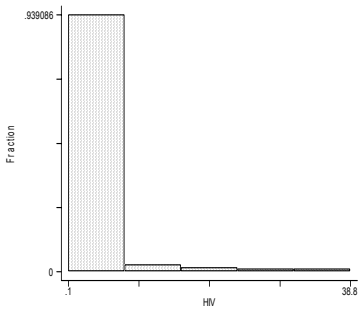
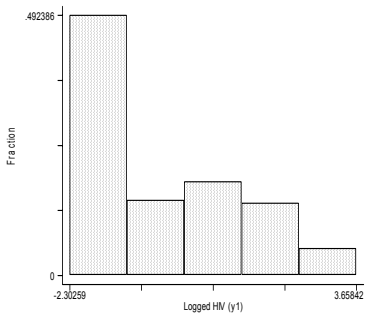
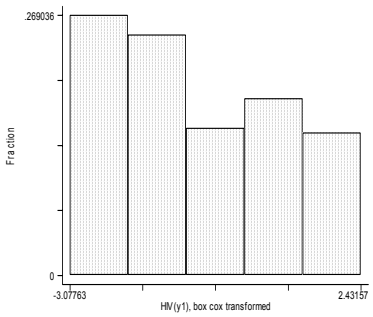
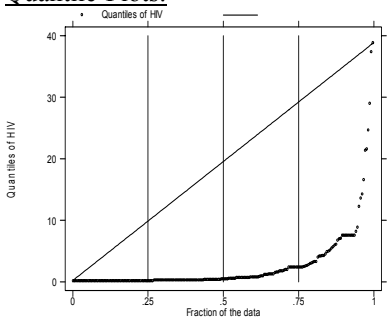
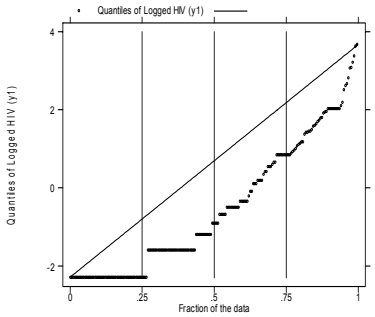
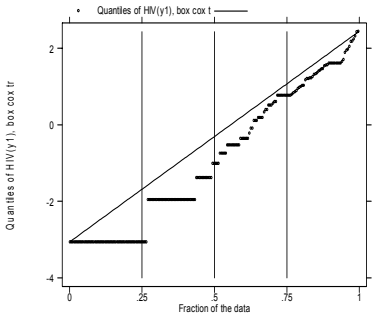
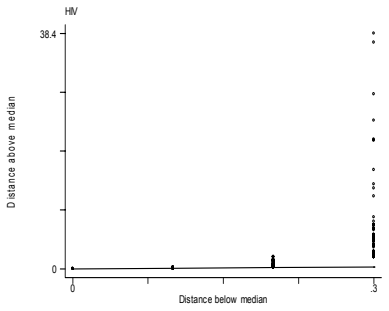
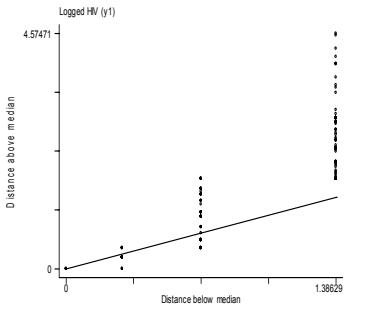
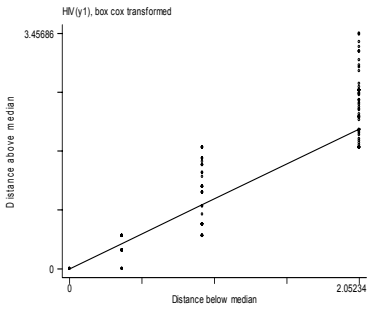
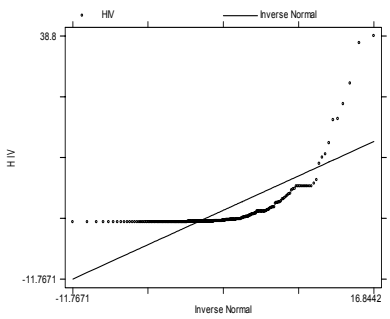
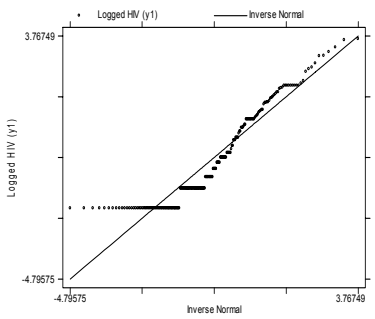
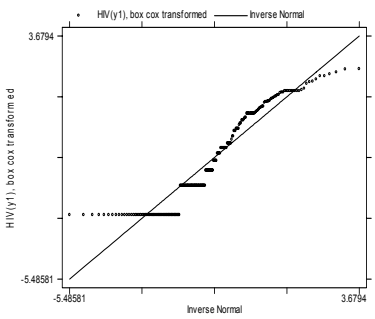
[198] See Fahrmeier et al. (2001: 425-429, 445-446).

Table 4: Summary Statistics of Transformed and Not-Transformed HIV-Data, by Groups of Comparison

GROUPS COMPARED		OBS.	MEAN HIV-PREVALENCE		SD	MIN.	MAX.
Conflict involvement	yes	58	(y1) ln(y1) boxcox(y1)	3.53 -.07 -.47	5.86 1.82 1.92	.1 -2.30 -3.08	28.9 3.36 2.31
	no	139	(y1) ln(y1) boxcox(y1)	2.12 -.70 -1.08	5.40 1.57 1.69	.1 -2.30 -3.08	38.8 3.66 2.45
War involvement	yes	30	(y1) ln(y1) boxcox(y1)	3.95 .34 .005	5.70 1.68 1.75	.1 -2.30 -3.08	24.6 3.20 2.23
	no	167	(y1) ln(y1) boxcox(y1)	2.29 -.67 -1.07	5.51 1.62 1.74	.1 -2.30 -3.08	38.8 3.66 2.43
Average no. of wars neighbors have been involved in	>=1	23	(y1) ln(y1) boxcox (y1)	7.16 .99 .59	9.21 1.72 1.63	.1 -2.30 -3.08	37.3 3.62 2.42
	<1	174	(y1) ln (y1) boxcox (y1)	1.93 -.71 -1.10	4.59 1.56 1.71	.1 -2.30 -3.08	38.8 3.66 2.43
Duration of conflict involvement	low	139	(y1) ln(y1) boxcox (y1)	2.12 -.70 -1.09	5.40 1.57 1.69	.1 -2.30 -3.08	38.8 3.66 2.45
	medium	34	(y1) ln(y1) boxcox (y1)	4.37 -.07 -.52	7.29 1.96 2.03	.1 -2.30 -3.08	28.9 3.36 2.31
	high	24	(y1) ln(y1) boxcox(y1)	2.35 -.06 -.39	2.51 1.62 1.81	.1 -2.30 -3.08	7.5 2.01 1.60

In addition, visual checks of normality (Histograms, Quantile-Plots, Symmetry-Plots and Normal-Plots) show the distribution of the HIV-data before and after the conducted transformations. The graphs are presented on the following page and clearly indicate that the transformed data are more normally distributed than data which has not been transformed.

Figure 1: Distribution of the Dependent Variable, HIV-Prevalence (y1), Before and After Transformations

DISTRIBUTION OF HIV-PREVALENCE RATES (Y1)	DISTRIBUTION OF HIV- PREVALENCE AFTER LOGARITHMIC TRANSFORMATION	DISTRIBUTION OF HIV- PREVALENCE AFTER BOX-COX TRANSFORMATION
Histograms: 		
Quantile-Plots: 		
Symmetry-Plots: 		
Normal-Plots: 		

Both statistical methods mentioned (rank sum tests on means and medians as well as t-tests on transformed data) have been applied to compare differences in HIV-prevalence levels. Generally, results indicate that in addition to being substantively significant, each of the difference of means or medians is also statistical significant at the 0.05 level, or in most cases even at the 0.01 level.[199] All methods reveal similar results in regard to the significance and substance of mean or median differences. For this reason, the following tables (Table 5.1.-5.4.) only report the results from the two-sample t-test on mean differences using logged HIV-data. The tables present the logged means, standard errors in parenthesis, the number of observations for each group of comparison, the differences of logged means, the 95% confidence intervals for the differences of logged means and the levels of significance. In order to get a correct interpretation, figures given in the tables need to be transferred back to their original scale using inverse logarithmic transformation.

As given in Table 5.1 below, the difference of logged mean HIV-prevalence for countries without and with conflict involvement is -0.64 (transferred back: 0.53). This indicates that mean HIV-prevalence in countries with conflict involvement is approximately twice the mean HIV-prevalence of countries without conflict involvement. In 95% of the time this multiplying factor will be between the antilog(0.13) and the antilog(1.14) (from confidence interval), that is between 1.14 and 3.13.

Table 5.1.: Two-Sample T-Test on Means, using logged HIV-data

GROUPS COMPARED	HIV-PREVALENCE (Y1), LOGGED MEAN	NO. OF OBS.
No conflict involvement	-.70 (.13)	139
Conflict involvement	-0.07 (.24)	58
Difference of logged means (Ha= diff<0)	**-.64 ***** (.26)	95% confidence interval ln[-1.14; -.13]

[199] Insignificant results are only found when rank sum tests on means and medians are applied in order to compare HIV-prevalence of countries with a medium conflict duration with countries with a low or high conflict duration. However, applying t-tests using logged or Box-Cox transformed data reveals significant differences (at the 0.05 level) in HIV-prevalence between these two groups. For all other groups and all other tests applied (non parametric ranks sum tests on means and medians as well as t-test using transformed data) results indicate that differences in HIV-prevalence are significant at the .01 or .05 level.

Similarly, the difference of logged mean HIV-prevalence for countries without and with war involvement is -1.01 (transferred back: 0.36). Therefore, HIV-prevalence in countries with war involvement is almost three times higher compared to mean HIV-prevalence in countries without war involvement. The confidence interval for the multiplying factor ranges from 1.45 to 5.21 (see Table 5.2.).

Table 5.2.: Two-Sample T-Test on Means, using logged HIV-data

GROUPS COMPARED	HIV-PREVALENCE (Y1), LOGGED MEAN	NO. OF OBS.
No war experience	-.67 (.13)	167
War experience	.34 (.31)	30
Difference of logged means (Ha= diff<0)	**-1.01** *** 95% confidence interval ln[-1.65;-.37] (.32)	

Table 5.3 indicates that mean HIV-prevalence in countries surrounded by neighbors with high war involvement is over five times the mean HIV-prevalence of countries in peaceful environments. In 95% of the time, the multiplying factor will be between 2.77 and 10.91.

Table 5.3.: Two-Sample T-Test on Means, using logged HIV-data

GROUPS COMPARED	HIV-PREVALENCE (Y1), LOGGED MEAN	NO. OF OBS.
Average number of wars in neighboring countries <1	-.71 (.12)	174
Average number of wars in neighboring countries >=1	.99 (.36)	23
Difference of logged means (Ha= diff<0)	**-1.70** *** 95% confidence interval ln[-2.39;-1.02] (.35)	

For countries with a medium duration of conflict involvement mean HIV-prevalence is about 1.7 times higher compared to the group of countries with a very short or long duration of conflict involvement. In this case, the confidence interval for the multiplying factor ranges from 1.08 to 3.19 (see Table 5.4.).

Table 5.4.: Two-Sample T-Test on Means, using logged HIV-data

GROUPS COMPARED	HIV-PREVALENCE (Y1), LOGGED MEAN	NO. OF OBS.
Short or long duration of conflict involvement	-.61 (.12)	163
Medium duration of conflict involvement (1-5 years)	-.07 (.34)	34
Difference of logged means (Ha= diff<0)	**-. 54** ** (.31)	95% confidence interval ln[-1.16; .08]

Notes Table 5.1. - 5.4.: ***indicates that Ha=diff <0 is significant at the 1% level, **indicates that Ha=diff <0 is significant at the 5% level, *indicates that Ha=diff <0 is significant at the 10% level;

In summary, the bivariate analysis clearly indicates support for the hypothesis that countries with prior conflict or war involvement have a significantly higher average HIV-prevalence rate at the end of 2003. Although a more direct analysis would compare countries' HIV-prevalence rates prior and after conflict involvement, comparable data is not available for that kind of analysis. Differences in mean HIV-prevalence rates are significantly higher, even at the 0.01 level, for countries surrounded by neighbors who experienced, on average, one or more wars between 1995 and 2003.

There is also initial support for the hypothesis that very extensive conflict involvement as well as very short conflict involvement seems to be associated with relatively low HIV-prevalence. Compared to the relatively high mean HIV-prevalence rate of countries with a medium duration of conflict involvement, the difference of means is significant. This points to a possibly non-linear, inverse U-curve relationship.

The first section of this paper already explains how low HIV-prevalence is related to low conflict involvement. However, it still remains to be explained how low HIV-prevalence could also be associated with very extensive or intense conflict involvement.

Angola serves as an example with very high conflict involvement, but, at the same time, a very low adult HIV-prevalence rate at the end of 1999. USAID explains this in the following manner: "Angola's national HIV-prevalence rate of 2.78% is lower than the rates of neighboring Zambia, Namibia, Zimbabwe, and Congo, largely because of the isolating effects of the civil war" (USAID: Angola and HIV/AIDS: 1999: 1). A note published in the U.N.Wire on July 26th of 1999 relies on a Washington Post article and confirms that the ongoing conflict has essentially quarantined this country, limiting people's exposure to the outside world and, consequently, to carriers of HIV.[200] In their 2004 global report on the AIDS epidemic, UNAIDS/WHO confirm that compared with surrounding countries, prolonged conflict may have acted as a brake on HIV-spread in Angola.[201] Therefore, it might be possible that long-term or intense conflict involvement delays the spread of AIDS.

This does not contradict what Santos-Ferreira et al. (1990) find in their study on the spread of HIV-infection in Angola as they examine the specific geographical distribution of HIV-cases. The authors find the highest HIV-rates among people in the northern areas near the Democratic Republic of Congo and conclude that penetration of HIV-infection in Angola is taking place from neighboring countries and spreading over from war zones.[202] However, it might still be the case that Angola's own extensive conflict involvement had a delaying effect on the growth of the national HIV-prevalence rate. In the absence of conflict, this rate may have reached the much higher, neighboring levels faster.

Similar arguments explain relatively low HIV-prevalence in "insulated" and war-torn Liberia. During the war, increased risk for HIV-infection resulted in relatively high prevalence among prostitutes. However, this did not translate into sustained increased infection among the general population. Movement within the country was very restricted and border-crossing migration became difficult due to conflict experience.[203] Recent research by Spiegel (2004) adds Sierra Leone and Southern Sudan to the list of countries in prolonged conflict with low HIV-prevalence relative to surrounding countries due to conflict-related isolation.[204]

[200] See U.N.Wire (July 26th of 1999).
[201] See UNAIDS/WHO: Report on the global AIDS epidemic (2004:180); see also Williams et al. (2002).
[202] See Santos-Ferreira et al. (1990: 784).
[203] See UNAIDS/WHO: Report on the global AIDS epidemic (2004: 178).
[204] See Spiegel (2004: 324).

Besides `isolation of communities´, Mock and Mathys (2002) mention other conflict-associated factors that might reduce the pace of infection. These include decreasing casual sex associated with trauma and depression, the disruption of sexual networks and increasing death rates among high-risk groups (soldiers) and others that are HIV-infected. All of these factors also particularly relate to extensive and intense conflict experience.

Davis et al. (2003) deliver another explanation and state that in prolonged conflicts and long-term displaced settlements, international humanitarian aid agencies become involved. Due to intense humanitarian involvement in these countries, better health care facilities, education and preventative measures become available, causing HIV-rates to plateau.[205]

It is because of these reasons that the question of whether there exists a non-linear relationship between the duration or intensity of conflict involvement and HIV-prevalence will be addressed with the following multiple regression analysis.

Limitations of the bivariate data analysis prevent us from drawing firm conclusions. Comparisons of means provide only initial exploration of the hypothesis that conflict involvement makes a difference in HIV-prevalence rates. The next step is to explore the question in a full multivariate analysis as bivariate tests cannot account for the possibility of a distorting influence of other variables. The next analysis employs a multivariate model that allows for the isolation of the impact of conflict involvement by controlling for factors previously shown to affect HIV-prevalence.

[205] Davis et al. (2003: 10).

9. Multiple Regression Analysis

9.1. The Control Variables

9.1.1. Economic Development and Wealth

The theoretical justification for the inclusion of many of the following control variables explaining cross-national variation in HIV-prevalence rates is based on a recent study published in the American Political Science Review of May 2003. In this article, Ghobarah et al. (2003) outline the causal connection between politics and public health.

The authors argue that the exposure to conditions that increase the risk for HIV/AIDS-related death and infection is influenced by a country's level of economic development and wealth. At the individual level, Davis et al. (2003) state that higher levels of wealth should limit HIV-proliferation due to "lack of penury that might compel women to become sex workers or engage in sexual activity in order to meet basic survival needs" (Davis et al. 2003: 11). Certainly the risk for HIV-infection assumes low priority among people's daily concerns in conditions of poverty[206] and, in accordance with prior arguments, biased risk perception affects HIV-risk behavior. In addition, high levels of income and wealth provide a larger pool of financial and human resources, which public and private actors can use to satisfy health care needs, to purchase medical technologies, for the development of human resources, for medical care, etc.[207]

Based on this, I hypothesize that the higher the level wealth and health spending is, the lower HIV-prevalence rates should be. Thus, the `**average per capita total expenditures on health at international dollar rate from 1997-2001´ (x24)** should serve as a good predictor of 2003 HIV-prevalence rates. This measure is calculated from data on public and private health spending given in the 2004 WHO World Health Report. It is argued that there exists some complementary between both public and private health spending in achieving health goals. For this reason, "the measure of total health expenditures has more explanatory power than either does alone" (Ghobarah et al. 2003: 194).

[206] See Buvé et al. (2002: 2014).
[207] See also Subramanian et al. (2002).

The second indicator used to measure the level of economic development and wealth is the **`2002 gross national per capita income (in thousands) converted to international dollars using purchasing power parity rates´ (x22)**. These data are provided by the World Bank and published as part of the 2004 World Development Indicators. Because the distribution of both measures is skewed, I take the natural logarithms. This also reflects the declining marginal product of additional dollars at higher levels of health expenditures, and models a non linear, logarithmic relationship between HIV-prevalence and economic development and wealth. [208]

9.1.2. Income Inequality

Subramanian et al. (2002) state that there are sound theoretical reasons for suggesting that inequality in the distribution of income has an impact on population health. "Given the same level of average income, a more unequal society is more likely to have greater numbers of people living in poverty, both in absolute and relative senses" (Subramanian et al. 2002: 295). In particular, those people who are adversely affected by income inequality are more likely to engage in risky health behavior. This is in line with Kawachi (2000) who states that disparity in income results in poor health through direct psychological pathways, effects of relative deprivation or frustration.[209]

Generally, the distribution of wealth has an impact on people's ability to gain access to health care and education services. In particular, once infected with HIV or opportunistic diseases, access to treatment is increasingly a matter of economic resources.[210]

In addition, income inequalities also often translate into political inequality. This might result in the negligence of health needs of high-risk but low-income groups.[211] It is argued that social exclusion and marginalization of the poor leaves them with little or no political influence and eventually results in the breakdown of community solidarity. This is supported by recent research indicating that citizens living in countries characterized by high income disparity tend to be more mistrustful of each other and tend to belong to fewer civic associations.[212]

[208] See Ghobarah et al. (2003: 193); Kawachi (2000: 77).
[209] See Kawachi (2000: 86-88).
[210] See Altmann (1999: 572).
[211] See Ghobarah et al. (2003: 191); Subramanian et al. (2002: 297).
[212] See Kawachi (2000: 87).

Thus, income inequality is negatively correlated with social capital and erodes social cohesion. This connects with the previously described Jaipur Paradigm. For this reason, the inclusion of the income inequality variable also serves as a test to the social cohesion concept of the Paradigm. In summary, it is expected that the afore mentioned processes result in lower overall levels of health performance (and higher HIV-prevalence) in countries with high income inequality.

Various studies have already supported a strong cross-country and within-country relationship between the level of disparity in income and health outcomes.[213] The **`gini index of income distribution´ (x27),** which theoretically ranges from 0.0 (perfect equality) to 1.0 (perfect inequality), is the most common indicator used. Thus, the following study also relies on the gini index of income distribution as provided by UNDP within the 2004 Human Development Report.

Confounding factors from mediating variables, e.g. poverty, educational attainment or levels of urbanization, need to be controlled for in order to find out whether the effect of income inequality on HIV-prevalence appears to be entirely explained by a compositional effect (e.g. a greater concentration of poor people who face higher risk for HIV-infection in countries with high income inequality). Only if the coefficient of the income inequality variable stays significant even when mediating variables are controlled for, there is evidence to support a contextual effect of income inequality on HIV-prevalence.

9.1.3. Urbanization

The exposure to conditions that increase the risk for HIV/AIDS-related deaths and infection is also influenced by the level of urbanization and the pace of urbanization a country undergoes.

[213] For an overview see Kawachi (2000).

9.1.3.1. Level of Urbanization

The greater prevalence of marginalized, underrepresented populations in cities results in a greater burden on available health care systems. Persons of lower socio-economic status face more barriers to care or receive poor quality care. In addition, "the prevalence of well-equipped, lucrative, practice opportunities in the same city decreases the likelihood that service providers will work in lower paid, public clinics" (Vlahov 2002: 8). This further promotes disparity in care in urban areas. Immunization, sanitation and the attainment of safe water also become more difficult in countries with large urban populations. This facilitates the spread of infectious diseases, including STDs and HIV.[214] Often, high levels of in- and out-migration (e.g. rural-urban labor-migration) of infected and susceptible persons contribute to more rapid spread of HIV in countries with higher urbanization levels.[215]

At the individual level, the "social environment" of the urban community (e.g. cultural milieu, social norms and networks) affects individual risk behavior. "Urbanization and modernization exchange traditional village norms for an urban modern ethos with fewer restrictions on sexual behavior and marriage…Furthermore, loss of culture and erosion of social networks are associated with social problems such as drug abuse, which encourages high risk behavior" (Buvé et al. 2002: 2014). Thus, altered sexual and drug use patterns, high levels of violence, psychological stressors and the presence of drugs and marginalized populations with high-risk behavior are principal features of the urban environment related to HIV/AIDS.[216] Evidence comes from Deheneffe et al. (1998), who find that at the individual level urban residence correlates with a higher prevalence of reported non-marital sex, commercial sex and symptoms of STDs.

9.1.3.2. Pace of Urbanization

It has been argued that health care systems often lag in large urban areas experiencing rapid population growth, fast growing urban poverty, rapid increases in prostitution, collapsing city infrastructure and deteriorating health services.[217] Thus, the pace of urbanization is of importance.

[214] See Ghobarah et al. (2003: 191, 194); or Guha-Sapir et al. (2002: 27).
[215] See Poundstone et al. (2004: 28).
[216] See Vlahov et al. (2002: 6).
[217] See Buvé et al. (2002: 2015).

Pace of urbanization will be measured through the **`average annual rate of change (%) of the urban population from 1995 to 2000´(x20).** These data are published by the United Nations Population Division (UNPD), Department of Economic and Social Affairs in the `World Urbanization Prospects, the 2003 revision´. The same report also provides the measure of urbanization levels, as it publishes data on countries' **`total percentage of urban population in 2003´ (x19)**.

9.1.4. Education

9.1.4.1. Level of Education

Both, the ability of groups in society to gain access to the health care system and their risk for infection are influenced by the level of education in society. Better educated people are likely to be more knowledgeable of health risk factors and prevention. Treatment programs become more widespread and effective and the demand for better health care rises.[218] As HIV/AIDS epidemics progress and effects on morbidity and mortality become clearly apparent, it is expected that higher educated people are better advantaged to adopt a safer lifestyle quicker due to formal instruction in school and easier access to information.

Gregson et al. (2001) find support for their hypothesis that there exists a trend towards reduced relative risk for HIV-infection among more educated individuals, which subsequently might be copied by their less educated contemporaries. The authors identify safer sexual behavior practices among young people with secondary education in contemporary rural Zimbabwe. In four Sub-Saharan African cities condom use has also been shown to be correlated with higher levels of education for non-spousal partnerships.[219] In accordance with previous findings, I, therefore, expect the model to reveal a negative correlation between levels of education and HIV-prevalence rates even after the confounding factors from mediating variables are controlled for. In order to overcome problems with limited data availability of other measures, I will use **`adult literacy rates (% of all 15 years old and above) in 2002´(x28)** to measure the level of education**.** These data were published by UNDP as one of the Human Development Indicators given in the 2004 Human Development Report.

[218] See Ghobarah et al. (2003: 191, 194).
[219] See Lagarde et al. (2001).

9.1.4.2. Disparity in Education

According to Gregson et al. (2001), another factor related to bigger HIV/AIDS epidemics is disparity in education levels between males and females. The measure of education inequality is the **`average ratio of literate women to men, 15-24 year old, 1995-2003´ (x29)**, which is provided by the United Nations Statistics Division as one of the Millennium Indicators.[220] This indicator is supposed to measure the enhanced socio-economic status of women associated with their rising education. Therefore, it partly captures and proxies for the gender dynamics of the disease grounded on economic, social, sexual and cultural sub-ordination of women.[221]

Once women are in a stronger position to build upon their socio-economic status, they can be more careful and selective in their sexual relationships. Education for women not only enhances their sense of self-efficacy, which reduces fatalism and inadvertent involvement in high-risk relationships, but also enhances their negotiating skills when it comes to condoms use with male partners. In contrast, the low social status of women in many societies encourages discrimination as well as domestic and sexual violence or abuse. Socially and economically subordinate girls remain socially and materially dependent on their much older husbands who, as a sign of virility, often have multiple sexual partners and adopt risky sexual practices. As a consequence, women are often infected by their polygamous husbands.

Additionally, Over (1998) argues that a larger gap between male and female literacy rates is likely to mean fewer conventional job opportunities or lower conventional wages for women. Therefore, greater inequality in education also means greater income inequality and increases in bargaining power of men in the upper income-brackets who would like to purchase commercial sex. The overall result is an increase in the demand as well as the supply of commercial sex.[222] Zierler et al. (1997) agree that women's struggles with and resistance to social and economic subordination include strategies for survival that bear the burden of drug use, violence, hunger, social disintegration and sexual risk.[223] In particular, women who drop out of school are more likely to resort to commercial sex or seek early marriages. All this places them at greater risk for HIV.

[220] See http://millenniumindicators.un.org/unsd/mi/mi_goals.asp, 10.7.2004.
[221] See O'Sullivan (2001); Johnson (2003); Buvé (2002).
[222] See Over (1998: 41).
[223] See Zierler et al. (1997: 411).

These arguments support the HBM and ARRM and are based on a "Political Economy of Health Approach," which assumes that the relative social and economic positioning of people shapes their risk behavior. The (power) relation between the subordinate and dominant group affects patterns of disease through material and social inequalities.[224]

An overview of studies examining women's risk for HIV-infection with respect to economic inequality and economic policies in Brazil, Haiti and Zaire is given by Zierler et al. (1997). Additional empirical evidence comes from Over (1998), who finds that gender inequalities, whether measured through the ratio of males to females in urban centers or the gap between adult male and female literacy rates, associates with higher HIV-prevalence.[225]

9.1.5. Population Density

Davis et al. (2003) hypothesize that population density is likely to raise the prevalence of HIV. They argue that more densely populated areas experience higher spread of HIV particularly in countries where HIV is mainly sexually transmitted. This is simply because levels of interaction among people exceed those of more sparsely populated regions.[226] Thus, I include the **`number of people per sq. km in 2002´(x21)** as a measure of population density. These data are taken from the 2004 World Development Indicators as provided by the World Bank.

9.1.6. Culture

In the absence of data on cultural norms that shape a society's sexual practices, I use the **`proportion of the population that is Muslim or Jewish in 2003´ (x23).** These data are taken from the 2003 CIA World Factbook and do not include the immigrant population. In accordance with previous findings by Over (1998), Mahal (2001), Bonnel (2000) or Quigley (2000), I expect this variable to be negatively correlated with both the supply of and demand for risky sexual behavior. In addition and as explained before, high levels of male circumcision are expected to significantly lower the risk for HIV-infection in countries with large Muslim or Jewish populations.

[224] See Zierler et al. (1997: 408).
[225] See Over (1998: 47).
[226] See Davis et al. (2003: 11).

9.1.7. Youth Bulge

Because the young sexually active age group is at greatest risk for HIV-infection, I include a "youth bulge" variable in the model. It is expected that countries with higher proportions of a young population face higher HIV-prevalence rates. I use data on the military manpower availability (total number of males age 15-49) as given in the 2003 CIA World Factbook to calculate the **`proportion of males age 15-49 in total population in 2003´(x25).** However, it is possible that a problem of endogeneity exists in countries that are heavily affected by HIV/AIDS and already face high AIDS death rates among the young adult population.

9.1.8. Lagged HIV-Prevalence

Finally, HIV/AIDS rates are highly correlated over time. Therefore, past values are expected to explain a great deal of year-to-year variability in current HIV-infection rates. Thus, I include lagged HIV-prevalence rates (**`end of 2001 HIV-estimates´ (x33)**) in order to address the temporal dependence in HIV-data. These data were published by UNAIDS/WHO in their 2002 report on the global AIDS epidemic.

9.2. Analytical Weight: Population Size

As this analysis deals with data containing national averages, analytical weights are appropriate. Thus, population size has not been included as a control variable, but all observations in the sample have been weighted according to their population size.
I used data on the **`total population of urban and rural areas in millions at mid-2003´ (x18)** as given in the `World Urbanization Prospects: the 2003 revision´ published by the United Nations Population Division (UNPD), Department of Economic and Social Affairs.

9.3. Missing Data

Missing data have been replaced by regional mean values wherever possible.[227] Otherwise, mean values according to each country's human development rank or mean values according to each country's income level have been used.[228] Due to lack of data, eleven cases have been dropped and deleted from the sample,[229] which now contains 197 countries.[230]

9.4. Non-Linearity

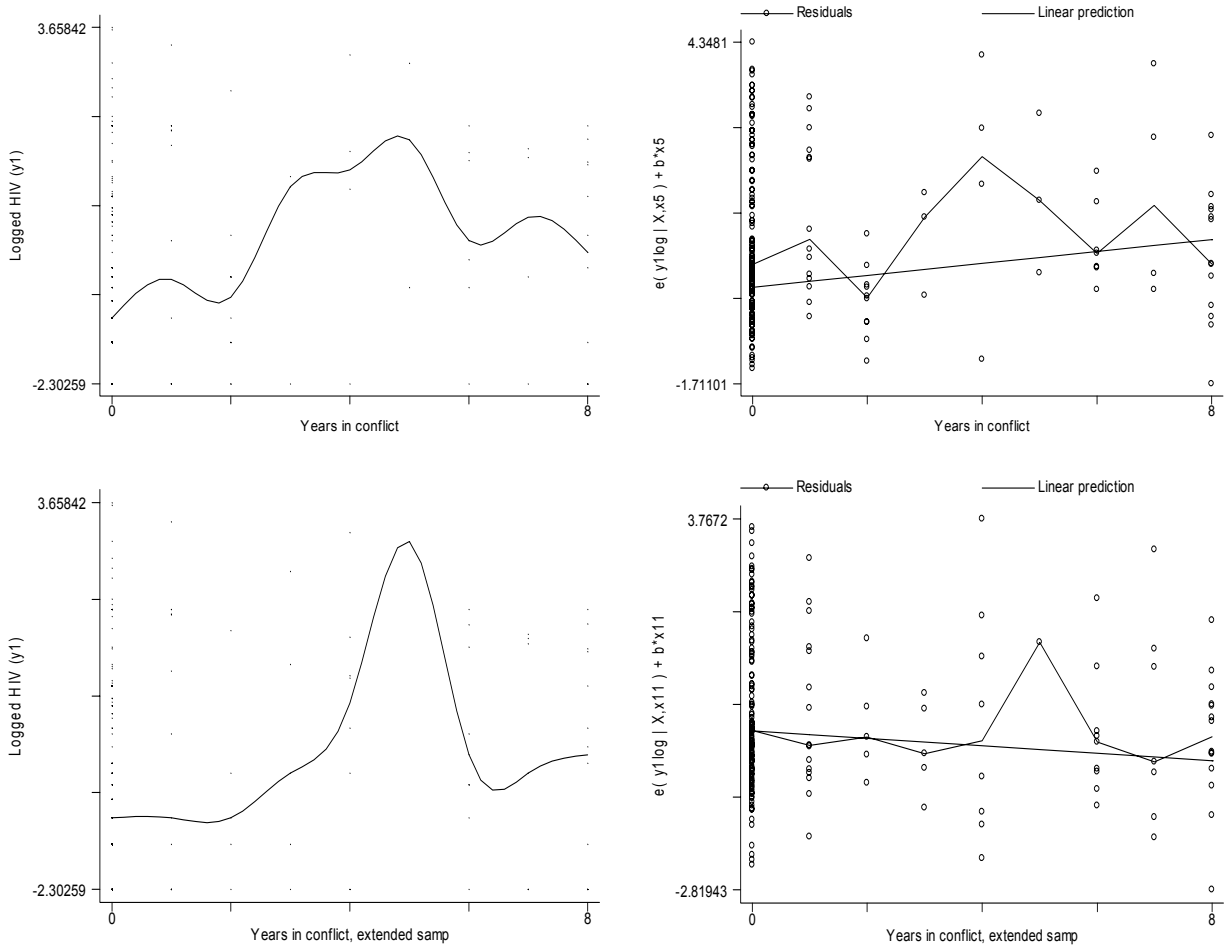
In order to produce the best linear unbiased estimator by applying OLS regression analysis, the assumed non-linear relationship between HIV-prevalence (dependent variable) and `extensive or intense conflict involvement´ (independent variable) needs to be taken into account. This requires a transformation of the independent variable.[231] Out of the conflict involvement measures used in this analysis, particularly x5 and x11 measure extensive conflict involvement as both refer to the duration of conflict involvement; in contrast x2 (measuring the number of wars a country was involved in) refers to intense conflict involvement in terms of battle deaths. In order to model the assumed inverse U-curve relationship, I, therefore, additionally include the squared values of x5, x11 or x2 in the models containing these variables.

As argued before, models including x9 or x12 (the peace duration since last conflict involvement) as the key explanatory variable are also supposed to test a non-linear, inverse U-curve relationship. This again requires a transformation of the independent variable and the additional inclusion of the squared value in the models.

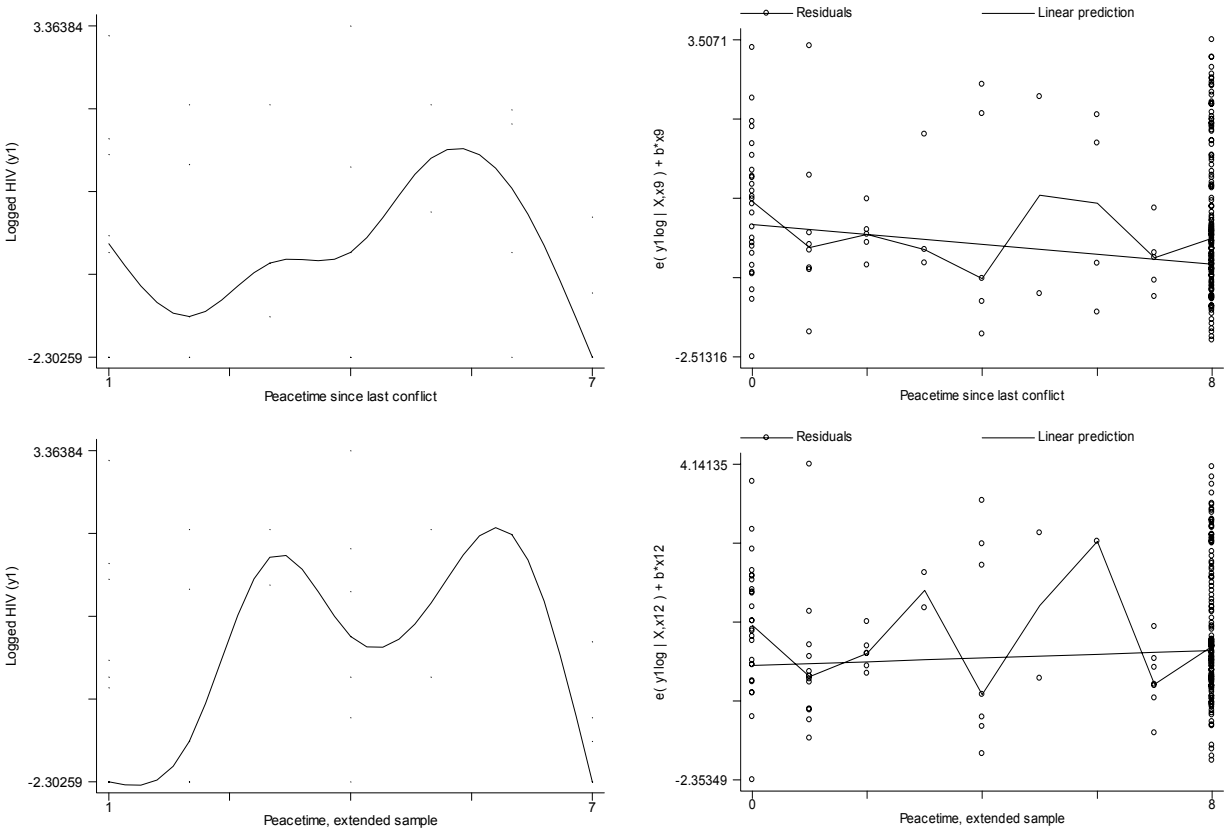
[227] For more information on replacement of missing values see Annex G. For the regional categorization, I refer to UNPD (World Urbanization Prospects: the 2003 revision: 15-17), the World Bank Regional Grouping (http://www.worldbank.org/data/countryclass/classgroups.htm, 13.7.2003) and the UNDP Regional Grouping (Human Development Report 2004: 281-282).
[228] For categorization into high, medium and low human development countries, I refer to UNDP (Human Development Report 2004: 279). Missing countries on the Human Development Index have been categorized as low, medium or high human development countries according to their income level. For categorization into high-income, middle-income and low-income economies I again refer to the World Bank Grouping.
[229] Deleted cases with missing values on more than six out of fourteen indicators used to measure the control variables are: Aruba, Bermuda, Channel Islands, Faeroe Islands, French Polynesia, Greenland, Isle of Man, Liechtenstein, Mayotte, Virgin Islands, and West Bank and Gaza.
[230] A list of the 197 countries included in the final sample is given in Annex F.
[231] See Kohler et al. (2001: 198, 199, 227-231).

Bivariate Scatter-Plots displayed in the left column of Figure 2 use Median-Traces as Scatter-Plot Smoothers and support the assumed non-linear relationships. Component-Plus-Residual-Plots presented in the right column of the same figure are already based on multiple regression analyses. Even when controlled for the effects of other independent variables, the graphs illustrate a non-linear relationship between HIV-prevalence and extensive conflict involvement and between HIV-prevalence and peace duration.

Figure 2: Bivariate Scatter-Plots with Median Traces vs. Multivariate Component-Plus-Residual-Plots [232]

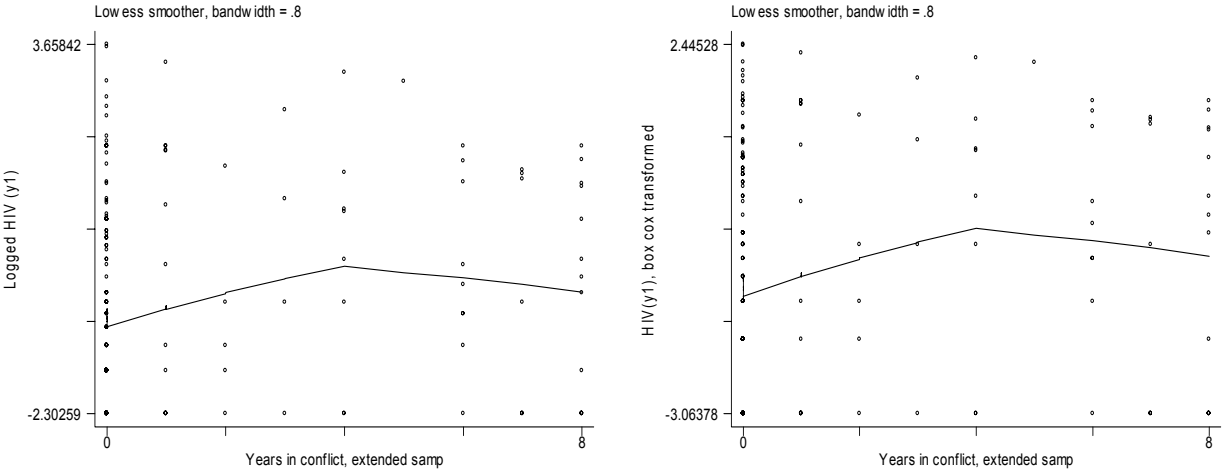


[232] The Multivariate Component-Plus-Residual-Plots are based on a regression command including the key independent variables (x5, x11, x9 or x12), x17, x20, x22, x27 and x28 as independent variables as well as x18 as the analytical weight.



Applying non-parametric bivariate regression analysis to model the relationship between HIV-prevalence and conflict duration (x11) also supports a modest non-linear relationship (see Figure 3).

Figure 3: Bivariate, Non-Parametric Regression Analysis (Lowess Smoother)



Non-linearity is also assumed in the case of the impact of income (x22) and health expenditures (x24) on HIV-prevalence rates. For this reason, the following regression analyses also include the logged values of these two independent variables.

9.5. Transformation of the Dependent Variable

The percentage ($0 \leq p \leq 100$) or the proportion ($0 \leq p \leq 1$) of a population infected by any contagious disease typically follows a S-shaped curve, starting slowly, then accelerating and then leveling off.[233] Thus, the underlying structure of the HIV-data is assumed to be non-linear. Applying linear multiple regression analysis in this case requires a transformation of the dependent variable. The recommended and applied transformation for proportions is Logit-Transformation, which assures that the predicted values fall between zero and one.[234] However, Logit-Transformation is not defined when y equals zero or one. Thus, cases with a low estimate of HIV-prevalence rates (y2) of zero percent would be lost. In order to avoid dropping these observations, low HIV zeros are set to 0.1% (or 0.001 as a proportion).

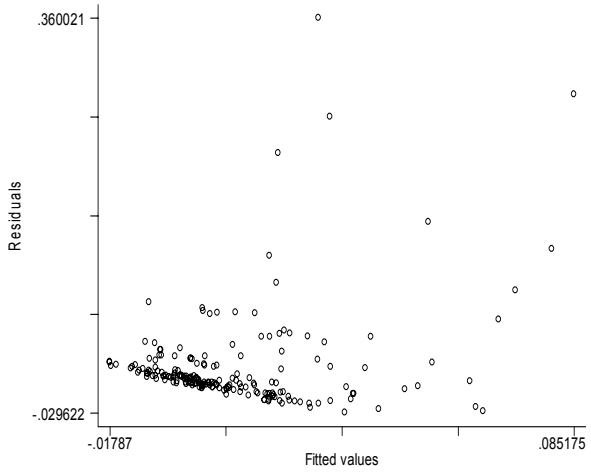
In addition and in accordance with the Gauss-Markov-Conditions, OLS regression coefficients are not efficient in case of heteroscedasticity.[235] Solving the problem statistically requires an additional Box-Cox Transformation of the positively skewed logit-transformed proportions. The Box-Cox Transformation requires all values of the dependent variable to be positive. If the dependent variable has zero or negative values, a constant, which is greater than the absolute value of the most negative value of the dependent variable, can be added to each score of the dependent variable. As the logged proportions contain negative values, a constant (+10) is added to each score before Box-Cox Transformation is applied.

In summary, this analysis takes the logit- and then Box-Cox transformed proportion of the HIV-positive population in each country as the dependent variable.[236] Residual–vs.-Fitted-Plots demonstrate that the conducted transformations resulted in a constant variance of error terms, the mean value of residuals equals locally zero and the estimated coefficients are, therefore, likely to be unbiased (see Figure 4 on the following page).

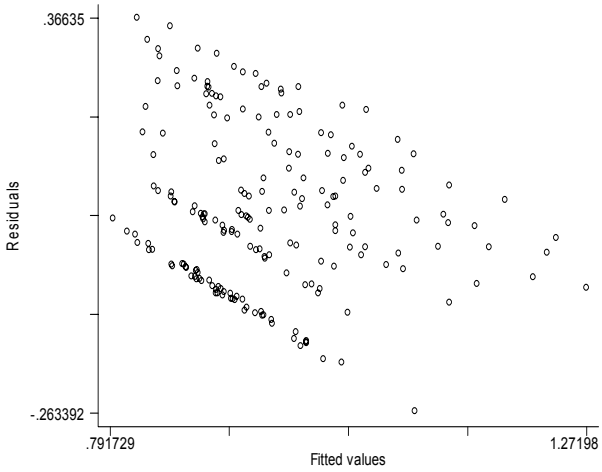
[233] See Whiteside et al. (2003: 8).
[234] See Schnell (1994: 81, 82); or http://apus.wiwi.hu-berlin.de/~sigbert/diplom/zanter/node10.html, 3.1.2005.
[235] See Kohler et al. (2001: 199, 216).
[236] An overview on data transformation methods used in this analysis is given in Annex D.

Figure 4: Residual-vs.-Fitted Plots [237]

Before Transformation of the Dependent Variable



After Transformation of the Dependent Variable



9.6. Bivariate Multicollinearity

After having specified all variables included in the model, it is possible to calculate the bivariate correlation matrix.[238] This confirms the validity of the different conflict involvement indicators as they correlate significantly with each other. The same holds true for the two measures of war involvement of neighboring countries.

In particular, a country's own war involvement correlates significantly and positively with HIV-prevalence. A medium duration of conflict involvement is positively and significantly correlated with involvement in intermediate and minor conflicts as well as with high estimates of HIV-prevalence. This supports the hypothesized non-linear relationship.

The inclusion of the control variables in the model is also justified because they exhibit statistical significant t-statistics of the proper sign. Only the negative sign obtained for the bivariate correlation among HIV-prevalence and the youth bulge variable is contrary to expectations.

[237] Both graphs are based on a regression command including x5, x17, x20, x22, x27 and x28 as independent variables as well as x18 as the analytical weight. The dependent variable is the proportion of HIV-positive people (y1) before and after transformation.
[238] See Annex C.

Population density is not significantly correlated with HIV-prevalence. The measure of disparity in education as well as the measure of the cultural variable tends to be significantly correlated only with transformed HIV-prevalence rates. The level of urbanization (x19) correlates significantly and negatively with HIV-prevalence, whereas the correlation coefficient of pace of urbanization shows a statistical significant but positive sign.

Bivariate multicollinearity can only be detected in the following two cases; the measures of income levels (x22) and health spending (x24) correlate highly with each other (0.9); correlation is also high (0.77) between the level of education (x28) and disparity in education (x29). In these two cases, correlation coefficients are near 1.0, which violates the assumption of no perfect collinearity while increasing the standard error of the beta-coefficients. This makes the assessment of the unique role of each independent difficult or impossible. For this reason, models will only include one, not both of the highly correlated variables.

Bivariate inter-correlation is strong but still acceptable between the transformed variables measuring economic wealth and development (x22, x24) and the variable measuring the level of urbanization (x19). All other correlation coefficients measuring the level of inter-correlation of independent variables remain far below a critical value of 0.9.

There are also no signs of bivariate multicollinearity among the measures of conflict involvement and other independent variables included in the model. Even inter-correlation between countries' own conflict involvement and conflict involvement of neighboring countries does not exceed a maximum value of 0.55 for the bivariate correlation coefficients.

9.7. Functional Form

The analysis applied is based on a simple multiple regression equation of the following functional form:

$$Y = b_1 * x_1 + b_2 * x_2 + \ldots b_k * x_k + c + e, \quad (1)$$

where Y is the true dependent variable, the b's are the regression coefficients of the corresponding x (independent/control) terms, c is the constant or intercept, and e is the error term reflected in the residuals.

The OLS regression includes the transformed dependent variable (HIV-prevalence rates), the key explanatory variable (conflict involvement), the squared value of the key explanatory variable in case of an assumed inverse U-curve relationship, the variable measuring war involvement of neighboring countries and the control variables [population density, economic development and wealth (transformed per capita income or per capita total expenditures on health), urbanization (pace of urbanization or level of urbanization), income inequality, education (level of education or disparity in education), the cultural variable (proportion of Muslims or Jewish population), the youth bulge variable (proportion of males age 15-49) as well as the lagged HIV-prevalence rates (end of 2001)]. The regression equation takes the following functional term:

$HIV_{(i,t)}$ (transformed) = b_1*(conflict$_{(i,t-T)}$) + b_2*(conflict$_{(i,t-T)}$)² + b_3*(population density$_{(i,t-T)}$) + b_4*(income inequality$_{(i,\ t-T)}$) + b_5*(HIV$_{(i,t-T)}$) + b_6*(youth bulge$_{(i,t-T)}$) + b_7*(culture$_{(i,t-T)}$) + b_8*(education $_{(i,t-T)}$) + b_9*(urbanization$_{(i,t-T)}$) + b_{10}*(level of economic development and wealth $_{(i,t-T)}$) + b_{11}*(neighboring war$_{(i,t-T)}$) + c + e [analytical weight = population size] (2)

where i represents the country, t the time period and T the time lag depending upon the availability of data.

9.8. The Final Baseline Model and Multivariate Multicollinearity

The inclusion of x29 (disparity in education), which requires the exclusion of x28 (level of education) due to multicollinearity tends to reduce R^2-values and/or significance of other coefficients. In addition, the effect of disparity in education remains insignificant in most cases. This is probably due to the presence of the income inequality variable in the same specification, which already partly captures the effect. Therefore, the level of education (x28) performs better in explaining HIV-prevalence and is included in the final baseline model. Similarly, x31 (measuring the proportion of neighbors experiencing war between 1995 and 2002) performs better in explaining HIV-prevalence compared to the other available indicator: x17 (measuring the average number of wars neighboring countries have been involved in between 1995 and 2002). Using x17 instead of x31 tends to reduce R^2-values and significance of the respective conflict coefficients. For this reason, the final baseline model includes x31 as the measure of war involvement of neighboring countries.

The coefficient of the `population density´ variable stayed insignificant, and the inclusion of this variable did not add much to the predictive power of the models. Thus, I dropped the population density variable from the analysis. This agrees with prior research, which also did not find a significant effect of population density on HIV-prevalence.[239]

As far as the remaining independent variables in the models are concerned, prior inspection of the correlation matrix revealed only bivariate multicollinearity. Therefore, I calculated Variance-Inflation Factors, which examine the correlation of each independent on all other independent variables in order to assess multivariate multicollinearity. Variance-Inflation Factors are high in cases of multicollinearity and instability of the b and beta-coefficients. A common cut-off criterion for deciding when a given independent variable displays "too much" multicollinearity is a value exceeding 4.0, although some researchers use the more lenient cut-off of 5.0 or even much higher values.[240]

Variance-Inflation Factors for the final regression and robust regression Models 1-10 (presented in Annex E) stay far below a value of 4.0. The robust regression of Model 9 is an exception with the maximum but still acceptable value of 4.19 for the Variance Inflation Factor of x28 (level of education). Thus, multivariate multicollinearity cannot be detected.

In summary, the final baseline model uses adult literacy rates (x28) as the measure of education levels, the transformed per capita health expenditures (x24) as the measure of wealth and the proportion of neighbors involved in war (x31) as the measure of war involvement of neighboring countries. As the inclusion of x19 (level of urbanization) tends to increase the level of multivariate multicollinearity, the final baseline model refers to x20 (pace of urbanization) as the urbanization measure. Each model also includes x27 (the level of income inequality), x33 (the lagged HIV-prevalence rates), x25 (the proportion of males age 15-49), x23 (the proportion of Muslims or Jewish population) as well as one of the twelve conflict involvement indicators. The results from the corresponding twelve regression models are presented in Annex E.
It shows the afore mentioned that out of all possible combinations of independent variables and their differing measures, this final baseline model delivers best results in terms of high R^2-values and significance as well as low levels of multivariate multicollinearity.

[239] See Davis et al. (2003).
[240] See e.g. http://www2.chass.ncsu.edu/garson/pa765/regress/htm, 22.8.2004; http://www.stat.ufl.edu/~winner/mar5621/mar5621.doc, 28.1.2005.

9.9. Results from the Multiple Regression Analysis

The calculated multiple regression analyses presented in Annex E show high R^2-values, which indicate that the models explain up to 68.57% of the variation in HIV-prevalence.[241] In all cases, the sum of squared residuals is significantly reduced by including the independent variables in the models.

As I transformed the dependent variable the interpretation of the coefficients is more complicated. Although relations between the transformed dependent and independent variables are assumed to be linear, relations are non-linear in terms of the original variables. Due to this it is not possible to draw any conclusion on the effects such as `increasing the explanatory variable by one unit of measurement leads to an increase of HIV-prevalence by x%´. In addition, although most of the coefficients are highly significant, this does not mean that their effects are theoretically important, proven or strong. The probability of finding a significant result, even if there exists no actual effect, equals the level of significance (α-error). Thus, significance tests are not tests of the strength of effects.[242] Where possible, I, therefore, refer to the standardized beta-coefficients given in italic figures in Annex E. This allows for interpretation and comparison of the strengths of effects of different variables included in the same model.

9.9.1. Results Concerning the Effect of Conflict Involvement on HIV-Prevalence

In summary, there is evidence that countries' own prior conflict or war involvement exerts a statistical significant and positive effect on HIV-prevalence rates. The coefficients of the dummy variables measuring conflict or war involvement are positive and highly significant at the 0.01 level. As expected, no or less than one year of conflict involvement correlates negatively and significantly with HIV-prevalence, whereas a medium or long duration of conflict involvement is associated with higher HIV-prevalence rates.

[241] Due to the rather big sample size of N=197 the adjusted R^2 values do not differ much from the R-squared values as given in the Stata output. Thus, I will only refer to the R-squared values in my interpretation of results.
[242] See Schnell et al. (1999: 416-418).

The number of all types of conflicts and the number of intermediate conflicts involved correlates significantly and positively with HIV-prevalence at the highest level of significance. Surprisingly, the same holds true for the indicator measuring the number of minor conflicts involved. The respective standardized beta-coefficient (in Model 9) seems even larger compared to the standardized beta-coefficients of the indicators measuring the more intense intermediate level of conflict involvement (in Model 8) or even war involvement (in Model 10). This rather strong effect on HIV-prevalence might be explained by the fact that low intensity conflict involvement is correlated with a medium duration of conflict involvement, which itself correlates positively with HIV-prevalence.

In summary, the expectation that the type of conflicts involved (wars, intermediate or minor conflicts) is important in regard to its impact on HIV-prevalence cannot be supported. Instead, involvement in all types of conflicts is significantly and positively correlated with HIV-prevalence.

Less significant results are found for the effect of war involvement of neighbors on HIV-prevalence in bordering countries. Contrary to expectations and prior findings by Ghobarah et al. (2003), the coefficients are negative. This does not support the major argument for a positive effect of neighboring war involvement on HIV-prevalence in bordering countries, which draws on international spillover effects, such as refugee flows. In addition, a comparison of the standardized beta-coefficients indicates that the effect of a country's own conflict involvement on HIV-prevalence at home is much stronger compared to the effect of neighboring war involvement on HIV-prevalence at home. For instance, increasing x31 (the proportion of neighbors involved in war) by one standard deviation would result in a decrease of the transformed dependent variable (HIV-prevalence at home) by only -0.13 standard deviations, whereas increasing a country's own number of conflicts involved by one standard deviation increases the transformed dependent variable by 0.43 standard deviations (see Model 6). Generally, any type of a country's own conflict involvement exerts a strong (and positive) effect on HIV-prevalence at home compared to a much weaker and negative effect of neighboring war involvement on HIV-prevalence in bordering nations.

These findings point to the importance of intra-state, conflict-related mechanisms, such as increasing mobility of soldiers and internally displaced people. Increases in the incidence of rape and prostitution might also contribute to high HIV-prevalence in conflict-torn countries.
The spread of HIV to neighboring countries via international, war-related effects, such as refugee-flows, might be less important or not even necessarily associated with intra-state war. Where prevalent, these mechanisms, which would theoretically increase HIV-prevalence in neighboring countries, might be offset by the isolating effect of neighboring war involvement. As mobility and trade between countries is reduced, the spread of the virus across borderlines is hindered. This agrees with Davis et al. (2003), who do not find a significant effect for their indicator measuring refugee flows on HIV-prevalence in receiving countries.

As already mentioned, using x17 (instead of x31) as the measure of neighboring war involvement tends to reduce R^2-values and the significance of the respective conflict coefficients. This also supports the afore mentioned isolating effect as both measures account for the number of neighboring countries and indicate whether there has been neighboring war involvement. However, the average number of wars neighboring countries have been involved in (x17) can assume very high values simply because one or few of the neighboring nations have been involved in several wars. In contrast, x31 (measuring the proportion of neighbors experiencing war) puts emphasis on the distribution of war involvement among neighboring countries. Taking this into account improves the predictability of the model. In the most extreme scenario, where a country is completely surrounded by neighbors, which have been involved in war between 1995 and 2002, HIV-prevalence levels are expected to be low. Isolation, interruption of trade and decreasing border-crossing mobility are again plausible explanations.
In addition, it has been argued that civil war in one country reduces economic growth rates of neighboring countries. Due to this, war involvement of neighbors indirectly affects HIV-prevalence in bordering countries through its direct impact on wealth in these countries. However, economic spillover effects are already captured by the variable measuring the level of wealth (x24), which might partly explain the relatively weak effect found for neighboring war involvement on HIV-prevalence in bordering nations.

The last four columns of Annex E give the regression results from the analyses modeling a non-linear relationship between the duration of conflict involvement and HIV-prevalence (Model 11), and also between the peace time since last conflict involvement and HIV-prevalence (Model 12).

As argued before, there is theoretical reason and support from the descriptive and bivariate analyses to expect inverse U-curve relationships. The correlation coefficients of the non-transformed conflict terms (β_1), which indicate the overall linear trend in the relationships, are therefore expected to be positive, whereas the coefficients of the squared conflict terms (β_2), which indicate the direction of curvature, are expected to be negative. However, results concerning the direction of effects are contrary to these expectations, which is probably due to the fact that the addition of the quadratic term changed the signs of the coefficients. This indicates severe multicollinearity between the polynomial and linear components and is a common problem in polynomial models.[243] Centered-score regression reduces the level of multicollinearity but only partly reverses the signs of the coefficients.

Therefore, and as far as the reversed signs of the beta-coefficients are due to the afore mentioned reason, there is preliminary support for inverse U-curve relationships. Compared to analyses not modeling the non-linear relationships, the models including the quadratic terms perform much better. The additional inclusion of the transformed conflict measures does not only result in substantively higher R^2-values of the models but the coefficients of the conflict variables become highly significant. In addition, effects are strong which also supports the assumed non-linear relationships.

However, there is no support for a non-linear relationship between intense conflict involvement and HIV-prevalence. Instead, intense conflict involvement (in terms of battle deaths and measured through the number of wars a country has been involved in) is associated with a steadily increasing HIV-prevalence (see Model 10). Adding a non-linear term does not result in an improvement of variance explained by the model. In addition, the squared term of the conflict involvement measure stays insignificant in the robust regression. Thus, it is rather extensive conflict involvement (in terms of duration) instead of intense conflict involvement (in terms of battle deaths), which exerts a significant, non-linear effect on HIV-prevalence. This is explained by the fact that particularly long-term conflict involvement is associated with isolating effects and a delaying impact on the spread of HIV-infection, which might not be the fact in cases of frequent and intense, but relatively short war involvement.

[243] See Hamilton (1992: 152, 153); or http://www.nd.edu/~rwilliam/stats2/l11.pdf, 28.1.2005; http://www.fordham.edu/economics/vinod/multiple-regression.doc, 28.1.2005.

9.9.2. Results Concerning the Effects of the Independent Variables

In regard to the remaining independent variables, the strongest effect and highly significant and positive coefficients are found for the lagged HIV-prevalence variable. This indicates strong temporal dependence in HIV-data.

The analyses also reveal a highly significant and strong effect for the youth bulge variable (x25). However, contrary to expectations, results indicate that the higher the proportion of males aged 15-49 years, the lower HIV-prevalence levels are. As already suggested, the negative sign might be explained by a problem of reverse causation. Although the existence of a large sexually active and highly at risk population increases societies' susceptibility to the disease, young adults are most likely to die of AIDS. Ainsworth et al. (1998) compare socio-economic characteristics of adults who died from AIDS and those who died from other causes in Kagera Region Tanzania. The authors find that on average those who died of AIDS were nearly 15 years younger.[244] Therefore, HIV/AIDS reduces the overall proportion of young adults, especially in heavily affected countries facing generalized epidemics.

As expected, the level of education (x28) correlates negatively and significantly with HIV-prevalence. However, the effect of education levels on HIV-prevalence is weaker compared to the effect of the conflict involvement variable or of other independent variables. Recent research delivers several possible explanations for this negative but rather weak effect.

For example, particularly during the first decade of the HIV/AIDS epidemic, HIV mostly affected the higher educated population.[245] This is because knowledge on the modes of transmission and prevention was still low among the higher educated while they were at a high risk for HIV-infection. Rising education levels and decreasing disparity in education is related to greater personal autonomy, spatial mobility/travel, urban residence and longer periods between first having sex and getting married.[246] This results in an accumulation of larger numbers of partners as compared to the less educated people. Rising education also associates with rising income, which increases the demand for commercial sex and/or the ability to support multiple partners.[247]

[244] See Ainsworth et al. (1998: 100).
[245] See Ainsworth et al. (1998: 101, 102); see also Shell et al. (2000).
[246] See Gregson et al. (2001).
[247] See Filmer (1998: 121).

However, with progressing epidemics, knowledge of health risk factors and prevention improved particularly among the higher educated population with easy access to information. Therefore, a shift in the pattern of infection to those people with lower socio-economic status can be identified.[248] Thus, I conclude that current patterns of infection are still likely to reflect the correlates of infections in the past, but, at the same time, already reflect new patterns of infection. This might explain the rather weak effect found. Along with this argument, Gregson et al. (2001) propose to control for the stage of the epidemic.

In addition, interacting effects e.g. of urban residence or type of occupation with education levels also need to be taken into account. The rather weak effect found for education on HIV-prevalence might also be due to the presence of other variables in the same specification (e.g. level of wealth or urbanization) which partly capture the effect of education on HIV/AIDS. Corresponding multivariate analyses by Filmer (1998) and Deheneffe et al. (1998) use individual-level survey data from African, Latin American, Caribbean and Asian countries.[249] Both studies examine the socio-economic correlates of high-risk sexual behavior and control for rural/urban residence of the household members interviewed. Findings indicate that the separate effect of education on the prevalence of commercial sex becomes insignificant when controlling for urban residence and type of occupation.[250] In addition, Deheneffe et al. (1998) also find that increasing educational attainment is not significantly correlated with higher condom use when urban residence in controlled for.

In summary, the relationship between education and HIV/AIDS might be more complex than the simple linear relationship assumed. In addition, interacting effects and control variables (e.g. the stage of the epidemic) have not been taken into account.

The strength of the effect of income inequality (x27) on HIV-prevalence is comparable to the strength of the effect of the education variable. Again, coefficients are highly significant at the 0.01 level even when mediating variables (level of wealth, education and urbanization) are controlled for. This supports a contextual effect of income inequality on HIV-prevalence.

[248] See Ainsworth et al. (1998: 96, 105), Mahal (2001: 45).
[249] Filmer (1998) relies on data from Kenya, Tanzania, Uganda, Zimbabwe, Burkina Faso, the Central African Republic, Cote d´Ivoire, Brazil (northeast region) and Haiti. The sample sizes range from 4225 household members in Tanzania to 8099 in Cote d´Ivoire (Filmer 1998: 111, 114). Deheneffe et al. (1998) use data from Burundi, the Central African Republic, Cote d´Ivoire, Tanzania, Kenya, Lesotho, Lusaka (Zambia), Manila (Philippines), Thailand, Rio de Janeiro (Brazil), Singapore and Sri Lanka. Sample sizes range from 1341 in Rio de Janeiro to 4166 in Tanzania (Deheneffe et al. 1998: 133).
[250] See Deheneffe et al (1998).

Results also support the Jaipur Paradigm, which expects higher levels of income inequality (as one dimension of the social cohesion concept) to be positively correlated with HIV-prevalence. In addition, the coefficients of the cultural variable measuring the proportion of Muslim or Jewish population are also highly significant and show the expected negative sign.

However, the effects of the remaining two independent variables (level of urbanization and health expenditures) are comparatively weak and less significant. Regression results for the urbanization measures indicate that the higher the pace of urbanization (x20) the lower HIV-prevalence rates are; whereas the alternative measure (x19, level of urbanization) would be significantly, but positively correlated with HIV-prevalence.[251] Thus, I conclude that higher levels of urbanization are generally correlated with higher levels of HIV-prevalence. However, where urbanization takes place, the pace of urbanization seems to make a difference. In particular, rapid urbanization might be associated with lower HIV-prevalence because it avoids long periods of short term rural-urban labor-migration.

Finally, findings indicate that high levels of per capita health expenditures are not significantly correlated with lower HIV-prevalence. This again is probably due to an endogenous relationship. Although high levels of health spending might be related to lower HIV-prevalence due to better prevention and controlling options, the level of HIV-prevalence vice versa affects the level of health spending. In addition, a time frame of eight years might not be long enough to observe successful reductions in HIV-prevalence following high levels of health spending. In addition, the validity of the often insignificant measure of health spending could be questioned as it does not give any information on the distribution of health expenditures (whether equal or unequal, for treatment or prevention).

[251] These results contradict prior research by Ghobarah et al. (2003), who find pace of urbanization to be positively correlated at least with loss in healthy life expectancy. The rate of urban population growth stays insignificant when included in an analysis by Over (1998), which examines the effects of societal variables on urban rates of HIV-infection in developing countries.

9.10. Regression Using Low-Estimates of HIV-Prevalence

Using low estimates of HIV-prevalence (y2) to measure the dependent variable tends to reduce R^2-values for all of the models. However, significance of the conflict variables only decreases in models including measures of the duration of conflict involvement (Model 4, 5, and Model 11). In the remaining nine out of twelve models, the coefficients of the conflict variables stay significant, usually at the 0.01 level.

9.11. Regression Analysis Excluding Cases with Missing HIV-Data

Using a smaller sample (N=151), which excludes those countries for which HIV-data are missing,[252] does not change the significant results found for the conflict variables. The strength of effects only slightly changes and R^2-values of all models increase.

However, it can be argued that countries with high conflict involvement are more likely to be among those cases for which HIV-prevalence levels are not even reported. This would result in a systematic underestimation of the effect and of HIV-prevalence in conflict-torn countries.

A look at the data reveals that HIV-data are missing for 46 cases, which often belong to the group of small island countries. Only seven of these missing cases have been involved in conflicts between 1995 and 2002. Two of the seven cases have experienced conflict involvement for a very short period, whereas Sierra Leone, Afghanistan and Turkey have respectively been involved in three, one or two different conflicts for the entire period of eight years. According to the hypothesis of this analysis, prolonged conflict situations (and a very high proportion of Muslim population) might have contributed to relatively low HIV-prevalence in these countries. Therefore, most of the seven excluded cases with missing HIV-data but conflict involvement are expected to face rather low HIV-prevalence levels. The only missing case with a theoretically high HIV-prevalence rate and potentially biased effect on results is Somalia, which has been involved in three different conflicts for a period of six out of eight years.

[252] 2003 HIV-data are missing for Afghanistan, Albania, American Samoa, Andorra, Antigua and Barbuda, Bhutan, Cape Verde, Cayman Islands, Macao, Comoros, Cyprus, Dominica, East Timor, Equatorial Guinea, Grenada, Guam, Guinea-Bissau, Kiribati, North Korea, Kuwait, Maldives, Marshall Islands, Mauritius, Micronesia, Monaco, Netherlands Antilles, New Caledonia, Northern Mariana Islands, Palau, Puerto Rico, Qatar, Samoa, San Marino, São Tomé and Principe, Saudi Arabia, Seychelles, Sierra Leone, Solomon Islands, Somalia, St. Kitts and Nevis, St. Lucia, St. Vincent and the Grenadines, Tonga, Turkey, United Arab Emirates and Vanuatu.

10. Measurement Error Hypothesis on Uncertainty in HIV-Data

Still, available national HIV-prevalence rates of conflict-torn countries might be systematically underestimated due to the breakdown of national disease surveillance systems. According to this Measurement Error Hypothesis, the uncertainty in HIV-estimates (measured through the length of distance between low and high estimates) might be correlated with the fact that countries have been involved in conflict. The magnitude of uncertainty in estimates should be systematically larger in countries with high conflict involvement compared to countries without conflict involvement. Thus, the following tests whether conflict involvement, war involvement or a medium duration of conflict involvement co-varies with the magnitude of uncertainty in estimates.

The corresponding test of co-variance reveals values of η^2 (ETA-squared) as given in Table 6.1. Values of this measure of co-variance, which ranges from zero to one, remain very small. For instance, the fact whether a country has been involved in any conflict or war between 1995 and 2002 explains less than 4% or 7% of the variance in the magnitude of uncertainty in estimates.

Table 6.1: Test of Co-Variance (η^2)[253]

CONFLICT MEASURE	DISTANCE BETWEEN LOW AND HIGH ESTIMATES OF HIV-PREVALENCE
Conflict involvement (dummy)	0.04 (η^2)
War involvement (dummy)	0.07 (η^2)
Medium duration of conflict involvement (dummy)	0.03 (η^2)

[253] For more information on the statistics see http://www.sowi.uni-mannheim.de/lehrstuehle/lessm/veranst/stat1/folien/folien_5.pdf, 3.1.2005; or http://www.politik.uni-mainz.de/kai.arzheimer/Lehre-Stata/Deskriptive-Dateien/frame.html#slide0001.html, 3.1.2005.

Similarly, the level of co-variance is explored between the magnitude of uncertainty in estimates and the duration of conflict involvement, the peace duration since last conflict involvement or the number of different types of conflicts involved. Again, results indicate that the different measures of conflict involvement do not systematically co-vary with the level of uncertainty in estimates. Values of Pearson's-r, which normally range from minus one to plus one, remain rather small (see Table 6.2).

Table 6.2: Test of Co-variance (Pearson's r)

CONFLICT MEASURE	DISTANCE BETWEEN LOW AND HIGH ESTIMATES OF HIV-PREVALENCE
Number of conflicts involved (x1)	0.29 (Pearson's-r)
Number of wars involved (x2)	0.32 (Pearson's-r)
Number of Intermediate conflicts involved (x3)	0.12 (Pearson's-r)
Number of minor conflicts involved (x4)	0.23 (Pearson's-r)
Duration of conflict involvement (x5)	0.18 (Pearson's-r)
Duration of conflict involvement, extended sample (x11)	0.16 (Pearson's-r)
Peace duration since last conflict involvement (x9)	-0.26 (Pearson's-r)
Peace duration, extended sample (x9)	-0.24 (Pearson's-r)

In summary, data available for this analysis do not support the Measurement Error Hypothesis. The magnitude of uncertainty in estimates does not appear to be systematically larger in countries with high conflict involvement and is, therefore, not correlated with the fact whether countries have been involved in conflict or not. Thus, the significant results found for the effect of conflict involvement on HIV-prevalence is unlikely to be subject to the afore mentioned systematic bias in HIV-data.

11. Robust Regression Analysis

Via Added-Variable-Plots and the calculation of Cook's D, multivariate outliers have been identified, which have a potential big influence on single regression coefficients or on the entire regression model.[254] The following robust regression analyses as a possible solution to the problem of multivariate outliers exclude multivariate outliers with a critical value of Cook's D >4/e(N).

11.1. Results from the Robust Regression Analyses

Results from the robust regression analyses indicate that the exclusion of the identified multivariate outliers does not result in a significant change in the values of regression coefficients. Instead, R^2-values drastically increase and the coefficients of the conflict involvement variables stay or become even more significant. This gives further support to the main hypothesis.

The robust model with the highest R^2-value of 0.75 is Model 2. Compared to predictions made by simply using mean values, the inclusion of the independent variables results in a sum of squared residuals which is almost four times lower. All independent variables in the model are highly significant at the .01 level and show the expected sign. Exceptions are the coefficients of the youth bulge variable and the variable measuring war involvement of neighboring countries, which are again both negative.

The exclusion of twelve outliers[255] substantively increases the coefficients of the conflict measures. Again, the conflict variable contributes the most to the predictive power of the model: excluding the dummy for a country's own war involvement from the analysis would reduce the R^2-values and predictive power of the model by as much as 11.41%, whereas an exclusion of x23 (proportion of Muslims or Jewish population) only results in a reduction of R^2 of about nine percent. Excluding x24 (health expenditures), x20 (pace of urbanization), x27 (income inequality), x28 (level of education), x25 (youth bulge) or x31 (neighboring war involvement) would reduce the R^2-values by only four to six per cent. Alone the lagged HIV-prevalence rates add more predictive power to the model than the conflict variable.

[254] See Kohler et al. (2001: 210-215).
[255] Robust Regression Model 2 excludes the following twelve multivariate outliers: Philippines, Pakistan, Somalia, Sri Lanka, Zimbabwe, Laos, Lesotho, Swaziland, Botswana, North Korea, the Maldives and Vanuatu.

11.2. Explaining Multivariate Outliers

Identified multivariate outliers in every of the 12 calculated robust regression analyses are Somalia, Laos, Zimbabwe, Lesotho, Botswana, Swaziland, the Maldives and Vanuatu.[256]

The outlier cases of Somalia and the Maldives are probably due to inappropriate imputation of regional averages for missing HIV-data. In addition, Garcia-Calleja et al. (2004) examine the quality of HIV sero-surveillance systems in 132 countries in 2002 as well as trends in quality between 1995 and 2002. According to this study, HIV sero-surveillance systems in Somalia, the Maldives and in a third outlier case (Laos) are poor or non-functioning. For the entire period, all three countries have been assigned very low or lowest values on the quality rating index, which captures the frequency and timeliness of data collection, the appropriateness of populations under surveillance, the consistency of the sites and groups measured over time and the coverage or representativeness of groups studied.

Contrary to the cases of Somalia, the Maldives and Laos, the remaining outliers cannot be explained through the poor quality of their sero-surveillance systems. Already since 1995, Lesotho, Zimbabwe, Botswana and Swaziland have high quality surveillance systems in place. All these countries face generalized epidemics with 2003 adult HIV-prevalence levels ranging from 24.6 (Zimbabwe) to 38.8 (Swaziland). These figures are even much higher than the Sub-Saharan African regional average of 7.5%. However, contrary to the main hypothesis of this analysis, these countries did not experience any kind of conflict involvement between 1995 and 2002 (Botswana and Swaziland) or relatively little or short conflict involvement (Lesotho and Zimbabwe).

The geographical closeness of these countries is probably their most obvious commonality. This points to spatial effects in the spread of HIV/AIDS as examined and supported by Davis et al. (2003). Therefore, a corresponding alternative explanation for these outlier cases stresses a high level of gender segregated, short-term, international labor-migration in the region.[257] This might not be fully captured by other variables included in the models.

[256] North Korea has been identified as a multivariate outlier in nine, the Philippines in seven, Pakistan in four and Sri Lanka in two out of the twelve robust regression analyses.
[257] See Whiteside et al. (2003: 12, 13, 32).

A number of articles deal with this issue and confirm that, particularly in African and Asian countries, profiles of HIV-infection are linked to regional patterns of international labor-migration.[258] Border-crossing migrants of South Africa have traditionally come from the outlier countries which are among South Africa's neighboring nations. Labor migrants from Lesotho, Mozambique, Malawi, Botswana, Zambia and Swaziland represent the majority of international mine workers in South Africa.[259] In Lesotho, 60% of the male labor force is usually absent from home in order to work in South Africa.[260]

The migrants' work involves mobility, in particular the obligation to travel regularly as single men for periods of up to a year and to live away from spouses. Often, housing and other facilities necessary for the development of a familial life, which leads to more stable sex relations, are not provided. The separation of familial and stable sexual relationships and the presence of commercial sex in mining towns lead miners to have sexual partners at the labor centers while continuing a long-distance relationship with their regular partners back in the rural areas. The frequency of sexual intercourse among migrant workers and their return visits back home intensifies the spread of HIV/AIDS. Therefore, short-term labor-migration offers the optimal context for HIV-transmission when men have money and are located in low-income communities where women's limited access to education, employment, credit or income can force them to resort to commercial or transactional sex.

"Although less is known about the miners' partners, there is some evidence that they also have other sexual partners while their husbands are away" (Gebrekristos et al. 2003: 11). The IOM (2003) warns that in Southern Africa, most migrants are men who move to urban areas, leaving their partners in rural areas. As those areas from which migrants originate are often underdeveloped and impoverished, women, in the absence of their partners, might have to sell or trade sex in order to survive. In addition, women of migrating men are left behind with less economic power and become even more dependent on their husbands' incomes. This enables them with less decision-making power in issues related to sexual relationships.[261]

[258] See Decosas (1998); Hope (2000); Beesey (2000); Duckett (2000); Sharma (2003); Gebrekristos et al. (2003); Buvé et al. (2002); Mabey (1997); Shell (2000).
[259] See Gebrekristos et al. (2003: 10, 11); Shell (2000).
[260] See Deheneffe et al. (1998: 137).
[261] See Jones (1998: 62-63).

In summary, the structure and particular characteristics of sexual networks, in which mobile populations participate, are central in understanding how mobility and migration facilitates the spread of HIV.[262] Other labor-migration-related factors, which encourage risky behavior are separation from socio-cultural norms that regulate behavior in stable communities, work in isolated environments with limited recreation and easy access to drugs and alcohol, episodic homelessness and sporadic periods of unemployment, limited access to health facilities, HIV/AIDS prevention and care programs, single sex and overcrowded hostels, dangerous working conditions, workplaces dominated by men, transactional sex, sexual abuse, sexual violence, a sense of anonymity which allows for more sexual freedom, xenophobia and discrimination and finally the lack of legal rights and legal protection.[263]

Based on the information given, it is not surprising that alarming levels of STDs and HIV have been found among female factory workers, female sex workers, seasonal workers in Swaziland and among the South African mining community.[264] The South African Presidential Aids Advisory Panel Report refers to a study comparing seventy couples where one partner was a migrant and fifty couples where both were non-migrants. Findings indicate that the level of HIV-discordance (where one person is positive and the other negative) is 30% in migrant couples and 12% in non-migrant couples.[265] Regression analysis by Over (1998) also confirms that countries with larger immigrant populations (measured as the percentage of the population that is foreign-born) have significantly larger HIV/AIDS epidemics.[266] Therefore, a higher degree of openness of the economy and of the borders to labor force migration might serve as an additional explanatory variable for outlier cases.

An alternative explanation for high HIV-prevalence in outlier countries might be political or structural factors not yet included in the analysis or not captured by other variables of the models. For example, high levels of HIV-prevalence in Zimbabwe have been brought into connection with long lasting processes of political instability, repression and ethnic tensions, which associates with high levels of social inequality.[267]

[262] See Myer et al (2003: 190).
[263] See Shell (2000), IOM: Mobile populations and HIV/AIDS in the Southern African region (2003).
[264] See Whiteside et al. (2003: 20, 33); Myer et al (2003: 190).
[265] See Government of South Africa: Presidential Aids Advisory Panel Report (2001: 44).
[266] See Over (1998: 48).
[267] See Decosas et al. (2002: i40).

Certainly, the income inequality variable included in the model does not fully capture this potentially important dimension of low social cohesion.[268] Political infighting, inappropriate political support or mismanagement might also have contributed to high HIV-prevalence in Zimbabwe.[269]

In addition, culturally determined sexual practices are not well captured by the included cultural variable (proportion of Muslim or Jewish population). For example, the patriarchal Shona and Ndebele societies in Zimbabwe practice polygamy and intimacy between married men and their wife's younger sisters, which contributes to the spread of the HIV-virus.[270] Similarly, Swaziland is a society where polygamy is legal and practiced; indeed the Royal Family has traditionally been one of the main proponents of this way of life. Also included among the cultural practices contributing to the spread of HIV are arranged marriages and widow inheritance.[271]

Gregson et al. (2002) stress sexual mixing patterns and provide empirical evidence for the fact that age differentials in sexual relationships contribute to extremely high HIV-prevalence levels in young women in Swaziland. Sexual behavior patterns are again rooted in culture and the expectation that women should marry earlier than men.[272] Sexual relationships between older men and younger women have also been identified as a crucial factor contributing to the HIV/AIDS epidemic in Botswana.[273] Sexual behavior of women might also help to explain the outlier case of Lesotho. Generally, the level of reported, non-marital sexual contact is much higher among men. However, survey data reveal that the female rate of reported, non-marital sexual contact is extraordinary high in Lesotho.[274] Deheneffe et al. (1998) also find that male rates of non-marital sexual contact as well as commercial sex rates for women and men in Lesotho are among the highest in their sample, which includes data from one South-American, seven Sub-Saharan African and four Asian countries.

[268] See Zierler et al. (1997) for a review of the epidemiology of HIV and AIDS among women in the U.S. in light of four conceptual frameworks linking HIV-infection and social inequality.
[269] See Ahmad (2001: 942); DeCastella (2003: 46).
[270] See Decosas et al. (2002: i41).
[271] See Whiteside et al. (2003: 26, 27).
[272] See Gregson et al. (2002); see also Brody et al. (2004).
[273] See Whiteside et al. (2003: 24).
[274] See Deheneffe et al. (1998: 133).

In addition, a high prevalence of bacterial vaginosis or viral STIs, due to weak immune systems undermined by hunger, also play a role in the rapid spread of the epidemic in the region.[275] In 2002, there were 14.4 million people at risk for starvation in six countries: Zimbabwe, Zambia, Lesotho, Swaziland, Malawi and Mozambique. Until 2000, the outlier cases are among the countries that faced marked declines in food production.[276]

At the same time health workers struggle to provide good quality care. In particular, high levels of brain drain in the medical sector as well as shortage in medical equipment become major constraints, as seen in Zimbabwe.[277]

In summary, identified multivariate outliers can partly be explained through measurement error in HIV-data. Additional variables not included in the models (such as international labor-migration, cultural determined sexual practices, a high prevalence of biological co-factors (STDs) or political factors) could also serve to better explain outlier cases such as Lesotho, Botswana, Swaziland and Zimbabwe.

12. Conclusion

Initial bivariate analysis demonstrates some statistical significant higher average HIV-prevalence rates in countries experiencing conflict and war as compared to countries without conflict or war experience. Additionally, a medium duration of conflict involvement is associated with higher average HIV-prevalence rates. Results from the multiple and robust regression analyses, in which factors known or expected to affect HIV-prevalence are controlled for, also clearly support the main hypothesis that countries' prior conflict involvement significantly correlates with higher HIV-prevalence in 2003. However, differentiation between the type of conflicts (wars, intermediate or minor conflicts) as well as the type of involvement (extensive or intense) reveals interesting insights.

[275] See Decosas et al. (2004: i43); DeCastella (2003: 47); Whiteside et al. (2003: 20-22, 31).
[276] See Whiteside et al. (2003: 31).
[277] See DeCastella (2003: 47).

Generally, involvement in all types of conflict is significantly and positively correlated with HIV-prevalence. The strong conflict involvement effect remains even when low estimates of HIV-prevalence are used. Results clearly indicate, that the higher the number of conflicts a country was involved in, the higher its national HIV-prevalence rates are. Mechanisms explaining this positive effect include increasing domestic mobility of soldiers and internally displaced populations, increasing incidences of rape and prostitution and the breakdown of health care systems.

In addition, a comparison of standardized beta-coefficients shows that countries' own conflict involvement exerts a much stronger effect on HIV-prevalence as compared to the effect of war involvement of neighboring countries on HIV-prevalence in bordering nations. The coefficient measuring the effect of neighboring war involvement on HIV-prevalence in bordering countries is statistical significant but negative. Thus, I conclude that war involvement of neighboring countries might be associated with outside isolation and reduced international mobility (although domestic mobility of the countries involved in war might increase). Spill-over effects, such as refugee flows, which could lead to an increase in HIV-prevalence in bordering countries, might be comparatively weak or offset by isolating mechanisms explaining the negative sign.

Additionally, there is preliminary support that the duration of conflict involvement is related to HIV-prevalence in a non-linear, inverse U-curve way, since the coefficient of the transformed key independent variable (x11) is statistical significant at a significance level of .01. This indicates that the more years a country spent in conflict between 1995 and 2002, the higher its HIV-prevalence rates are at the end of 2003. However, very extensive conflict involvement is associated with a decreasing HIV-prevalence.

This non-linear relationship does not hold true for intense conflict involvement in terms of battle deaths, which is measured through the number of wars a country has been involved in. Therefore, it is rather extensive conflict involvement (in terms of duration) instead of intense conflict involvement (in terms of battle deaths) which has a significant, non-linear effect on HIV-prevalence. In addition, the effect of the duration of conflict involvement on HIV-prevalence is rather strong. Due to this and prior mentioned outcomes, I also conclude that it is rather the duration of involvement instead of the type of conflicts involved which is important in regard to HIV-prevalence.

Finally, results also preliminary support the hypothesis that the peace duration since last conflict involvement is related to decreasing HIV-prevalence only in the long run, whereas in the short run peace and development after conflict involvement might be related to processes associated with increasing HIV.

13. Policy Implications

These results have important implications for AIDS forecasting and control in countries which have been involved in conflicts. In general, the significant effect found for conflict involvement on HIV-prevalence confirms the importance of key interventions in conflict and peacekeeping settings, such as caring for displaced populations or rebuilding devastated and over-burdened health care systems.

In addition, countries recovering from armed conflict need to integrate a response to AIDS in their recovery programs (e.g. in humanitarian assistance or demobilization programs). For example, there is urgent need to test outgoing but also incoming military personnel as well as to counsel and prepare HIV-positive soldiers for demobilization and a return to civilian life. Confidential or voluntary counseling and testing in armies and peacekeeping forces needs to be expanded, health care for soldiers needs to be improved, and secrecy, stigma and shame that cloak the epidemic particularly in the army need to be offset.

However, uniformed services can also be excellent agents of change and an important human resource to build on for advocacy and social mobilization efforts through HIV/AIDS prevention training.[278] They are generally perceived as role models, particularly for young people in their society. In addition, the military has strong traditions of organization and discipline, which gives them an "organizational" advantage in the fight against HIV/AIDS. Soldiers are used to learning new skills, following orders and taking initiative and can be a captive audience. Similarly, returning displaced populations can also become an important HIV-prevention resource if they are properly trained.

[278] See UNAIDS: Fact Sheet 3: HIV/AIDS and uniformed services (2002); UNAIDS/WHO: Report on the global AIDS epidemic (2004: 177).

Results from this analysis further indicate that HIV/AIDS treatment and prevention programs in conflict or post-conflict settings should focus on countries, which have been involved in a large number of different conflicts. Additionally, such programs should focus on countries with a medium duration of conflict involvement. In this context, particularly the impact of less intense forms of conflict involvement on HIV-prevalence should not be underestimated.

In regard to its effect on HIV-prevalence, it also seems to be of importance how development takes place. For example, increasing levels of urbanization are expected to be positively correlated with HIV-prevalence rates. However, the pace of urbanization seems to make a difference. Secondly, increasing income levels are negatively correlated with HIV-prevalence rates. However, the level of income inequality is also of importance. At the current state of generalized epidemics in most countries, rising education levels are also expected to be associated with decreasing HIV-prevalence - as long as this does not result in increasing income inequalities.[279]

In summary, it is important that structural interventions be developed with a view to the socio-economic and political determinants of HIV-risk, as well as the macro-impacts of AIDS epidemics. However, political scientists rarely study the linkage between politics and health and "(…) there is [also] massive disinterest amongst AIDS researchers in such macro-analyses of the epidemic" (Altmann 1999: 561). Others mention that the potential of structural interventions for HIV-prevention has received only limited attention to date although in practice, many individual-level interventions have only had limited impact at the population level.[280]

[279] See also Whiteside et al. (2003: 35, 36), who agree with the conclusion that there is not a simple causal relation between the epidemic and poverty/development. According to them, part of the answer may also lie in the type of economic growth (rapid or slow, equally or unequally distributed).
[280] See Myer et al. (2003: 191).

14. General Limitations of HIV-Data

One of the major constraints of this analysis is the lack of quality HIV/AIDS data. Although estimated figures are based on all available data (including surveys of pregnant women, population-based household surveys as well as other surveillance information),[281] surveillance systems vary in completeness and implementation among countries.[282] Thus, the quality of the data basis for the estimation of national prevalence rates as well as the estimation method itself can be criticized.

In countries facing generalized epidemics, estimates of adult HIV-prevalence rates are based on data from blood samples of pregnant women attending a selected number of sentinel antenatal clinics. However, the use of pregnant mothers attending antenatal clinics raises questions on the representativeness of this sentinel population to the general population or even to women in general. Sentinel surveillance in antenatal clinics has an inherent selection bias for sexual activity and against women using modern contraceptives. Women who have adopted safer sexual behaviors, such as consistent condom use, are unlikely to become pregnant and, therefore, not attend antenatal care. Infertile women are also unlikely to be captured by antenatal clinics data. Infertility caused by STIs or HIV would tend to result in a systematic underestimation of HIV-prevalence rates, particularly in countries with more mature epidemics or in the older age groups where infertility is most common.

Fortunately enough, validation studies have shown that HIV-data from antenatal clinics closely reflect HIV-prevalence in the reproductive age group.[283] This is due to the fact that even in badly affected HIV/AIDS areas of Sub-Sahara Africa, high proportions of women have access to antenatal services.[284] UNAIDS/WHO also adjusts data to reflect the ratio between the prevalence in antenatal clinics and that for women who are within the reproductive age in the general population. Adjustments also account for changes in fertility of women infected with HIV, differing rates of HIV-infection between women and men as well as the location of sentinel sites, which are mostly located in urban areas with lower HIV-prevalence.[285]

[281] See http://www.unaids.org/Unaids/EN/Resources/Epidemiology/, 25.1.2005; or Walker & Grassly & Garnett & Stanecki & Ghys (2004: 2183).
[282] See Garcia-Calleja et al. (2004).
[283] See Grassly et al. (2004: i33); Whiteside et al. (2003: 11); http://www.unaids.org/Unaids/EN/Resources/Epidemiology/, 25.1.2005.
[284] See Walker & Grassly & Garnett & Stanecki & Ghys (2004: 2181).
[285] See Walker & Grassly & Garnett & Stanecki & Ghys (2004: 2180-2182).

Therefore, data from antenatal clinics are accepted as providing a good measure of HIV-prevalence in the general adult population. Still, UNAIDS/WHO are aware of additional sources of error that are not yet accounted for e.g. in the calculation of the plausibility bounds. For instance, remaining sources of error result from laboratory testing or contraceptive use of women.[286]

In countries with low or concentrated epidemics, where transmission is assumed to occur mainly in high-risk groups (such as sex workers, their clients or injecting drug users), national HIV-estimates are based on estimates of the population size at high-risk and on surveillance data collected from these populations. Estimates for each group are then summed to obtain the estimated number of adults with HIV.[287] This method involves critical decisions, such as to decide how many groups at risk exist or how many geographically distinct epidemics and estimates will be made. In addition, estimates of the size of the populations most exposed to HIV/AIDS (e.g. clients of sex workers) are needed, which is one of the estimation procedure's main weaknesses.[288]

Only recently, it has become acceptable to include HIV-testing in population based surveys. Since then, large-scale household surveys became the new "gold-standard" in HIV-prevalence estimation.[289] However, household-based survey data on HIV-prevalence or (sexual) risk behavior face problems of systematic selection bias, sampling error, high refusal rates, recruitment bias and subject cooperation.

Loss of people from the study sample is a problem if there is reason to believe that these people are significantly different from the others included. Systematic bias can be introduced if refusal to be tested or asked tends to be related with higher HIV-prevalence.[290] For instance, among the hard to reach population might be those who deny their diagnosis or those who are already sick. Baumann (1986) notes that, particularly in research involving patient-subjects, variation in the physical and mental capacities of subjects to complete lengthy survey interviews might be a source of bias.

[286] See Walker & Grassly & Garnett & Stanecki & Ghys (2004: 2182-2183).
[287] See Walker & Grassly & Garnett & Stanecki & Ghys (2004: 2181); for more information on the software used to produce the final national HIV-prevalence estimates in countries with generalized, concentrated or low epidemics see Garnett et al. (2004); Walker & Stover & Stanecki & Zaniewski & Grassly & Garcia-Calleja & Ghys (2004); or http://www.unaids.org/Unaids/EN/Resources/Epidemiology/EPI_SoftwareTools.asp, 6.12.2004.
[288] See Walker & Stover & Stanecki & Zaniewski & Grassly & Garcia-Calleja & Ghys (2004).
[289] See Boerma et al. (2003).
[290] See Boerma et al. (2003: 1930).

"The more seriously ill the subject is, the less energy and ability he will have to provide adequate responses and the less complete and less accurate the data are likely to be" (Baumann 1986: 20). Surveys may also better reach rural populations with generally lower HIV-levels than urban populations.[291] Often, high-risk populations are systematically excluded. In particular, (migrant) men that are away (and, therefore, not captured in a household-based sero-survey) may be disproportionately more likely to have risky health behavior and to be HIV-positive than those who are found at home.[292]

Response validity of survey data on (sexual) risk behavior is threatened by a non-random response bias. The accuracy of information requested of a respondent depends in part on the degree to which the information is in possession. Often, people tend to forget a great deal of routine behavior. Sometimes people simply do not have important information, for instance, on the behavior and attitudes of sexual partners. If the material under study is distressing (e.g. questions on the experience of sexual violence), respondents may repress events. Generally, a respondent's motivation to provide accurate and complete information is influenced by the potential embarrassment or threat posed by the questions. In regard to HIV/AIDS, participants might fear that their homosexuality or the diagnosis of AIDS will become public in societies where HIV sero-positivity is surrounded by stigma and discrimination. In particular, retrospective questioning by medical personnel or doctors may be threatened by socially accepted response bias of sensitive information on sexual behavior. As respondents are tempted to present a favorable image of themselves to health care providers, they tend to over-report sexual behavior considered responsible (e.g. use of condoms) and under-report high-risk behavior.

Interviewer effects (gender effects) might also play a role as well as the sponsorship of a research study. As far as participation in surveys means immediate concrete benefit (such as information, counseling, free medical care or monetary payments), specific subpopulations might be over- or under represented in the sample. Random sampling would be an appropriate method to solve this problem.

[291] See UNAIDS/WHO: Report on the global AIDS epidemic (2004: 179).
[292] See UNAIDS/WHO Working Group on Global HIV/AIDS/STI Surveillance: Guidelines for second generation HIV surveillance (2000: 33).

In summary, survey data on sexual behavior are subject to a number of biases. Although steps are taken in the estimation process of national HIV-prevalence levels to counter these difficulties, residual bias will remain.

However, HIV-data are constantly improving. Contrary to some claims, UNAIDS refers to its HIV-estimates as "among the most reliable and accurate of all global health problem estimates."[293] Thus, current research (e.g. by Davis et al. (2003) or Ghobarah et al. (2003)) relies heavily on UNAIDS/WHO data, as it is the only data available for this kind of large-scale quantitative analysis.

Using data on AIDS-deaths instead of HIV-data would be even more problematic. It is uncertain how consistent AIDS is coded as the cause of death on the death certificate and not the specific opportunistic disease.[294] In addition, it must be considered whether or not all those who get AIDS are equally likely to come to the attention of health professionals. As far as data is based on reporting of AIDS cases, this means that data are biased by differential access to medical care.[295] Paul Scherrer (2000) refers to the 2000 WHO World Health Report which explains that AIDS death rate figures based on household survey data are for the most part underestimates, because many households which have been hardest hit by AIDS have been completely wiped out and are, consequently, not counted. Finally, the long latent incubation period means that the disease (AIDS) is a reflection of historic, rather than current spread. In contrast, the HIV-virus is immediately detectable. Typically, HIV-tests yield positive results already three to four weeks after infection, as soon as a person "sero-converted" and started to produce antibodies to the virus.[296] Thus, AIDS-data will not provide information about recent trends in new infections. In addition, research using AIDS-data would need to account for appropriate lag times between HIV-infection, outbreak of AIDS, and eventually, death. Therefore, relying on HIV-prevalence data reduces some difficulties in choosing the proper lag time.[297]

[293] See http://www.unaids.org/en/default.asp, 12.3.2004.
[294] See Flam et al. (1986: 67).
[295] See Flam et al. (1986: 67).
[296] See Johnson (2003).
[297] See Johnson (2003).

15. Limitations of the Analysis

One major limitation of this analysis is that HIV-prevalence levels prior to conflict involvement are not controlled for. Often, conflict takes place in countries where rates of HIV-infection are already high. In other cases (Sierra Leone[298] or Bosnia and Herzegovina[299]), pre-war HIV-prevalence has been very low and prolonged conflict might have contributed to relatively low post-war HIV-prevalence. However, if HIV is not already prevalent in a country in conflict, the virus cannot take advantage of conditions conducive to its spread. Although the inclusion of a control variable measuring the "stage of the epidemic" could serve to overcome the problem,[300] data on HIV-prevalence rates prior and after conflict involvement would be desirable. However, available successive UNAIDS/WHO estimates are not comparable between different years due to changes in the modeling methodology.[301] Therefore, the design of this analysis does not give any information on conflict related increases in HIV-prevalence. Anyways, comparing HIV-prevalence rates over time is problematic in the context of this study, as HIV-prevalence rates are calculated by defining the number of HIV-cases within a stable population. However, when the population is continuously shifting because of conflict, it is difficult to determine whether changes in rates are due to changes in risk or to fluctuating denominators.

In this context, another concern is measurement error in HIV-data, which is systematically related to conflict. However, a Measurement Error Hypothesis, which states that the uncertainty in HIV-estimates (measured through the distance between low and high estimates) is correlated with the fact whether countries have been involved in conflict, has been tested and could not be supported.

In summary, the baseline model of this study needs to be specified through the inclusion of additional control and independent variables (e.g. the stage of the epidemic, the presence of co-factors of transmission or the level of border-crossing labor-migration). This could help to explain outlier cases with high prevalence rates but low conflict involvement.

[298] See Spiegel (2004: 325).
[299] See UNAIDS/WHO: Report on the global AIDS epidemic (2004: 178, 179).
[300] Over (1998) and Bonnel (2000) use the age of the epidemic (the number of years since the first case of HIV-infection or AIDS was reported) as a measure of the stage of the epidemic. The corresponding coefficients are statistical significant.
[301] See http://www.unaids.org/Unaids/EN/Resources/Epidemiology/, 25.1.2005.

In addition, interacting effects (e.g. between education levels and the stage of the epidemic) need to be taken into account. Direct measurement of relevant variables, such as mobility of soldiers, rape, prostitution or refugee flows would also improve the analysis. However, this would significantly reduce the sample size due to missing values and difficulties in measurement.

Finally, analysis at the macro-level might be too large-scale. National averages in HIV-prevalence obscure the regional effects of the pandemic as well as differences in HIV-prevalence among specific risk populations. More systematic sub-group analysis is needed (e.g. in regards to the long- and short-term effects of conflict and peace on internally displaced populations, refugees, women or prostitutes). Health and nutritional conditions of internally displaced populations or residents of conflict zones are also rarely studied, although they constitute a considerable proportion of conflict affected populations.

16. Comment

Last but not least, the complexity of the relationship between conflict and HIV/AIDS needs to be mentioned, because HIV/AIDS might also have a reverse-causal effect on social and economic variables decisive for the outbreak of conflict. This agrees with prior research by Ted Robert Gurr and his colleagues from the State Failure Task Force Project, which identifies health indicators as factors related to state failure and political instability.[302]

The question, how the explosion of HIV/AIDS contributes to instability and increases the risk for conflict, needs to be examined in the coming years as the epidemic unfolds and the necessary AIDS-death data become available. Some authors, however, have already begun discussing the question how HIV/AIDS might threaten internal and external security of severely affected societies. For instance, AIDS related loss of military personnel negatively affects continuity at the command level and within the ranks. It also increases costs for recruitment and training of young and often inexperienced successors. Overall military preparedness and stability is reduced, particularly in military administrations.[303]

[302] See Goldstone et al (2000: 14).

[303] See UNAIDS: AIDS and the military (1998); similarly to the loss of employees within the military forces, HIV/AIDS has the potential to wipe out the next generation of political and economic leaders, teachers, lawyers and students, who constitute the segment of population often most severely affected by AIDS, namely the educated elite (see Fredland 1998: 554). For further discussion of the impact of HIV/AIDS on democratic governance,

In already weak countries another threat to internal stability is rising crime rates associated with the increasing number of AIDS orphans. By 2010 approximately 40 million children worldwide will be orphans because of the HIV/AIDS epidemic. Most of these orphans will, in a decade's time, be between the ages of 15-24 years, which is where people's propensity to commit crime is at its highest. Orphans are particularly prone to resort to crime and pose a risk on internal stability as they grow up in impoverished conditions, badly supervised by relatives and welfare organizations. They lack guidance, education, health care and immunization while facing malnutrition, challenges of disintegration, prejudice and social exclusion.[304]

Their increased (poverty related) demand for labor becomes an additional factor that exposes orphans to greater risks of HIV-infection. Examples of `hazardous labor´, which directly link HIV-orphans to conflict, would be prostitution or the recruitment of child soldiers who are arbitrarily seized from the streets and orphanages.[305]

Another field of research deals with the destabilizing economic impact of AIDS, which can also be linked to increasing risk for conflict. As mortality rates rise, particularly among the skilled, the composition of the labor force is expected to change.[306] The World Bank predicts that the usual post-epidemic effect of higher wages (due to the shrinking labor force) will not emerge following an AIDS epidemic, because this effect will be replaced by costs of treatment and lost production.[307] Already some years ago, the costs of treating HIV-infected people exceeded the per capita GNP in a number of countries, such as Kenya, Malaysia, Rwanda, Tanzania and Zimbabwe.[308] Employers face increased absenteeism, increased training costs, sickness payments and higher pension demands.[309] The overall increase in production costs is a direct result of workers falling sick and dying. For example, Fourie et al. (2001) predict that South Africa's important mining sector will suffer, because by 2015 almost eight mine workers will be dead or dying due to AIDS-related diseases as compared with one `normal´ death.[310]

---

internal stability, social fragmentation, political polarization and the risk for ethnic violence see USIP: Special Report: AIDS and violent conflict in Africa (2001: 6, 7); Fourie et al. (2001: 12); Guha-Sapir et al. (2002: 24-36); Manning (2002); USIP: Plague upon plague: AIDS and violent conflict in Africa (2001).

[304] See Schönteich (1999: 1, 3); Fourie et al. (2001: 14, 5).

[305] See Pyne - Mercier (2001); Machel (2000).

[306] See Panos Institute: The hidden cost of AIDS: the challenge of HIV to development (1992: 68-88).

[307] See BBC News: AIDS `economic catastrophe´ looms (July 26th of 2003).

[308] See Brown (1997); Altmann (1999: 572, 573); Bonnel (2000: 7, 8).

[309] See Fourie et al. (2001: 9).

[310] See Fourie et al. (2001: 9); see Bollinger (2002) discussing the impact of HIV/AIDS on trade and commerce.

The causal relationship by which disease affects the economy not just works through sick labor, but also through lost capital and purchasing power, due to decreasing income at the household level.[311] Coping behaviors in response to illness and death of primary earners often include labor substitution, the mobilization of cash reserves or savings and the sale of livestock.[312] Along with high medical and funeral costs,[313] the result is low levels of saving and investment, which in return affects production levels and increases poverty.[314] In addition, foreign direct investment is also greatly affected by HIV/AIDS epidemics as investors' willingness to make any long-term investment diminishes.[315]

As a result, GDP growth rates are estimated to shrink by as much as one to 2.6 percentage points annually in countries with HIV-prevalence rates over 20%. Heavily affected countries are predicted to lose more than 20% of GDP by 2020.[316] A recently published study by Ouattara (2004) calculates an endogenous growth model including HIV-infection as a negative shock to economic growth. Results confirm prior findings and indicate that increases of HIV-infection rates negatively affect the growth rates of economies. Furthermore, the author even finds that a high and sustainable economic growth rate is incompatible with a high incidence of HIV-infection in the economically active population. This is particularly true if countries lack a health policy, which effectively stabilizes the HIV-infection rate.[317]

Another consequence of declining individual productivity and decreasing income levels is a drop in governmental tax revenue. At the same time, though, government resources need to be shifted from productive investment to health sector expenditures and health care. Therefore, AIDS also lowers the returns to important public investment in agriculture, research, health (to tackle other diseases), clean water, sanitation and education, which negatively affects economic development.

[311] For a summary of research on the (regional) micro- and macro-economic impact of the HIV/AIDS epidemic see Mills et al. (2004). For alternative perspectives see Evans (2004) and van der Gaag (2004).
[312] See Mills et al. (2004: 68).
[313] See BBC News: AIDS `economic catastrophe´ looms (July 26th of 2003).
[314] See Bonnel (2000: 5) for a quantitative analysis of the effect of HIV/AIDS on domestic saving.
[315] See Fourie et al. (2001: 8, 9); or Guha-Sapir et al. (2002: 31).
[316] See Fourie et al (2001); Bonnel (2000: 6).
[317] See Ouattara (2004).

In summary, at least two of the afore mentioned economic variables (income and GDP growth), which are severely affected by the HIV/AIDS epidemic, have been identified as leading to the outbreak of conflict.[318] Thus, HIV/AIDS can be viewed as an indirect cause of conflict due to its direct impact on economic variables decisive for the outbreak of conflict.

Although research started to explore the political consequences of HIV/AIDS, the debate consists, at least at the moment, merely of hypotheses. Theoretical clarification is required in order to better understand how political processes interact with public welfare issues at community and individual levels. In addition, quantitative data to permit discussions on causal relationships and to substitute theories are, to a large extent, not available. Nevertheless, debating the links between HIV/AIDS and political instability is important as this also helps to answer the question in how far the delivery of health services might play a role in reducing conflict and maintaining peace. At least a preliminary analysis by MacQueen et al. (2000) suggests that health initiatives have indeed been successfully used as peace initiatives.[319]

[318] See Collier et al. (2000); The Economist: special report civil wars: the global menace of local strife (May 24th of 2003).
[319] See MacQueen et al. (2000: 293).

This study concludes with the sad fact that in 2002 alone HIV/AIDS killed approximately sixteen times more people worldwide than war.[320] Two years earlier, when the U.N. Security Council first discussed the security implications of HIV/AIDS, the number of African AIDS-deaths per year had outstripped the number of battle deaths in all civil wars fought during the 1990s.[321] By now, HIV/AIDS has become the leading killer in Sub-Saharan Africa.[322]

Until 1999, eleven million Sub-Saharan African people had died due to HIV/AIDS - as many Africans as were transported across the Atlantic for slave trade. It is predicted that by 2010 AIDS will have claimed more lives than all the major world pandemics combined.[323] Shell (2000) continues that unless checked by 2050, one may add to that all the people killed in the Second World War.[324]

At the moment, approximately 39.4 million people around the world are living with HIV, and about another 15.000 get infected every week. An estimated 4.9 million people acquired HIV in 2004 alone, while UNAIDS attributes 3.1 million deaths to AIDS during this year. Sub-Saharan Africa remains the most affected region with 64% of people living with HIV even though only about 10% of the world's population lives there.[325]

In addition, the number of ongoing civil wars has steadily increased.[326] Based on the main result of this preliminary analysis, this could be devastating in regard to HIV-prevalence levels in developing countries, which additionally face problems of increasing levels of urbanization and income inequality as well as low levels of education and wealth.

Clearly these figures indicate that the "war on AIDS" will be costly, but perhaps at an extra US$ 7-10 billion a year,[327] the costs would be not much more than 1% of the world's steadily increasing annual military spending of approximately $956 billion.[328]

---

[320] See WHO: World Health Report (2004: 120, 124).
[321] See U.N.: A more secure world: our shared responsibility (2004: 25).
[322] See Fourie et al. (2001: 2).
[323] This includes the Spanish Influenza Epidemic of 1918/1919, the Smallpox devastation of the New World in the 15th and 16th century and the Black Death in Europe in the 14th century (see Shell 2000).
[324] See Shell (2000: 10).
[325] See UNAIDS/WHO: AIDS Epidemic Update-December 2004 (2004).
[326] See Fearon et al. (2003: 77, 78).
[327] See Mills et al. (2004) for a cost benefit analysis of HIV/AIDS interventions as well as a discussion on measurement problems; see also U.N.: A more secure world: our shared responsibility (2004: 28).
[328] 2003 data (in current dollars) as given in the SIPRI Yearbook (2004: Chapter 10).

Annex A:

Summary Statistics, (all variables)

Variable	N	Mean	SD	Min	Max
Dependent Variable: HIV/AIDS					
y1 : Adult HIV-prevalence	197	2.54	5.56	.1	38.8
y2 : Adult HIV-prevalence (low estimates)	197	1.90	5.02	.01	37.2
y3 : Adult HIV-prevalence (high estimates)	197	3.65	6.44	.2	40.4
Key Independent Variable: Conflict Involvement					
x1 : No. of armed conflicts involved	197	.82	1.86	0	13
x10: " (extended sample)	197	1.07	2.40	0	15
x2 : No. of wars involved	197	.28	.89	0	7
x3 : No. of intermediate armed conflicts involved	197	.30	.77	0	5
x4: No. of minor armed conflicts involved	197	.24	.70	0	6
x5: Years spent in conflict	197	1.26	2.46	0	8
x11: " (extended sample)	197	1.48	2.64	0	8
x9 : Peace duration since last conflict (in years)	197	6.23	3.04	0	8
x12: " (extended sample)	197	6.03	3.15	0	8
x31: Proportion of neighbors involved in war	197	.15	.22	0	1
x17: Average no. of wars, neighbors	197	.29	.54	0	2.67
confinvol: Dummy: conflict involvement	197	.29	.46	0	1
gconfinvol: " (extended sample)	197	.32	.47	0	1
lyears: Dummy: less than one year in conflict	197	.71	.46	0	1
glyears: " (extended sample)	197	.68	.47	0	1
myears: Dummy: 1-5 years in conflict	197	.17	.38	0	1
gmyears: " (extended sample)	197	.17	.38	0	1
hyears: Dummy: 6-8 years in conflict	197	.12	.33	0	1
ghyears: " (extended sample)	197	.15	.36	0	1
warinvol: Dummy: war involvement	197	.15	.36	0	1
nwarhigh: Dummy: high war involvement of neighbors	197	.12	.32	0	1
Independent Variables / Control Variables					
x18: Populations size (millions) / analytical weight	197	31.95	123.80	.02	1304.2
x19: Level of urbanization (% of urban pop.)	197	56.01	24.04	7.6	100
x20: Pace of urbanization (rate of change of urban pop.)	197	2.44	2.21	-3.63	18.36
x21: Population density (people per km²)	197	279.63	1379.56	2	16842
x22: Income (per capita GNI, PPP, thousand int.$)	197	8.83	9.58	.5	53.29
x23: Culture (proportion of Muslim or Jewish pop.)	197	.24	.37	0	1
x24: Total health expenditure (per capita, int.$)	197	540.90	723.09	12	4318
x25: Youth bulge (proportion of males age 15-49)	197	.27	.03	.21	.4
x27: Income inequality (gini index)	197	40.15	8.63	24.4	70.7
x28: Level of education (adult literacy rates)	197	81.51	19.53	12.8	100
x29: Disparities in education (literacy women/men)	197	.93	.13	.42	1.2
x33: Lagged HIV- prevalence (end of 2001)	197	2.82	6.03	.01	38.8

Annex B:

Measurement and Data Sources (all variables)

VARIABLE	MEASURE	SOURCE
HIV-Prevalence Measures / Dependent Variable		
`Adult HIV-prevalence rates´	►Adult HIV-prevalence rates (% of the whole adult population (15-49 years old) estimated to be HIV positive in the end of 2003, (y1)	UNAIDS/ WHO, 2004 Report on the Global AIDS Epidemic
	►Low estimates of HIV-prevalence rates, end of 2003, (y2)	
	►High estimates of HIV-prevalence rates, end of 2003, (y3)	
Conflict Involvement Measures / Key Independent Variable		
`Conflict involvement´	►Total number of all types of conflicts (wars, intermediate and minor conflicts) a country was involved in between 1995 and 2002, (x1)	Gleditsch et al. 2004 (2.1. version of the dataset on armed conflicts 1946–2001, dataset on unclear cases of armed conflicts 1946-2001, and dataset on armed conflicts active in 2002)
`Conflict involvement, extended sample´	►Total number of all types of conflicts a country was involved in between 1995 and 2002, including unclear cases of conflict involvement, (x10)	
`War involvement´	►Total number of wars a country was involved in between 1995 and 2002, (x2)	
`Involvement in intermediate armed conflicts´	►Total number of intermediate armed conflicts a country was involved in between 1995 and 2002, (x3)	
`Involvement in minor armed conflicts´	►Total number of minor armed conflicts a country was involved in between 1995 and 2002, (x4)	
`Years spent in conflict (duration of conflict involvement)´	►Total number of years a country spent in conflict between 1995 and 2002 (each country's involvement in at least one conflict (minor armed conflict, intermediate armed conflict or war) in each year was coded as a one, non-involvement as a zero), (x5)	
`Years spent in conflict, extended sample´	►Total number of years a country spent in conflict between 1995 and 2002, including unclear cases of conflict involvement, (x11)	
`Peace duration´	►Total number of years since last conflict involvement (whether this has been a war, intermediate or minor conflict), (x9)	
`Peace duration, extended sample´	►Total number of years since last conflict involvement, including unclear cases of conflict involvement, (x12)	
`Average number of wars neighboring countries have been involved in´	►Total number of wars neighboring countries have been involved in between 1995 and 2002 divided through the number of neighbors (x17)	
`Percentage of neighboring countries experiencing war´	►Total number of neighboring countries experiencing war between 1995 and 2002 divided through the number of neighbors (x13)	

VARIABLE	MEASURE	SOURCE
Measurement of Control Variables		
`Population Size´ (analytical weight)	►Total population of urban and rural areas in millions at mid -2003, (x18)	UNPD, World Urbanization Prospects, 2003 Revision
`Urbanization´	►Total percentage of urban population in 2003, (x19)	UNPD, World Urbanization Prospects, 2003 Revision
	►Average annual rate of change (%) of the urban population from 1995 to 2000, (x20)	
`Population Density´	►Total number of people per sq. km in 2002, (x21)	World Bank, 2004 WDI
`Culture´	► Proportion of Muslim or Jewish population in 2003 (x23)	CIA, 2003 World Factbook
`Economic development and wealth´	►2002 gross national per capita income (in thousands) converted to international dollars using purchasing power parity rates, (x22)	World Bank, 2004 WDI
	►Average per capita total expenditures on health at international dollar rate from 1997-2001, (x24)	WHO, 2004 World Health Report
`Youth Bulge´	► Proportion of males age 15-49 in the total population in 2003, (x25)	CIA, 2003 World Factbook
`Income Inequality´	►Gini index of income distribution, (x27) (survey years range from 1992-2002).	UNDP, 2004 Human Development Report
`Education´	►Adult literacy rates (% of all 15 years old and above) in 2002 (level of education), (x28)	UNDP, 2004 Human Development Report, HDI
	►Average ratio of literate women to men, 15-24 year old, 1995-2003, (disparity in education), (x29)	U.N. Statistics Division, see http://millenniumindicators.un.org
`lagged HIV-prevalence rates´	► Adult HIV-prevalence, end of 2001, (x33)	UNAIDS/ WHO, 2002 Report on the Global AIDS Epidemic

Annex C:

Correlation Matrix (all variables)

	y1	y1log	y1box	y1afinal	y2	y3	x1
y1	1.0000						
y1log	0.7285*	1.0000					
y1box	0.6271*	0.9861*	1.0000				
y1afinal	0.6159*	0.9797*	0.9990*	1.0000			
y2	0.9908*	0.6602*	0.5560*	0.5462*	1.0000		
y3	0.9745*	0.8103*	0.7190*	0.7060*	0.9370*	1.0000	
x1	0.1160	0.2529*	0.2511*	0.2448*	0.0778	0.1712*	1.0000
transx1	0.0675	0.2002*	0.2053*	0.2013*	0.0347	0.1195	0.8982*
x2	0.1758*	0.2672*	0.2563*	0.2507*	0.1381	0.2298*	0.8151*
transx2	0.1451*	0.2266*	0.2170*	0.2119*	0.1107	0.1972*	0.7138*
x3	0.0195	0.1424*	0.1476*	0.1427*	0.0015	0.0490	0.7959*
transx3	0.0031	0.1123	0.1237	0.1215	-0.0094	0.0247	0.6836*
x4	0.0634	0.1767*	0.1799*	0.1759*	0.0295	0.1092	0.7510*
transx4	0.0336	0.1341	0.1410*	0.1388	0.0111	0.0627	0.6242*
x5	0.0592	0.1594*	0.1566*	0.1503*	0.0355	0.0977	0.7907*
transx5	0.0261	0.1261	0.1269	0.1214	0.0082	0.0584	0.7489*
x9	-0.1247	-0.2026*	-0.1892*	-0.1815*	-0.0916	-0.1718*	-0.7614*
transx9	-0.1302	-0.2002*	-0.1853*	-0.1773*	-0.1003	-0.1719*	-0.7344*
x10	0.0729	0.2043*	0.2077*	0.2029*	0.0382	0.1241	0.9593*
transx10	0.0342	0.1646*	0.1759*	0.1736*	0.0070	0.0767	0.8650*
x11	0.0318	0.1078	0.1021	0.0952	0.0102	0.0673	0.7548*
transx11	0.0026	0.0785	0.0755	0.0691	-0.0137	0.0321	0.7320*
x12	-0.1003	-0.1632*	-0.1478*	-0.1392	-0.0687	-0.1459*	-0.7274*
transx12	-0.1052	-0.1632*	-0.1468*	-0.1382	-0.0762	-0.1467*	-0.6914*
confinvol	0.1158	0.1746*	0.1586*	0.1503*	0.0907	0.1483*	0.6813*
lyears	-0.1158	-0.1746*	-0.1586*	-0.1503*	-0.0907	-0.1483*	-0.6813*
myears	0.1506*	0.1228	0.0977	0.0915	0.1307	0.1693*	0.2185*
hyears	-0.0127	0.1014	0.1082	0.1037	-0.0246	0.0111	0.6968*
warinvol	0.1078	0.2188*	0.2167*	0.2117*	0.0766	0.1611*	0.7112*
glyears	-0.0901	-0.1335	-0.1163	-0.1078	-0.0660	-0.1228	-0.6344*
gmyears	0.1426*	0.1096	0.0847	0.0788	0.1250	0.1580*	0.1390
ghyears	-0.0325	0.0587	0.0625	0.0577	-0.0454	-0.0061	0.6807*
x17	0.3387*	0.3402*	0.3000*	0.2896*	0.2917*	0.3951*	0.4359*
x31	0.1779*	0.2023*	0.1771*	0.1684*	0.1355	0.2355*	0.3800*
x18	-0.0638	-0.0740	-0.0700	-0.0705	-0.0577	-0.0742	0.2474*
x19	-0.2952*	-0.3958*	-0.3756*	-0.3655*	-0.2683*	-0.3262*	-0.3384*
x20	0.1974*	0.3854*	0.3823*	0.3766*	0.1541*	0.2560*	0.3516*
x21	-0.0607	-0.0791	-0.0704	-0.0655	-0.0513	-0.0730	-0.0567
x22	-0.2195*	-0.3748*	-0.3632*	-0.3523*	-0.1812*	-0.2716*	-0.2448*
transx22	-0.2560*	-0.4652*	-0.4486*	-0.4353*	-0.2014*	-0.3280*	-0.3361*
x24	-0.2076*	-0.3265*	-0.3106*	-0.2999*	-0.1749*	-0.2541*	-0.2143*
transx24	-0.2439*	-0.4465*	-0.4295*	-0.4159*	-0.1948*	-0.3121*	-0.3905*
x27	0.5287*	0.5264*	0.5107*	0.5135*	0.5063*	0.5410*	0.0841
x28	-0.1916*	-0.4141*	-0.4171*	-0.4105*	-0.1433*	-0.2640*	-0.3585*
x29	0.0184	-0.1404*	-0.1419*	-0.1341	0.0524	-0.0347	-0.2319*
x23	-0.1043	-0.1439*	-0.1594*	-0.1660*	-0.0982	-0.1021	0.0892
x25	-0.2241*	-0.2897*	-0.2657*	-0.2551*	-0.1945*	-0.2579*	-0.2431*

	transx1	x2	transx2	x3	transx3	x4	transx4
transx1	1.0000						
x2	0.7627*	1.0000					
transx2	0.7998*	0.9057*	1.0000				
x3	0.6391*	0.4518*	0.3680*	1.0000			
transx3	0.5994*	0.3225*	0.2817*	0.9063*	1.0000		
x4	0.7187*	0.4003*	0.3427*	0.4464*	0.4152*	1.0000	
transx4	0.6862*	0.3087*	0.2426*	0.3560*	0.3252*	0.8784*	1.0000
x5	0.5491*	0.6530*	0.4481*	0.7504*	0.5897*	0.4501*	0.3212*
transx5	0.5627*	0.6350*	0.4529*	0.7272*	0.6064*	0.3871*	0.3058*
x9	-0.4748*	-0.5980*	-0.3925*	-0.6923*	-0.5099*	-0.5060*	-0.3133*
transx9	-0.4370*	-0.5604*	-0.3618*	-0.6648*	-0.4764*	-0.5120*	-0.3000*
x10	0.8609*	0.6964*	0.6027*	0.8025*	0.7432*	0.7865*	0.6705*
transx10	0.9439*	0.6045*	0.6133*	0.6900*	0.7274*	0.7761*	0.7661*
x11	0.5060*	0.5959*	0.4058*	0.7227*	0.5507*	0.4575*	0.3155*
transx11	0.5269*	0.5868*	0.4145*	0.7285*	0.5820*	0.4021*	0.3029*
x12	-0.4470*	-0.5612*	-0.3679*	-0.6649*	-0.4818*	-0.4923*	-0.3002*
transx12	-0.4066*	-0.5197*	-0.3353*	-0.6270*	-0.4442*	-0.4909*	-0.2846*
confinvol	0.3780*	0.4862*	0.3083*	0.6060*	0.4160*	0.5297*	0.2838*
lyears	-0.3780*	-0.4862*	-0.3083*	-0.6060*	-0.4160*	-0.5297*	-0.2838*
myears	-0.0016	0.0681	0.0138	0.1548*	0.0216	0.3253*	0.0840
hyears	0.5286*	0.5989*	0.4136*	0.6655*	0.5547*	0.3622*	0.2984*
warinvol	0.4948*	0.7411*	0.4699*	0.5358*	0.3473*	0.3615*	0.2800*
glyears	-0.3520*	-0.4528*	-0.2871*	-0.5643*	-0.3874*	-0.4932*	-0.2643*
gmyears	-0.0295	0.0530	0.0107	0.0670	-0.0158	0.2290*	0.0432
ghyears	0.4898*	0.5344*	0.3629*	0.6650*	0.5216*	0.4020*	0.2990*
x17	0.3643*	0.5525*	0.5082*	0.2082*	0.1479*	0.2282*	0.1175
x31	0.3119*	0.3655*	0.3224*	0.1916*	0.1503*	0.3360*	0.2106*
x18	0.2586*	0.0975	0.0231	0.2668*	0.3009*	0.2418*	0.2396*
x19	-0.2418*	-0.2983*	-0.2179*	-0.2144*	-0.1815*	-0.2858*	-0.1998*
x20	0.2804*	0.3622*	0.3316*	0.2451*	0.1720*	0.2057*	0.1238
x21	-0.0287	-0.0390	-0.0245	-0.0486	-0.0308	-0.0481	-0.0242
x22	-0.1586*	-0.1811*	-0.1301	-0.1702*	-0.1342	-0.2343*	-0.1343
transx22	-0.2215*	-0.2608*	-0.1862*	-0.2194*	-0.1538*	-0.3220*	-0.2090*
x24	-0.1454*	-0.1486*	-0.1139	-0.1506*	-0.1357	-0.2161*	-0.1220
transx24	-0.2689*	-0.2730*	-0.1953*	-0.3011*	-0.2633*	-0.3616*	-0.2474*
x27	0.0035	0.1083	0.0749	0.0687	0.0326	0.0105	-0.0472
x28	-0.2808*	-0.2465*	-0.2051*	-0.2522*	-0.1720*	-0.3638*	-0.2764*
x29	-0.1426*	-0.1106	-0.0497	-0.1525*	-0.0883	-0.3093*	-0.2142*
x23	-0.0168	-0.0444	-0.0795	0.1281	0.0472	0.1536*	0.0758
x25	-0.1492*	-0.2081*	-0.1470*	-0.1679*	-0.0769	-0.1978*	-0.1261

	x5	transx5	x9	transx9	x10	transx10	x11
x5	1.0000						
transx5	0.9714*	1.0000					
x9	-0.8975*	-0.8041*	1.0000				
transx9	-0.8715*	-0.7561*	0.9878*	1.0000			
x10	0.7527*	0.7064*	-0.7389*	-0.7165*	1.0000		
transx10	0.5344*	0.5469*	-0.4684*	-0.4333*	0.9070*	1.0000	
x11	0.9512*	0.8989*	-0.8907*	-0.8755*	0.7688*	0.5141*	1.0000
transx11	0.9487*	0.9455*	-0.8289*	-0.7915*	0.7412*	0.5368*	0.9727*
x12	-0.8585*	-0.7596*	0.9532*	0.9505*	-0.7346*	-0.4473*	-0.9171*
transx12	-0.8205*	-0.7046*	0.9279*	0.9463*	-0.7061*	-0.4094*	-0.8908*
confinvol	0.7937*	0.6549*	-0.9009*	-0.9449*	0.6692*	0.3788*	0.8130*
lyears	-0.7937*	-0.6549*	0.9009*	0.9449*	-0.6692*	-0.3788*	-0.8130*
myears	0.1707*	-0.0290	-0.4511*	-0.5500*	0.2414*	0.0137	0.2584*
hyears	0.9088*	0.9461*	-0.7342*	-0.6813*	0.6536*	0.5120*	0.8344*
warinvol	0.7838*	0.7328*	-0.7633*	-0.7369*	0.6155*	0.4129*	0.7172*
glyears	-0.7391*	-0.6098*	0.8389*	0.8799*	-0.6534*	-0.3561*	-0.8123*
gmyears	0.0831	-0.0753	-0.3183*	-0.4089*	0.1573*	-0.0299	0.1359
ghyears	0.8759*	0.8741*	-0.7587*	-0.7169*	0.6862*	0.4956*	0.9159*
x17	0.3462*	0.3278*	-0.3399*	-0.3155*	0.3715*	0.2708*	0.3327*
x31	0.3032*	0.2665*	-0.3250*	-0.3181*	0.3724*	0.2766*	0.3306*
x18	0.1723*	0.1861*	-0.1495*	-0.1327	0.2957*	0.3129*	0.2838*
x19	-0.3254*	-0.2767*	0.3238*	0.3496*	-0.3259*	-0.2367*	-0.3259*
x20	0.3114*	0.2932*	-0.2828*	-0.2646*	0.3185*	0.2189*	0.3115*
x21	-0.0655	-0.0501	0.0800	0.0847	-0.0606	-0.0300	-0.0751
x22	-0.2644*	-0.2254*	0.2654*	0.2837*	-0.2529*	-0.1587*	-0.2873*
transx22	-0.3302*	-0.2738*	0.3547*	0.3766*	-0.3295*	-0.2090*	-0.3400*
x24	-0.2323*	-0.2046*	0.2057*	0.2250*	-0.2302*	-0.1521*	-0.2588*
transx24	-0.3959*	-0.3499*	0.3844*	0.4031*	-0.4023*	-0.2849*	-0.4091*
x27	0.1148	0.0685	-0.1498*	-0.1623*	0.0853	-0.0081	0.1398
x28	-0.3350*	-0.2917*	0.3401*	0.3520*	-0.3535*	-0.2667*	-0.3423*
x29	-0.2339*	-0.1865*	0.2563*	0.2699*	-0.2530*	-0.1534*	-0.2788*
x23	0.2455*	0.2120*	-0.2760*	-0.2953*	0.1393	0.0158	0.2992*
x25	-0.2256*	-0.1812*	0.2514*	0.2647*	-0.2130*	-0.1139	-0.2172*

	transx11	x12	transx12	confin~l	lyears	myears	hyears
transx11	1.0000						
x12	-0.8330*	1.0000					
transx12	-0.7848*	0.9885*	1.0000				
confinvol	0.7050*	-0.8820*	-0.9072*	1.0000			
lyears	-0.7050*	0.8820*	0.9072*	-1.0000	1.0000		
myears	0.0791	-0.4705*	-0.5489*	0.7070*	-0.7070*	1.0000	
hyears	0.8909*	-0.6854*	-0.6300*	0.5766*	-0.5766*	-0.1701*	1.0000
warinvol	0.6803*	-0.7147*	-0.6825*	0.6561*	-0.6561*	0.1803*	0.7060*
glyears	-0.6845*	0.9039*	0.9437*	-0.9312*	0.9312*	-0.6584*	-0.5369*
gmyears	-0.0539	-0.3893*	-0.4890*	0.5597*	-0.5597*	0.8223*	-0.1701*
ghyears	0.9489*	-0.7687*	-0.7158*	0.6251*	-0.6251*	-0.0066	0.8788*
x17	0.3146*	-0.3339*	-0.3115*	0.2978*	-0.2978*	0.1063	0.2921*
x31	0.2948*	-0.3433*	-0.3395*	0.3342*	-0.3342*	0.1963*	0.2389*
x18	0.2811*	-0.2600*	-0.2496*	0.1084	-0.1084	-0.0229	0.1774*
x19	-0.2741*	0.3299*	0.3499*	-0.3641*	0.3641*	-0.2096*	-0.2651*
x20	0.2964*	-0.2941*	-0.2834*	0.2472*	-0.2472*	0.0541	0.2820*
x21	-0.0597	0.0876	0.0923	-0.0904	0.0904	-0.0725	-0.0422
x22	-0.2484*	0.2900*	0.3068*	-0.3048*	0.3048*	-0.1848*	-0.2112*
transx22	-0.2817*	0.3755*	0.3954*	-0.3850*	0.3850*	-0.2278*	-0.2733*
x24	-0.2316*	0.2331*	0.2510*	-0.2519*	0.2519*	-0.1350	-0.1951*
transx24	-0.3653*	0.4108*	0.4260*	-0.4197*	0.4197*	-0.1999*	-0.3538*
x27	0.0906	-0.1715*	-0.1879*	0.1634*	-0.1634*	0.1726*	0.0283
x28	-0.2972*	0.3418*	0.3583*	-0.3405*	0.3405*	-0.1537*	-0.2969*
x29	-0.2362*	0.2883*	0.3007*	-0.2832*	0.2832*	-0.1799*	-0.1868*
x23	0.2783*	-0.2957*	-0.3145*	0.3030*	-0.3030*	0.1856*	0.2077*
x25	-0.1679*	0.2654*	0.2802*	-0.2735*	0.2735*	-0.1740*	-0.1801*

	warinvol	glyears	gmyears	ghyears	x17	x31	x18
warinvol	1.0000						
glyears	-0.6110*	1.0000					
gmyears	0.1429*	-0.6584*	1.0000				
ghyears	0.6461*	-0.6110*	-0.1936*	1.0000			
x17	0.3645*	-0.2917*	0.0946	0.2807*	1.0000		
x31	0.2963*	-0.3469*	0.1575*	0.2866*	0.8353*	1.0000	
x18	0.1464*	-0.2166*	-0.0203	0.3037*	0.0432	0.0924	1.0000
x19	-0.3031*	0.3562*	-0.2363*	-0.2158*	-0.2971*	-0.2431*	-0.0835
x20	0.3184*	-0.2788*	0.0616	0.2986*	0.3053*	0.1996*	0.0307
x21	-0.0569	0.0989	-0.0726	-0.0527	-0.0750	-0.0977	-0.0171
x22	-0.2157*	0.3221*	-0.1829*	-0.2275*	-0.2437*	-0.2536*	-0.0279
transx22	-0.3133*	0.3991*	-0.2494*	-0.2578*	-0.3626*	-0.3438*	-0.0198
x24	-0.1615*	0.2720*	-0.1306	-0.2171*	-0.2224*	-0.2213*	0.0047
transx24	-0.3241*	0.4294*	-0.2106*	-0.3383*	-0.3664*	-0.3462*	-0.0390
x27	0.1260	-0.1789*	0.1573*	0.0677	0.1330	0.1017	-0.0097
x28	-0.3249*	0.3586*	-0.1800*	-0.2781*	-0.2248*	-0.2575*	-0.0275
x29	-0.2419*	0.3127*	-0.1533*	-0.2464*	-0.1081	-0.2039*	-0.0341
x23	0.1231	-0.3388*	0.1781*	0.2543*	0.0062	0.1345	-0.0174
x25	-0.2445*	0.2961*	-0.2176*	-0.1571*	-0.2697*	-0.2776*	0.0126

	x19	x20	x21	x22	transx22	x24	transx24
x19	1.0000						
x20	-0.3327*	1.0000					
x21	0.2147*	-0.0413	1.0000				
x22	0.6144*	-0.3632*	0.2284*	1.0000			
transx22	0.7113*	-0.4915*	0.1885*	0.8768*	1.0000		
x24	0.5639*	-0.3671*	0.1672*	0.9143*	0.7791*	1.0000	
transx24	0.7153*	-0.5129*	0.1678*	0.8208*	0.9225*	0.8210*	1.0000
x27	-0.2273*	0.2832*	-0.0528	-0.4143*	-0.3734*	-0.3742*	-0.3288*
x28	0.5295*	-0.5800*	0.0852	0.4541*	0.6484*	0.4272*	0.6728*
x29	0.4127*	-0.4416*	0.0762	0.3825*	0.5577*	0.3324*	0.5528*
x23	-0.1057	0.2453*	-0.0629	-0.2107*	-0.2551*	-0.2653*	-0.3141*
x25	0.3293*	-0.1774*	0.1351	0.1601*	0.3441*	0.0482	0.3318*

	x27	x28	x29	x23	x25
x27	1.0000				
x28	-0.2987*	1.0000			
x29	-0.0922	0.7692*	1.0000		
x23	-0.0524	-0.3932*	-0.4264*	1.0000	
x25	-0.1704*	0.3791*	0.3668*	-0.0475	1.0000

Annex D:

OVERVIEW ON DATA TRANSFORMATION METHODS USED[329]

Box-Cox Transformation: $w_t = \begin{cases} \frac{y_t^{\lambda}-1}{\lambda}, & \lambda \neq 0 \\ \ln(y_t), & \lambda = 0 \end{cases}$ where power λ maximizes the logarithm of the likelihood function.	**Inverse Box-Cox Transformation:** $\hat{y}_t = \begin{cases} E\left[(\lambda w_t + 1)^{1/\lambda}\right], & \lambda \neq 0 \\ E\left[e^{w_t}\right] = \exp(\hat{w}_t + \frac{1}{2}\sigma_t^2), & \lambda = 0 \end{cases}$
Logarithmic Transformation: $w_t = \ln(y_t)$	**Inverse-Logarithmic Transformation**: $\hat{y}_t = \exp[-w_t]$
Logit Transformation: $w_t = \ln[y_t / (1-y_t)]$	**Inverse-Logistic transformation:** $\hat{y}_t = 1/[1+ \exp[-w_t]$

Notes: $y_t > 0$ is the original score, w_t the transformed score and $\hat{y}_t$ the predicted scores.

[329] See Schnell (1994: 81-82); http://www.itl.nist.gov/div898/handbook/pmc/section5/pmc52.htm, 7.12.2004; http://gsbwww.uchicago.edu/computing/research/SASManual/qc/chape/sect11.htm, 7.12.2004; http://apus.wiwi.hu-berlin.de/~sigbert/diplom/zanter/node10.html, 3.1.2005.

Annex E: Results of Final OLS Regression and Robust Regression Analyses (with HIV-Prevalence (y1 transformed) as the dependent variable)

Model	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6		Model 7	
Conflict measure	conflict dummy	(robust)	war dummy	(robust)	short duration	(robust)	medium duration	(robust)	high duration	(robust)	No. of conflicts	(robust)	No. of conflicts, extended sample	(robust)
Parameter:														
Conflict involvement	.073 *** (.013)	.074 *** (.013)	.070 *** (.014)	.100 *** (.013)	-.073 *** (.013)	-.074 *** (.013)	.038 ** (.019)	.043 ** (.019)	.068 *** (.015)	.092 *** (.014)	.015 *** *.434* (002)	.016 *** (.002)	.010 *** *.409* (.001)	.011 *** (.001)
Income inequality (x27)	.003 *** (.001)	.002 *** (.001)	.003 *** (.001)	.003 *** (.001)	.003 *** (.001)	.002 *** (.001)	.002 ** (.001)	.002 ** (.001)	.003 *** (.001)	.004 *** (.001)	.004 *** *.263* (.001)	.004 *** (.001)	.004 *** *.263* (.001)	.004 *** (.001)
HIV_{t-2} (x33)	.015 *** (.002)	.020 *** (.002)	.015 *** (.002)	.018 *** (.002)	.015 *** (.002)	.020 *** (.002)	.014 *** (.002)	.018 *** (.002)	.015 *** (.002)	.018 *** (.002)	.014 *** *.370* (.002)	.018 *** (.002)	.015 *** *.375* (.002)	.018 *** (.002)
Youth Bulge (x25)	-1.377 *** (.344)	-1.058 *** (.344)	-1.511 *** (.346)	-1.460 *** (.305)	-1.377 *** (.344)	-1.058 *** (.344)	-1.569 *** (.368)	-1.268 *** (.370)	-1.629 *** (.384)	-1.689 *** (.316)	-1.928 *** *-.298* (.324)	-1.811 *** (.312)	-2.107 *** *-.326* (.332)	-1.987 *** (.322)
Culture (x23)	-.151*** (.020)	-.154 *** (.019)	-.112 *** (.021)	-.086 *** (.018)	-.151*** (.020)	-.154 *** (.019)	-.147 *** (.022)	-.153 *** (.022)	-.129 *** (.020)	-.108 *** (0.19)	-.067*** *-.175* (.021)	-.067 *** (.020)	-.073 *** *-.190* (.021)	-.075 *** (.020)
Level of Education (x28)	-.003 *** (.001)	-.003 *** (.001)	-.002 *** (.001)	-.002 *** (.000)	-.003 *** (.001)	-.003 *** (.001)	-.003 *** (.001)	-.003 *** (.001)	-.003 *** (.001)	-.003 *** (.000)	-.001** *-.192* (.001)	-.001 ** (.001)	-.002 *** *-.230* (.001)	-.001 *** (.001)
Urbanization (Pace) (x20)	-.012 ** (.005)	-.015 *** (.005)	-.012 ** (.005)	-.012 *** (.004)	-.012 ** (.005)	-.015 *** (.005)	-.015 *** (.005)	-.018 *** (.005)	-.012 ** (.005)	-.013 *** (.004)	-.008 *-.092* (.005)	-.007 (.005)	-.010 ** *-.115* (.005)	-.009 ** (.005)
Health expenditure ln(x24)	-.012 * (.007)	-.013 *** (.006)	-.018 *** (.007)	-.018 *** (.006)	-.012 * (.007)	-.013 ** (.006)	-.021*** (.007)	-.023 *** (.007)	-.006 (.007)	-.001 (.007)	-.009 *-.065* (.010)	-.010 (.009)	-.008 *-.056* (.009)	-.009 (.009)
Neighboring Conflict (x31)	-.094 ** (.037)	-.121** (.037)	-.110 *** (.038)	-.119 *** (.034)	-.094 ** (.037)	-.121 *** (.037)	-.117 *** (.039)	-.146 *** (.039)	-.093 ** (.038)	-.090 ** (.036)	-.092** *-.134* (.036)	-.129 *** (.034)	-.093 ** *-.135* (.036)	-.129 *** (.035)
Observations (N)	197	188	197	185	197	188	197	188	197	185	197	188	197	188
R-squared	0.6588	0.6884	0.6477	0.7487	0.6588	0.6884	0.6099	0.6396	0.6404	0.7273	0.6857	0.7330	0.6804	0.7237
Prob > F	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000
Root MSE	.078	.074	.079	.066	.078	.074	.083	.080	.080	.069	.075	.069	.075	.070

Annex E (continuing):

Model	Model 8		Model 9		Model 10		Model 11		Model 12	
Conflict measure	No. of intermediate conflicts	(robust)	No. of minor conflicts	(robust)	No. of wars	(robust)	Years in conflict, extended sample	(robust)	Peace duration, extended sample	(robust)
Parameter:										
Conflict involvement	.029 *** *.351* (.005)	.035 *** (004)	.038 *** *.463* (.006)	.037 *** (.006)	.040 *** *.290* (.008)	0.44 *** (.007)	-.036 *** *-.926* (.009)	-.044 *** (.009)	-.057 *** *-1.498* (.012)	-.060 *** (.011)
(Conflict involvement)²	-	-	-	-	-	-	.005 *** *.980* (.001)	.006 *** (.001)	.007 *** *1.494* (.001)	.007 *** (.001)
Income inequality (x27)	.004 *** *.239* (.001)	.004 *** (.001)	.004 *** *.280* (.001)	.004 *** (.001)	.003 *** *.193* (.001)	.003 *** (.001)	.004 *** *.224* (.001)	.004 *** (.001)	.004 *** *.236* (.001)	.004 *** (.001)
HIV_{t-2} (x33)	.015 *** *.384* (.002)	.017 *** (.002)	.014 *** *.357* (.002)	.018 *** (.002)	.014 *** *.370* (.002)	.020 *** (.002)	.014 *** *.353* (.002)	.015 *** (.002)	.014 *** *.351* (.002)	.017 *** (.002)
Youth Bulge (x25)	-2.030 *** *-.314* (.335)	-2.236 *** (.309)	-2.089 *** *-.323* (.334)	-1.804 *** (.337)	-1.474 *** *-.228* (.344)	-1.094 *** (.342)	-1.920 *** *-.297* (.363)	-2.253 *** (.342)	-1.883 *** *-.291* (.349)	-1.828 *** (.343)
Culture (x23)	-.093*** *-.241* (.021)	-.067 *** (.019)	-.057 ** *-.148* (.023)	-.064 *** (.022)	-.093 *** *-.242* (.021)	-.092 *** (.021)	-.133 *** *-.346* (.021)	-.110 *** (.019)	-.118 *** *-.307* (.021)	-.125 *** (.020)
Level of Education (x28)	-.002 *** *-.269* (.001)	-.002 *** (.001)	-.001* *-.154* (.001)	-.001 ** (.001)	-.002 *** *-.324* (.001)	-.002 *** (.001)	-.003 *** *-.388* (.001)	-.003 *** (.001)	-.003 *** *-.376* (.001)	-.003 *** (.001)
Urbanization (Pace) (x20)	-.013 ** *-.127* (.009)	-.010 ** (.004)	-.005 *-.061* (.005)	-.008 (.005)	-.012 ** *-.143* (.005)	-.015 *** (.005)	-.012 ** *-.147* (.005)	-.013 *** (.005)	-.015 *** *-.174* (.005)	-.016 *** (.005)
Health expenditure ln(x24)	-.012 *-.010* (.007)	-.010 (.008)	-.003 *-.036* (.007)	-.005 (.007)	-.013 * *-.135* (.007)	-.013 ** (.006)	-.014 * *-.146* (.007)	-.008 (.007)	-.020 *** *-.213* (.007)	-.025 *** (.007)
Neighboring Conflict (x31)	-.074 ** *-.108* (.037)	-.067 ** (.034)	-.101 *** *-.147* (.036)	-.125 *** (.036)	-.125 *** *-.182* (.037)	-.157 *** (.036)	-.080 ** *-.116* (.040)	-.072* (.037)	-.075 * *-.110* (.039)	-.119 *** (.038)
Observations (N)	197	187	197	188	197	188	197	186	197	187
R-squared	0.6709	0.7476	0.6782	0.6999	0.6530	0.6907	0.6346	0.7106	0.6454	0.6896
Prob > F	0.0000	0.000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000
Root MSE	.077	.066	.076	.073	.077	.074	.081	.071	.080	.074

Notes Annex E:

Standardized beta-coefficients are shown in italic figures

Standard errors are shown in parentheses

* Indicates that the result is significantly different from zero at the 10% level
** Indicates that the result is significantly different from zero at the 5% level
*** Indicates that the result is significantly different from zero at the 1% (or less) level

The right columns of each model give results of robust regression analyses, which exclude cases with cook's D >4/e(N)

Model 1 uses a dummy for conflict involvement as the key explanatory variable
Model 2 uses a dummy for war involvement as the key explanatory variable
Model 3 uses a dummy for short duration of conflict involvement (<1 year) as the key explanatory variable
Model 4 uses a dummy for medium duration of conflict involvement (1-5 years) as the key explanatory variable
Model 5 uses a dummy for high duration of conflict involvement (6-8 years) as the key explanatory variable
Model 6 uses the number of conflicts involved (x1) as the key explanatory variable
Model 7 uses the number of conflicts involved, including unclear cases of conflict involvement (x10) as the key explanatory variable
Model 8 uses the number of intermediate armed conflicts involved (x3) as the key explanatory variable
Model 9 uses the number of minor armed conflicts involved (x4) as the key explanatory variable
Model 10 uses the number of wars involved (x2) as the key explanatory variable
Model 11 uses the years spent in conflict, including unclear cases of conflict involvement (x11) as the key explanatory variable as well as $(x11)^2$ to model a non-linear relationship
Model 12 uses the years since last conflict involvement, including unclear cases of conflict involvement (x12) as the key explanatory variable as well as $(x12)^2$ to model a non-linear relationship

Annex F:

LIST OF COUNTRIES	
1. Afghanistan	51. Denmark
2. Albania	52. Djibouti
3. Algeria	53. Dominica
4. Angola	54. Dominican Republic
5. American Samoa	55. East Timor
6. Andorra	56. Ecuador
7. Antigua and Barbuda	57. Egypt, Arab Rep.
8. Argentina	58. El Salvador
9. Armenia	59. Equatorial Guinea
10. Australia	60. Eritrea
11. Austria	61. Estonia
12. Azerbaijan	62. Ethiopia
13. Bahamas, The	63. Fiji
14. Bahrain	64. Finland
15. Bangladesh	65. France
16. Barbados	66. Gabon
17. Belarus	67. Gambia, The
18. Belgium	68. Georgia
19. Belize	69. Germany
20. Benin	70. Ghana
21. Bhutan	71. Greece
22. Bolivia	72. Grenada
23. Bosnia and Herzegovina	73. Guam
24. Botswana	74. Guatemala
25. Brazil	75. Guinea
26. Brunei	76. Guinea-Bissau
27. Bulgaria	77. Guyana
28. Burkina Faso	78. Haiti
29. Burundi	79. Honduras
30. Cambodia	80. Hungary
31. Cameroon	81. Iceland
32. Canada	82. India
33. Cape Verde	83. Indonesia
34. Cayman Islands	84. Iran, Islamic Rep.
35. Central African Republic	85. Iraq
36. Chad	86. Ireland
37. Chile	87. Israel
38. China	88. Italy
39. Hong Kong, China	89. Jamaica
40. Macao, China	90. Japan
41. Colombia	91. Jordan
42. Comoros	92. Kazakhstan
43. Congo, Dem. Rep. (Zaire)	93. Kenya
44. Congo, Rep. (Brazzaville)	94. Kiribati
45. Costa Rica	95. Korea, Dem. Rep.
46. Côte d'Ivoire	96. Korea, Rep.
47. Croatia	97. Kuwait
48. Cuba	98. Kyrgyz Republic
49. Cyprus	99. Lao PDR
50. Czech Republic	100. Latvia

101. Lebanon	151. Samoa
102. Lesotho	152. San Marino
103. Liberia	153. São Tomé and Principe
104. Libya	154. Saudi Arabia
105. Lithuania	155. Senegal
106. Luxembourg	156. Seychelles
107. Macedonia, FYR	157. Sierra Leone
108. Madagascar	158. Singapore
109. Malawi	159. Slovak Republic
110. Malaysia	160. Slovenia
111. Maldives	161. Solomon Islands
112. Mali	162. Somalia
113. Malta	163. South Africa
114. Marshall Islands	164. Spain
115. Mauritania	165. Sri Lanka
116. Mauritius	166. St. Kitts and Nevis
117. Mexico	167. St. Lucia
118. Micronesia, Fed. Sts	168. St. Vincent and the Grenadines
119. Moldova	169. Sudan
120. Monaco	170. Suriname
121. Mongolia	171. Swaziland
122. Morocco	172. Sweden
123. Mozambique	173. Switzerland
124. Myanmar	174. Syrian Arab Republic
125. Namibia	175. Tajikistan
126. Nepal	176. Tanzania
127. Netherlands	177. Thailand
128. Netherlands Antilles	178. Togo
129. New Caledonia	179. Tonga
130. New Zealand	180. Trinidad and Tobago
131. Nicaragua	181. Tunisia
132. Niger	182. Turkey
133. Nigeria	183. Turkmenistan
134. Northern Mariana Islands	184. Uganda
135. Norway	185. Ukraine
136. Oman	186. United Arab Emirates
137. Pakistan	187. United Kingdom
138. Palau	188. United States
139. Panama	189. Uruguay
140. Papua New Guinea	190. Uzbekistan
141. Paraguay	191. Vanuatu
142. Peru	192. Venezuela, RB
143. Philippines	193. Vietnam
144. Poland	194. Yemen, Rep.
145. Portugal	195. Yugoslavia, FR (Serbia/Montenegro)
146. Puerto Rico	196. Zambia
147. Qatar	197. Zimbabwe
148. Romania	
149. Russia	
150. Rwanda	

Annex G:

Missing Data

In the cases of missing 2003 HIV-prevalence rates (y1, y2, or y3), I used regional average HIV-prevalence rates as given in the 2004 UNAIDS/WHO Report on the global AIDS epidemic. Cape Verde, Equatorial Guinea, Comoros, Guinea-Bissau, Mauritius, Seychelles, Sierra Leone, Somalia and Sao Tome and Principe have been categorized as Sub-Saharan African countries. In the case of Belarus, with a low estimate (y2) of HIV-prevalence of 0.2% and a high estimate (y3) of 0.8%, I imputed the average (0.5%) for the missing value on y1.

Missing 2001 HIV-prevalence data has also been replaced by regional average HIV-prevalence rates given in the 2002 UNAIDS/WHO Report on the global AIDS epidemic. In cases of unclear plausibility bounds (where no specific value is given for the low estimate, the regular estimate is <=0.1 and the high estimate <0.2) I used a range of 0.01-0.2, with 0.01 being the low estimate, 0.1 the regular estimate and 0.2 the high estimate. Generally, values <0.1 have been changed to 0.01.

For cases with missing income data (x22), regional averages, which are provided by the World Bank (2004 World Development Indicators) or data according to each countries human development rank have been used.

In order to impute missing data on health expenditures (x24), I calculated regional averages according to the World Bank regional grouping.

In the same way, `average adult literacy rates´ (x28) by region have been used where missing. Additionally, I calculated the average value for adult literacy rates for high human development countries.

For the cases of missing income inequality data (x27), I calculated the average gini index of income inequality for high, medium and low human development countries.

Missing data on disparity in education (x29) have been replaced by regional data on the ratio of literate women to men (15-24 year-olds) in 2000. These data are published by the U.N. Statistics Division (http://millenniumindicators.un.org/unsd/mi/mi_worldregn.asp, 10.7.2004). Additionally, I calculated regional averages for Southern European Countries and Eastern European Countries.

Three missing values of population density (x21) have been replaced by own calculations. I divided available data on population size (x18) through CIA data on land surface area in sq. km provided by the CIA World Factbook 2003.

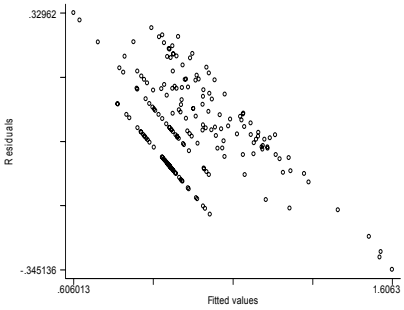
CIA data on the proportion of the Muslim and Jewish population (x23) was unclear in cases where ranges or a combined figure for several religious groups was given. In these cases I used average values. Gabon was assigned a value of 0.005 and Romania a value of 0.004. Missing CIA data was replaced by data given in the 2001 Fischer Weltalmanach for Austria, Eritrea, Malaysia, Singapore, Sweden, Ukraine, Yemen, and Russia. Vietnam was assigned a value of 0.0, Swaziland (0.10), Belarus (0.04), Estonia (0.044) and Lithuania (0.07) based on information from the following online sources:
http://www.auswaertiges-amt.de/www/de/laenderinfos/laender/laender_ausgabe_html?land_id=190&type_id=2, 18.12.2004, http://www.vietnam-freunde.net/seite01/html/religion___glauben_in_vietnam.html, 18.12.2004,
http://www.swasiland.de/c_swazis.asp?content=sprache_religion.html,
http://www.belarus-botschaft.de/de/presse41_2002_de.htm, 18.12.2004,
http://www.staatenderwelt.de/staaten/estland/estland.htm, 18.12. 2004.

Missing data on the proportion of males age 15-49 years (x25) have been replaced by CIA data (2003 World Factbook) on the proportion of males age 15-64 years. As the median age in the replaced countries is generally very low (between 17.4 and 43 years) this is not expected to result in a significant overestimation of the proportion of males age 15-49 years. The calculation of the value for East Timor is based on data provided by UNPD (World Population Prospects: the 2002 revision).

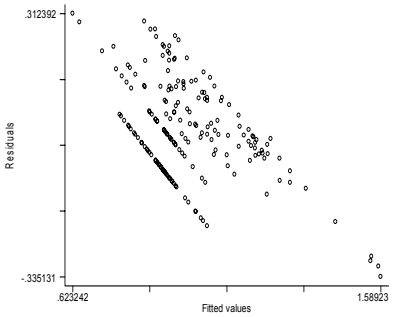
Annex H:

Residual-vs.-Fitted Plots of Final OLS Regressions

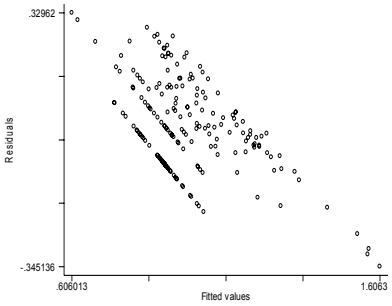
Model 1:



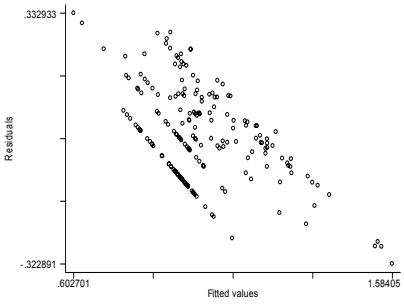
Model 2:



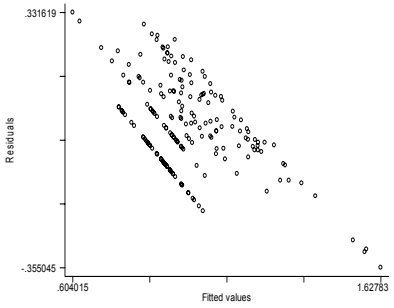
Model 3:



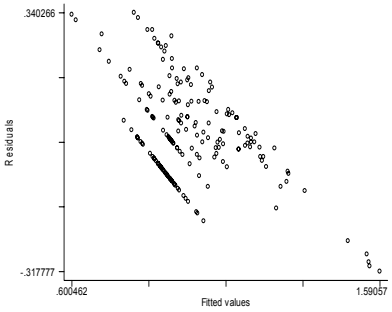
Model 4:



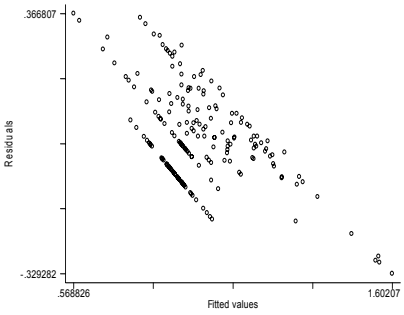
Model 5:



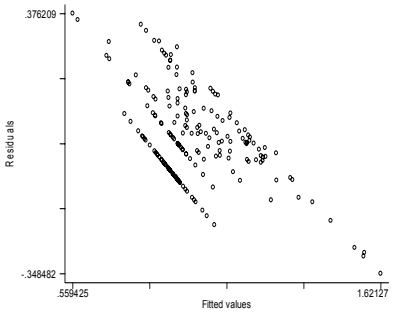
Model 6:



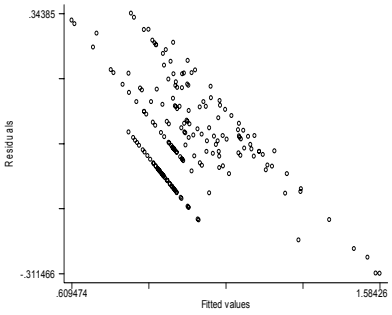
Model 7:



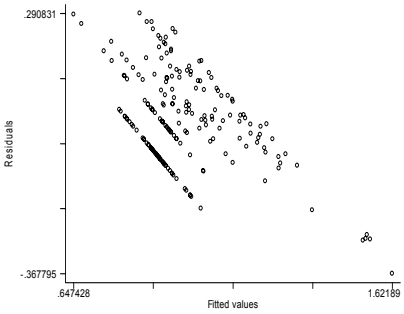
Model 8:



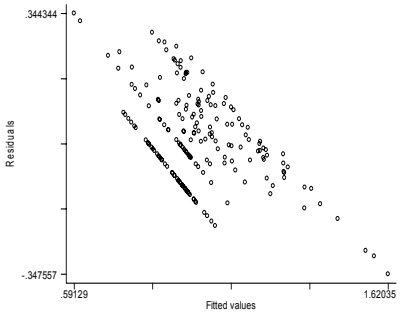
Model 9:



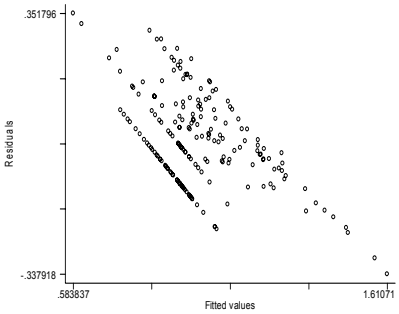
Model 10:



Model 11:



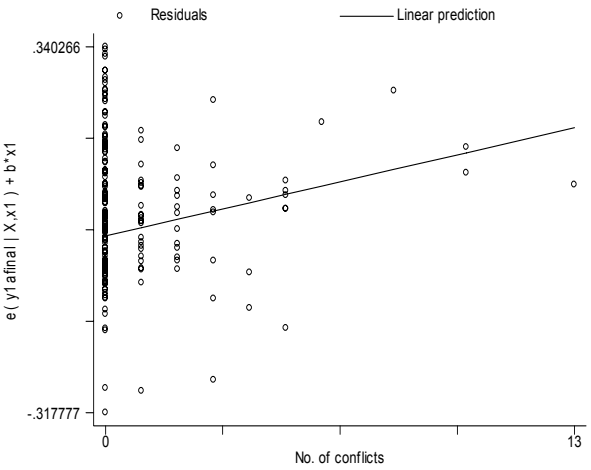
Model 12:



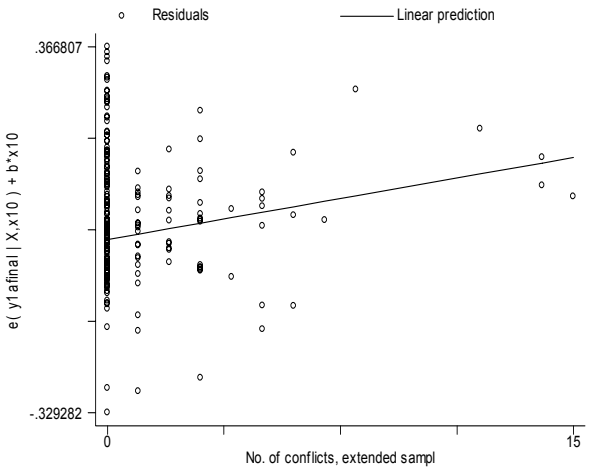
Annex I

Component-Plus-Residual Plots of Final OLS Regressions

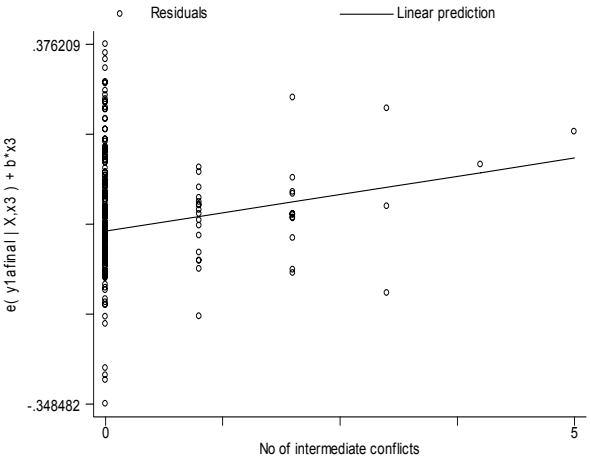
Model 6:



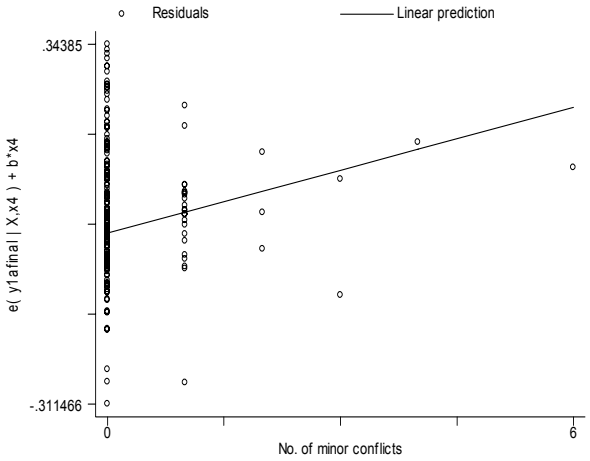
Model 7:



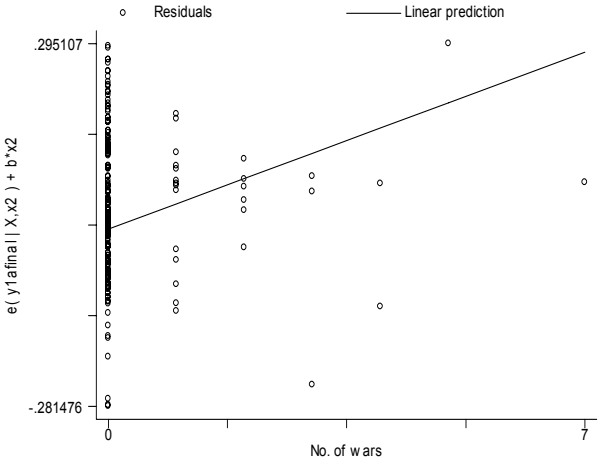
Model 8:



Model 9:



Model 10:



References

Ahmad, Khabir, 2001. `Political infighting causes Zimbabwe to dissolve AIDS council board´, The Lancet, Vol. 357: 942.

Ainsworth, Martha & Semali, Innocent, 1998. `Who is most likely to die of AIDS? Socioeconomic correlates of adult deaths in Kagera region, Tanzania´, in `Confronting AIDS: evidence from the developing world´, Ainsworth, M. & Fransen, L. & Over, M. (eds.) 1998: 95-111, World Bank background paper, The World Bank, Washington, DC.

Alban, Anita & Guinness, Lorna, 2000. `The economic impact of AIDS in Africa: a review of the literature´, UNAIDS background paper for the African Development Forum, `UNAIDS: the greatest leadership challenge´, Addis Ababa, December 2000, see PPP available online at http://www.stanford.edu/class/history48q/Materials/presentations_print/adf/tsld001.htm, 29.10.2004.

Altmann, Dennis 1999. `Globalization, political economy and HIV/AIDS´, Theory and Society, Vol. 28 (4): 559-584.

Amowitz, L.L. & Reis, Ch. & Lyons, K.H. & Vann, B.& Mansaray, B. & Akinsulure-Smith, A.M. & Taylor, L. & Iacopino, V., 2002. `Prevalence of war-related sexual violence and other human rights abuses among internally displaced persons in Sierra Leone´, Journal of the American Medical Association (JAMA), Vol. 287 (4): 513-521, http://www.phrusa.org/research/pdf/sl_jama.pdf, 5.12.2003.

Astill, James, 2001. `War injects Aids into the tragedy of Sierra Leone´, Special report on Aids in the military, The Guardian, May 12th of 2001, http://www.guardian.co.uk/sierra/article/0,2763,489744,00.html, 1.6.2001.

Auvert, B. & Buvé, A. & Lagarde, E. & Kahindo, M. & Chege, J. & Rutenberg, N. & Musonda, R. & Laourou, M. & Akam, E. & Weis, H. A., 2001. `Male circumcision and HIV infection in four cities in sub-Saharan Africa´, AIDS, Vol. 15 (supplement 4): 31-40.

Badura, Bernhard & Kickbusch, Ilona, 1991. `Introduction´, in `health promotion research-towards a new social epidemiology´, Badura, Bernhard & Kickbusch, Ilona (eds.) 1991: 1-7, WHO Regional Publications, European Series No. 37, WHO Regional Office for Europe, Copenhagen.

Barnett, T. & Whiteside, A. & Decosas, J., 2000. `The Jaipur Paradigm: understanding social susceptibility and vulnerability to HIV´, South African Medical Journal, Vol. 90 (11): 1098-1101.

Baumann, Laurie J. & Siegle, Karolynn, 1986. `Methodological issues in AIDS - related research´, in `the social dimensions of AIDS - method and theory´, Feldmann, Douglas A. & Johnson, Thomas M. (eds.), 1986: 15-41, Praeger Publishers, New York/Westport, Connecticut/London.

Beesey, Allan, 2000. ` HIV vulnerability and mobile populations: Thailand and its borders´, Development Bulletin, Vol. 52: 38-41.

Bellamy, Carol, 2001. `Speech to the Security Council on children and armed conflicts, November 20th of 2001, http://www.unicef.org/media/media_10383.html, 30.9.2003.

Benjamin, Judy A. 2001.`Conflict, post-conflict, and HIV/AIDS – the gender connections: women, war and HIV/AIDS: West Africa and the Great Lakes´, remarks presented at the World Bank, International Women's Day, March 8th of 2001.

Berkman, Lisa & Kawachi, Ichiro, 2000. `A historical framework for Social Epidemiology´, in `Social Epidemiology´, Berkman, Lisa & Kawachi, Ichiro (eds.) 2000: 3-12, Oxford University Press, New York.

Bloor, Michael, 1995. `The sociology of HIV transmission´, SAGE Publications, London.

Boerma, J. T. & Ghys, P.D. & Walker, N., 2003.`Estimates of HIV-1 prevalence from national population-based surveys as a new gold standard´, The Lancet, Vol. 362: 1929-1931.

Bollinger, Lori, 2002. `HIV/AIDS and its impact on trade and commerce´, in `State of the art: AIDS and economics´, Forsythe, Steven (editor), 2002: 38-48, special series prepared in June 2002 for the IAEN Economics of HIV/AIDS in Developing Countries Symposium, Barcelona http://www.iaen.org/conferences/stateofepidemic.php, 19.1.2005.

Bonnel, René, 2000.`Economic analysis of HIV/AIDS´, background paper, The World Bank ACTafrica AIDS Campaign Team for Africa, September 2000.

Brody, Alan & Ascroft, Joseph, 2004. `Message of hope for Swaziland´s young people: preliminary results from a baseline study on HIV risk factors´, http://www.sarpn.org.za/documents/d0000930/index.php, 31.12.2004.

Brown, Lynn R., 1997.`The potential impact of AIDS on population and economic growth rates´, International Food Policy Research Institute 2020, Vision Brief 43 (June), http://www.ifpri.org/2020/briefs/number43.htm, 15.9.2003.

Burkhalter, Holly J., 2002.`The violent transmission of HIV/AIDS´, Centre for Strategic and International Studies, Africa Program, http://www.csis.org/africa/0206_BurkhalterpaperFINAL.pdf, 5.12.2003.

Buvé, A. & Bishikwabo-Nsarhaza, K. & Mutangadura, G., 2002. `The spread and effect of HIV-1 infection in sub-Saharan Africa´, The Lancet, Vol. 359: 2011-2017.

Buvé, A. & Carael, M. & Hayes, R.J. & Auvert, B. & Ferry, B. & Robinson, N.J. & Anagonou, S. & Kanhonou, L. & Laourou, M. & Abega, S. & Akam, E. & Zekeng, L. & Chege, J. & Kahindo, M. & Rutenberg, N. & Kaona, F. & Musonda, R. & Sukwa, T. & Morison, L. & Weiss, H.A. & Lega, M., 2001.`The multicentre study on factors determining the differential spread of HIV in four African cities: summary and conclusions´, AIDS, Vol. 15 (Supplement 4): 127-131.

Campbell, C. & Williams, B. & Gilgen, D., 2002. `Is social capital a useful conceptual tool for exploring community level influences on HIV infection? An exploratory case study from South Africa´, AIDS Care, Vol.14 (1): 41-54.

Carballo, Manuel & Frajzngier, Vera, 2001. `Reproductive health in the context of forced migration´, UNHCR/UNFPA/International Centre for Migration and Health, Geneva.

Carballo, Manuel & Nerukar, Aditi, 2001. `Migration, refugees, and health risks´, Emerging Infectious Diseases, Vol. 7 (3, Supplement): 556-560.

Carballo, M. & Mansfield, C. & Prokop, M., 2000. `Demobilization and its implications for HIV/AIDS´, background paper, ICMH, CERTI Project, Payson Centre for International Development and Technology Transfer Tulane University, http://www.certi.org/publications/demob/Demobilization_Final.html, 13.12.2003.

Collier, Paul & Hoeffler, Anke, 2004. `Conflicts´, in `global crisis, global solutions´, Bjorn Lomborg (ed.) 2004: 129-156, Cambridge University Press, New York.

Collier, Paul & Hoeffler, Anke, 2000: `On the incidence of civil war in Africa´, first draft, research paper, The World Bank, Washington, DC, http://www.worldbank.org/research/conflict/papers/incidence.htm, 13.12.2003.

Connolly, Màire A. & Heymann, David L., 2002.`Deadly comrades: war and infectious diseases´, The Lancet, Vol. 360 (Supplement): 23, 24, http://www.who.int/disasters/hbp/general/lancet-war_and_infectious_diseases.pdf, 4.12.2003.

Cossa, H. A. & Gloyd, S. & Vaz, R.G. & Folgora, E. & Simbine, E. & Diniz, M. & Kreiss, J.K., 1994. `Syphilis and HIV infection among displaced pregnant women in rural Mozambique´, International Journal of STD & AIDS, Vol. 5: 117-123.

Crystal, Paul & Ehrlich, Lisa, 2004. `No product? No programme!-The logistics of reproductive health supplies in conflict-affected settings´, Forced Migration Review, Vol. 19: 18-19.

Dabis F. & Leroy, V. & Castetbon, K. & Spira, R. & Newell, M.-L. & Salamon, R., 2000. `Preventing mother-to-child transmission of HIV-1 in Africa in the year 2000´, AIDS Vol. 14 (8): 1017-1026.

Darrow, W.W. & Gorman, M.E. & Glick, B.P., 1986.`The social origins of AIDS: social change, sexual behavior, and disease trends´, in `the social dimensions of AIDS -method and theory´, Feldmann, Douglas A. & Johnson, Thomas M. (eds.), 1986: 95-107, Praeger Publishers, New York/Westport, Connecticut/London.

Davis, D.R. & Iqbal, Z. & Zorn, Ch., 2003.`Conflict and public health: modeling spatial effects´, working paper presented at the `workshop on geography, conflict, and cooperation´, joint sessions of workshops, ECPR, March 28th - April 3rd of 2003, Edinburgh, Scotland, http://www.emory.edu/POLS/zorn/Papers/DIZ.pdf, 5.9.2003.

Davis, David R. & Kuritsky, Joel N., 2002.`Violent conflict and its impact on health indicators in sub-Saharan Africa: 1980 to 1997´, paper presented at the annual meeting of the International Studies Association, New Orleans, L.A., http://userwww.service.emory.edu/~poldd/davis.pdf, 12.3.2004.

DeCarlo, Pamela, 1999. `Perceiving one's own risk´, Harvard AIDS Review, Spring 1999, http://www.aids.harvard.edu/news_publications/har/spring_1999/spring99-4.html, 10.12.2004.

DeCastella, Tom, 2003. `Health workers struggle to provide care in Zimbabwe´, The Lancet, Vol. 362: 46-47.

Decosas, Josef & Padian, Nancy S.., 2002. `The profile and context of the epidemics of sexually transmitted infections including HIV in Zimbabwe´, Sexually Transmitted Infections, Vol. 78 (Supplement I): i40-i46, http://stibmjjournals.com/cgi/reprint/78/suppl_1/40, 24.10.2004.

Decosas Josef, 1998. ` Labour migration and HIV epidemics in Africa´, AIDS Analysis Africa, 8(5):6-7.

Deheneffe, J.-C. & Carael, M. & Noumbissi, A.1998. `Socioeconomic determinants of sexual behaviour and condom use´, in `confronting AIDS: evidence from the developing world´, Ainsworth, M. & Fransen, L. & Over, M. (eds.) 1998: 131-146, World Bank background paper, The World Bank, Washington, DC.

DeHoltgrave, David R. & Crosby, Richard A., 2003.`Social capital, poverty, and income inequality as a predictor of gonorrhoea, syphilis, chlamydia and AIDS case rates in the United States´, Sexually Transmitted Infections, Vol. 79: 62-64.

DeHulsters, B. & Barreto, A. & Bastos, R. & Noya, A. & Folgosa, E. & Fransen, L., 2003.`Geographical focusing: an intervention to address increased risk for sexually transmitted diseases during repatriation and resettlement in post-war Mozambique´, Sexually Transmitted Infections, Vol. 79 (PostScript): 77.

Deneke, Ch. & Faltis, M. & Hildebrandt, H. & Trojan, A., 1991.`The role of community groups and voluntary organizations in health promotion´, in `health promotion research - towards a new social epidemiology´, Badura, Bernhard & Kickbusch, Ilona (eds.), 1991: 441-466, WHO Regional Publications, European Series No. 37, WHO Regional Office for Europe, Copenhagen.

Donovan, Paula, 2002. `Rape and HIV/AIDS in Rwanda´, The Lancet, Vol. 360 (supplement): 17-18.

Duckett, Margaret, 2000. `Migrants and HIV / AIDS´, Development Bulletin, Vol. 52: 18-20.

Elliott, Lyn, 1999. `Gender, HIV/AIDS and complex emergencies´, Relief and Rehabilitation Network Newsletter, 14 (June): 4-7, http://www.odihpn.org/pdfbin/newsletter014.pdf, 4.12.2003.

Elliott, Lyn, 1996.`Child prostitution: a case study in military camps in Liberia´, International Conference on AIDS, Jul 7-12, 1996, Abstract available at: http://www.aegis.com/conferences/11wac/TuD136.html, 29.10.2004.

Evans, David B., 2004.`Communicable diseases: alternative perspectives´, perspective paper 2.1 in `global crisis, global solutions´, Bjorn Lomborg (ed.) 2004: 115-123, Cambridge University Press, New York.

Fahrmeier, L. & Künstler R. & Pigeot, I. & Tutz, G., 2001. `Statistik: der Weg zur Datenanalyse´ (3. Auflage), Springer Verlag, Berlin/Heidelberg/New York.

Fearon, James D. & Laitin, David D., 2003. `Ethnicity, insurgency, and civil war´, American Political Science Review, Vol. 97 (1): 75-90.

Fee, Elizabeth & Krieger, Nancy, 1993. `Understanding AIDS: historical interpretations and the limits of Biomedical Individualism´, American Journal of Public Health, Vol. 83 (10): 1477-1486.

Filmer, Deon, 1998.`The socioeconomic correlates of sexual behaviour: a summary of results from an analysis of DHS data´, in `confronting AIDS: evidence from the developing world´, Ainsworth, M. & Fransen, L. & Over, M. (eds.) 1998: 111-131, World Bank background paper, The World Bank, Washington, DC.

Flam, Robin & Stein, Zena, 1986.`Behavior, infection, and immune response: a epidemiological approach´, in `the social dimensions of AIDS - method and theory´, Feldmann, Douglas A. & Johnson, Thomas M. (eds.), 1986: 61-76, Praeger Publishers, New York/Westport, Connecticut/London.

Fleshman, Michael, 2001. `AIDS prevention in the ranks: UN targets peacekeepers, combatants in war against the disease´, Africa Recovery, Vol. 15: 16-18, http://www.un.org/ecosocdev/geninfo/afrec/vol15no1/aidsmil.htm, 4.12.2003.

Fourie, Pieter & Schönteich, Martin, 2001. ` The impact of HIV/AIDS on human security in South and Southern Africa, paper delivered at a South African Association for Political Science (SAAPS) conference, October 7th of 2001, at the University of Durban, Westville, http://www.unaids.org/security/Information/Publications/docs/Canterburypaper.htm, 20.7.2003.

Fredland, Richard A., 1998. `AIDS and development: an inverse correlation? ´, Journal of Modern African Studies, Vol. 36 (4): 547-568.

Garcia-Calleja, J.M. & Zaniewski, E. & Ghys, P.D. & Stanecki, K. & Walker, N., 2004.`A global analysis of trends in the quality of HIV sero-surveillance´, Sexual Transmitted Infections, Vol. 80 (Supplement I): i25-i30.

Gardiner, Rosalie, 2001. `AIDS - the undeclared war´, Towards the Earth Summit 2002: Social Briefing No. 1, February 2001, http://www.earthsummit2002.org/es/issues/AIDS/hiv.pdf, 19.11.2004.

Garnett, G.P. & Grassly, N.C. & Boerma, J.T. & Ghys, P.D., 2004.`Maximising the global use of HIV surveillance data through the development and sharing of analytical tools´, Sexually Transmitted Infections, Vol. 80 (Supplement I): i1-i4.

Gebrekristos, Hirut & Lurie, Mark 2003.`The impact of family housing on HIV transmission among mining communities in South Africa´, Sexual Health Exchange 2003/2, Research Note: 10-11, http://www.kit.nl/exchange, 30.10.2004.

Ghobarah, H. & Huth, P. & Russett, B., 2003.`Civil wars kill and maim people - long after the shooting stops´, American Political Science Review, Vol. 97 (2): 189-202.

Gleditsch, N.P. & Wallensteen, P. & Eriksson, M. & Stollenberg, M. & Strand, H., 2004. `Armed conflict 1946-2001: version 2.1, March 11th of 2004´, http://www.prio.no/cwp/armedconflict/current/conflict_list_1946-2001.pdf, 15.8.2004. http://www.prio.no/cwp/armedconflict/current/unclear_cases _1946-2001.pdf, 15.8.2004. http://www.prio.no/cwp/armedconflict/current/active_conflicts_2002.pdf, 15.8.2004.

Gleditsch, N.P & Strand, H. & Wilhelmsen, L., 2004. `Armed conflict dataset codebook´, http://www.prio.no/cwp/armedconflict/current/codebook_v2_1.pdf, 15.8.2004.

Gleditsch, N.P. & Wallensteen, P. & Eriksson, M. & Stollenberg, M. & Strand, H., 2002. 'Armed conflict 1946–2001: a new dataset', Journal of Peace Research, Vol. 39(5): 615–637.

Gleditsch, N.P & Wallensteen, P. & Eriksson, M. & Stollenberg, M. & Strand, H. 2002. `Armed Conflict 1946-2001: A New Dataset´, Journal of Peace Research, Vol. 39 (5): 615-637, http://www.prio.no/cwp/ArmedConflict/, 28.9.2003.

Goldstone, J.A. & Gurr, T.R. & Harff, B. & Levy, M.A. & Marshall, M.G. & Bates, R.H. & Epstein, D.L. & Kahl, C. H. & Surko, P.T. & Ulfelder, J.C. & Unger, A.N., 2000.`State failure task force report: phase III findings´, McLean, VA: Science Applications International Corporation (SAIC), September 30th of 2000, http://www.cidcm.umd.edu/inscr/stfail/SFTF%20Phase%20III%20Report%20Final.pdf, 12.1.2005.

Gorbach, P. M. & Ryan, C. & Saphonn, V. & Detels, R., 2002. `The impact of social, economic and political forces on emerging HIV epidemics´, AIDS, Vol. 16 (supplement 4): 35-43.

Goyer, KC, 2001. `HIV and political instability in sub-Saharan Africa´, AIDS Analysis Africa Vol. 12 (1): 13, 16.

Grassly, N.C. & Morgan, M. & Walker, N. & Garnett, G. & Stanecki, K.A. & Stover, J. & Brown, T. & Ghys, P.D., 2004.`Uncertainty in estimates of HIV/AIDS: the estimation and application of plausibility bounds´, Sexually Transmitted Infections, Vol. 80 (Supplement I): i31-i38.

Gregson, S. & Nyamukapa, C. & Garnett, G.P. & Mason, P. R. & Zhuwau, T. & Carael, M. & Chandiwana, S.K. & Anderson, R.M., 2002.`Sexual mixing patterns and sex-differentials in teenage exposure to HIV-infection in rural Zimbabwe´, The Lancet, Vol. 359: 1896-1903.

Gregson, S. & Waddell, H. & Chandiwana, S.K., 2001. `School education and HIV control in sub-Saharan Africa: from discord to harmony? ´, in Journal of International Development, Vol. 13 (4): 467-485.

Guha-Sapir, Debarati & van Panhuis, Willem C, 2002. `Armed conflict and public health: a report on knowledge and knowledge gaps´, Centre for Research on the Epidemiology of Disasters (CRED), Brussels.

Hamilton, Lawrence C., 1992. `Regression with Graphics: a second course in applied statistics´, Books/Cole Publishing Company, Pacific Grove, California.

Hankins, C.A. & Friedman, S.R. & Zafar, T. & Strathdee, S.A., 2002.`Transmission and prevention of HIV and sexually transmitted infections in war settings: implications for current and future armed conflict´, AIDS, Vol. 16: 2245-2252.

Haour-Knipe, Mary & Grondin, Danielle 2003.`Sexual health and mobile and migrant populations´, Sexual Health Exchange 2003/2, Editorial: 1-5, http://www.kit.nl/exchange, 30.10.2004.

Hoeffler, Anke, 1999. `Challenges of infrastructure rehabilitation and reconstruction in war-affected economies´, background paper for the African Development Bank Report, REP/2000-2.

Hope, Kempe R., 2000. ` Mobile workers and HIV / AIDS in Botswana´, AIDS Analysis Africa, 10(4):6-7.

House, James S. & Williams, David R., 1991.`Stress, social support, control and coping: a social epidemiological view´, in `health promotion research - towards a new social epidemiology´, Badura, Bernhard & Kickbusch, Ilona (eds.), 1991: 147-173, WHO Regional Publications, European Series No. 37, WHO Regional Office for Europe, Copenhagen.

Hsu, Lee-Nah & du Guerny, Jaques, 2003. `Crossing national and sectoral boundaries in HIV/AIDS strategies - experience from South East Asia´, Sexual Health Exchange 2003/2, Special Article: 12-13, http://www.kit.nl/exchange, 30.10.2004.

Hynes, M. & Sheik, M. & Wilson, H. G. & Spiegel, P., 2002.`Reproductive health indicators and outcomes among refugee and internally displaced persons in postemergency phase camps´, Journal of the American Medical Association (JAMA), Vol. 288 (5): 595-603.

Johnson, Leigh 2003.`An introduction to the epidemiology of HIV/AIDS´, Research Paper, Centre for Actuarial Research, University of Cape Town, February 2003, available at http://www.commerce.uct.ac.za/care/Research/PAPERS/EpidemiologyIntro.doc, 29.10.2004.

Jones, Christine, 1998. `Does structural adjustment cause AIDS? One more look at the link between adjustment, growth, and poverty´, in `confronting AIDS: evidence from the developing world´, Ainsworth, M. & Fransen, L. & Over, M.(eds.) 1998: 53-65, World Bank background paper, The World Bank, Washington, DC.

Kark, Sidney L., 1946. `The social pathology of syphilis in Africans´, South African Medical Journal, Vol. 23: 77-84.

Kawachi, Ichiro, 2000.`Income inequality and health´, in `Social Epidemiology´, Berkman, Lisa & Kawachi, Ichiro (eds.) 2000: 76-94, Oxford University Press, New York.

Kawachi, Ichiro & Berkman, Lisa 2000.`Social cohesion, social capital, and health´, in `Social Epidemiology´, Berkman, Lisa & Kawachi, Ichiro (eds.) 2000: 174-190, Oxford University Press, New York.

Kirk, Paul, 2000. `Sixty percent of army may be HIV-positive´, The Mail & Guardian, March 31st of 2000, http://www.aegis.com/news/dmg/2000/MG000312.html, 11.3.2004.

Kohler, Ulrich & Kreuter, Frauke, 2001.`Datenanalyse mit Stata: allgemeine Konzepte der Datenanalyse und ihre praktische Anwendung´, München/Wien: R. Oldenbourg Verlag.

Krieger, Nancy, 2001.`Theories for social epidemiology in the 21st century: an ecosocial perspective´, International Journal of Epidemiology, Vol. 30: 668-677.

Lagarde, E. & Carael, M. & Glynn, J. & Kanhonou, L. & Abega, S.-C.& Kahindo, M. & Musonda, R. & Auvert, B. & Buvé, A., 2001.`Educational level is associated with condom use within non-spousal partnerships in four cities of sub-Saharan Africa´, AIDS, 2001, Vol. 15: 1399-1408.

Leaning, Jennifer, 2003. `Public health, war and terrorism´, Harvard Health Policy Issue, Vol. 4 (1), http://www.hhpr.org/currentissue/leaning.php, 4.12.2003.

Levin, B.R. & Bull, J.J. & Stewart, F. M., 2001.`Epidemiology, evolution, and future of the HIV/AIDS pandemic´, Emerging Infectious Diseases, Vol. 7 (Supplement 3): 505-511.

Lowndes, C.M. & Alary, M. & Meda, H. & Gnintoungné, C. A. B. & Mukenge-Tshibaka, L. & Adjovi, C. & Buvé, A. & Morison, M. & Kanhonou, L. & Anagonou, S., 2002.`Role of core bridging groups in the transmission dynamics of HIV and STIs in Cotonou, Benin, West Africa´, Sexual Transmitted Infections, Vol. 78 (supplement 1): 69-77.

Mabey, Davis & Mayaud, Philippe, 1997. `Sexually transmitted diseases in mobile populations´, Genitourinary Medicine, Vol. 73 (1): 18-22.

Machel, Graca, 2001. `Conflict fuels HIV/AIDS crisis´, SHAAN online: IPS e-zine on gender and human rights, http://www.ipsnews.net/hivaids/section1_2.shtml, 4.12.2003.

Machel, Graca, 2000. `The impact of armed conflict on children´, http://unifem.undp.org/machelrep.pdf, 1.6.2001.

Machel, Graca, 1996. `The impact of armed conflict on children´, report of the expert of the Secretary-General, document submitted to the fifty-first Session of the General Assembly, 48/157, August 26th of 1996.

Macintyre, Sally & Ellaway, Anne, 2000.`Ecological approaches: rediscovering the role of the physical and social environment´, in `Social Epidemiology´, Berkman, Lisa & Kawachi, Ichiro (eds.) 2000: 332-348, Oxford University Press, New York.

MacQueen, Graeme & Santa-Barbara, Joanna, 2000.`Conflict and health: peace building through health initiatives´, British Medical Journal (BMJ), Vol. 321: 293-29, http://www.bjm.com, 16.10.2004.

Mahal, Ajay, 2001. `The human development roots of HIV and implications for policy: a cross-country analysis´, Journal of Health & Population in Developing Countries, Vol. 4 (1): 43-60.

Mann, Jonathan, 1999.`The future of the global AIDS movement´, Harvard AIDS Review, Spring 1999.

Manning, Ryann, 2002. `AIDS and democracy: what do we know? a literate review´, prepared for `AIDS and democracy: setting the research agenda´, a workshop held in Cape Town, April 22nd – 23rd of 2002.

Manning, R. & Caesar, M. & Willan, S., 2002. `Proceedings of AIDS and democracy in Southern Africa: setting the research agenda: a workshop held in Cape Town, April 22nd – 23rd of 2002´, http://www.nu.ac.za/heard/workshopsreports/AIDS%20and%20Democracy%20Workshop%20Proceedings.pdf, 24.10.2004.

Mbori-Ngacha, D. & Nduati, R. & John, G. & Reilly, Me & Richardson, B. & Mwatha, A. & Ndinya-Achola, J. & Bwayo, J. & Kreiss, J., 2001.`Morbidity and mortality in breastfed and formula-fed infants of HIV-1-infected women´, Journal of the American Medical Association (JAMA), Vol. 286 (19): 2413-2420.

McGinn, T. & Purdin, S.J. & Krause, S. & Jones, R.K., 2001. `Forced migration and transmission of HIV and other sexually transmitted infections: policy and programmatic responses´, HIV InSite Knowledge Base Chapter, Center for HIV Information, available at http://www.hivinsite.ucsf.edu/InSite?page=kb-08-01-08#S2X, 10.3.2004.

McGinn, Theresa, 2000. `Reproductive health of war-affected populations: what do we know?´, International Family Planning Perspectives, Vol. 26, (4): 174-180, http://www.agi-usa.org/pubs/journals/2617400.html, 12.3.2004.

McKinley, 1998.`Ravaged by war and massacre, Rwanda faces scourge of AIDS´, The New York Times, May 28th of 1998.

Mills, Anne & Shillcut, Sam, 2004.`Communicable diseases´, in `global crisis, global solutions´, Bjorn Lomborg (ed.) 2004: 62-114, Cambridge University Press, New York.

Mock, Nancy & Mathys, Ellen, 2002.`Conflict and HIV: a framework for risk assessment to prevent HIV in conflict settings´, PowerPoint presentation, International Studies Association, Tulane University, New Orleans, http://www.certi.org/strategy/hiv_conflict/conflict_and_hiv.htm, 5.12.2003.

Murray, Ch.J.L. & King, G. & Lopez, A.D. & Tomijima, N. & Krug, E. G., 2002.`Armed conflict as a public health problem´, British Medical Journal (BJM), Vol. 321 (9): 346-349, http://www.bjm.com, 3.12.2003.

Myer, L. & Morroni, Ch. & Susser, E.S., 2003. `Commentary: the social pathology of the HIV/AIDS pandemic´, International Journal of Epidemiology, Vol. 32: 189-192.

Myer, Landon, 2002. `The Jaipur Paradigm´, South African Medical Journal, February 2002, Vol. 92 (2): 98.

Nanayakkara, Atula & Guy, Samantha, 2003. `Reproductive health services for internally displaced people in Sri Lanka´, Sexual Health Exchange 2003/2, Programme Feature: 15-16, http://www.kit.nl/exchange, 30.10.2004.

O'Sullivan, Sue, 2000. `Uniting across boundaries - HIV positive women in global perspective´, Agenda no. 44, http://www.agenda.org.za/sue.htm, 20.10.2004.

Ouattara, Mama, 2004. `HIV infection and economic growth´, The Journal of Policy Reform, September 2004, Vol. 7(3): 141-149.

Over, Mead, 1998.`The effects of societal variables on urban rates of HIV infection in developing countries: an exploratory analysis´ in `confronting AIDS: Evidence from the Developing World´, Ainsworth, M. & Fransen, L. & Over, M. (eds.) 1998: 39-51, World Bank background paper, The World Bank, Washington, DC.

Poundstone, K.E. & Strathdee, S.A. & Celentano, D.D., 2004.`The social epidemiology of Human Immunodeficiency Virus/Acquired Immunodeficiency Syndrome´, Epidemiologic Reviews, Vol. 26: 22-35.

Pyne-Mercier, Lee, 2001. `Corridors of hope in Southern Africa: HIV prevention needs and opportunities in four border towns´, AIDS Analysis Africa, Vol. 11(6): 4, http://www.und.ac.za/und/heard/AAA/AAA.htm, 14.12.2003.

Quigley, M. A. & Morgan, D. & Malamba, S.S. & Mayanja, B. & Okongo, M.J. & Carpenter, L. M. & Whitworth, J. A., 2000.`Case-control study of risk factors for incident HIV infection in rural Uganda´, Journal of Acquired Immune Deficiency Syndromes (JAIDS), Vol. 23 (5): 418.425.

Renaud, Tamar, 2001. `HIV/AIDS and children affected by armed conflict: we are too busy with the war to be worried about HIV/AIDS´, UNICEF, April 2001, New York, http://home.tiscali.nl/~xp115801/my_project/docs/Armed%20Conflict-HIV.doc, 19.11.2004

Rey, J-L. & Milleliri, J-M. & Soares, J-L. & Boutin, J-P. & Desfontaine, M. & Merouze, F. & Van de Perre, P., 1995. `HIV seropositivity and cholera in refugee children from Rwanda´, AIDS 9(10): 1203-1204.

Salama, P. & Laurence, B. & Nolan, M. L., 1999. `Health and human rights in contemporary humanitarian crisis: is Kosovo more important than Sierra Leone? ´, British Medical Journal (BMJ), Vol. 319: 1569-1571.

Santos-Ferreira, M.O. & Cohen, T. & Lourenco, M.H. & Almeida, M.J. & Chamaret, S. & Montagnier, L. 1990.`A study of seroprevalence of HIV-1 and HIV-2 in six provinces of People's Republic of Angola: clues to the spread of HIV infection´, Journal of Acquired Immune Deficiency Syndromes (JAIDS), Vol. 3 (8): 780-786.

Shanks, Leslie & Schull, Michael, J., 2000. `Rape in war: the humanitarian response´, Journal of the American Medical Association (JAMA), Vol. 163 (9): 1152-1156.

Sharma, Nirmala 2003. `HIV/AIDS awareness for migrant communities in the hills of Nepal´, Sexual Health Exchange 2003/2, Program Feature: 9-10, http://www.kit.nl/exchange, 30.10.2004.

Scherrer, Paul, 2000.`UN Report on AIDS paints picture of devastation - part 1´, World Socialist Web Site, July 17th of 2000, http://www.wsws.org/articles/2000/jul2000/un1-j17_prn.shtml, 21.10.2004.

Schnell, Rainer & Hill, Paul B. & Esser, Elke, 1999.`Methoden der Empirischen Sozialforschung´, R. Oldenbourg Verlag, München Wien.

Schnell, Rainer, 1994. `Graphisch gestützte Datenanalyse´, Oldenbourg, München/Wien.

Schönteich, Martin, 1999. `AIDS and age: South Africa's crime time bomb´, AIDS Analysis Africa, Vol. 10 (2): 1, 3, 4, http://www.und.ac.za/und/heard/AAA/aids10-2.pdf, 1.6.2001.

Schowengerdt, A. & Spiegel, P. & Spielberg, F., 1998. `Health interventions in complex emergencies - a case study of Liberia´, CERTI Publication, http://www.certi.org/publications/case_study/Liberiaframe.htm, 4.12. 2003.

Shell, Robert C.H. (2002) `Trojan Horses: HIV/AIDS and military bases in Southern Africa´, http://www.science.uwc.ac.za/stats/research/16_trojan.pdf, 10.3.2004.

Shell, Robert C.H. (2000). `Halfway to the Holocaust: the economic, demographic and social implications of the AIDS pandemic to the year 2010 in the Southern African region´, in `HIV/AIDS: a threat to the African Renaissance? `, a compilation of articles published by the Konrad Adenauer Stiftung, http://www.kas.org.za/Publications/OccasionalPapers/Aids/Robshell2.pdf, 24.10.2004.

Shell, Robert C.H. & Zeitlin, Rebecca 2000.`Positive outcomes: the chances of acquiring HIV/AIDS during the school-going years in the Eastern Cape, 1990-2000´, The Social Work Practitioner Researcher, December 2000, Vol. 12 (3): 139-154 , paper originally presented to the South African Epidemiological Conference, East London, February 2000, to IIED Forum (UNESCO), Paris, September 26th of 2000, and to UWC Educational Faculty, Tuesday, November 13th of 2001, http://www.science.uwc.ac.za/stats/research/26_education.pdf, 20.10.2004.

Smallman-Raynor, Matthew R. & Cliff, Andrew D., 1991. `Civil war and the spread of AIDS in Central Africa´, Epidemiology and Infection, Vol. 107 (1): 69-80.

Smith, Ann, 2002. `HIV/AIDS and emergencies: analysis and recommendations for practice´, Network Paper No. 38, 2002, Humanitarian Practice Network (HPN), Overseas Development Institute (ODI), London.

Spiegel, Paul & Nankoe, Alia, 2004. `UNHCR, HIV/AIDS and refugees: lessons learned´, Forced Migration Review, Vol. 19: 21-23.

Spiegel, Paul, 2004. `HIV/AIDS among conflict-affected and displaced populations: dispelling myths and taking action´, Disasters, Vol. 28 (3): 322-339.

Stillwaggon, Eileen, 2001. `AIDS and poverty in Africa: prevention and treatment require a focus on overall health and development´, The Nation, May 21st of 2001:22, http://www.comminit.com/TheDrumRole/sld-1992.html, 13.12.2003.

Subramanian, KS, 2002. `Impact of conflict on HIV/AIDS in South Asia´, background paper, August 2002, http://www.sasnet.lu.se/southasiahiv.pdf, 13.11.2004.

Subramanian, S.V. & Belli, P. & Kawachi, I., 2002. `The macroeconomic determinants of health´, Annual Review of Public Health, Vol. 23: 287-302.

Swiss, Shana, 1999.`Rape and sexual abuse in armed conflict and political violence´, briefing note `gender, armed conflict and political violence, The World Bank, http://www.worldbank.org/gender/events/Swiss.doc, 5.12.2003.

Swiss, S. & Jennings, P. J. & Aryee, G. V. & Brown, G. H. & Jappah-Samukai, R. M. & Kamara, M. S. & Schaack, R.D.H. & Turay-Kanneh, R.S., 1998.`Violence against women during the Liberian civil conflict´, Journal of the American Medical Association (JAMA), Vol. 279: 625-629.

Swiss, Shana & Giller, Joan E., 1993. `Rape as a crime of war´, Journal of the American Medical Association (JAMA), Vol. 270 (5): 612-615.

Toole, Michael, 2000.`Displaced persons and war´, in `War and public health´, Levy, Barry S. & Sidel, Victor W. (eds.), 1997: 197-212, Oxford University Press, New York and Oxford.

Toole, Michael J. & Waldman, Ronald J., 1993. `Refugees and displaced persons´, Journal of the American Medical Association (JAMA), Vol. 270 (5): 600-605.

Ursin, Holger 1991. `Psychobiology of stress and attachment: the biobehavioural view´, in `health promotion research - towards a new social epidemiology 1991: 173-187, Badura, Bernhard & Kickbusch, Ilona (eds.), WHO Regional Publications, European Series No. 37, WHO Regional Office for Europe, Copenhagen.

VanBeelen, Neil 2003. `HIV/AIDS and the military: fighting the war against HIV/STIs´, Sexual Health Exchange 2003/2, Special Article: 6-8, http://www.kit.nl/exchange, 30.10.2004.

Van der Gaag, Jaques, 2004.`Communicable diseases: alternative perspectives´, perspective paper 2.2 in `global crisis, global solutions´, Bjorn Lomborg (ed.) 2004: 124-128, Cambridge University Press, New York.

Van der Heijden, Thomas, 1997. `Health care between war and peace - an exploration of issues and strategies´, HealthNet International, discussion paper 1, June 15th of 1997, http://www.who.int/disasters/hbp/general/Documents/healthbetweenwarandpeace.htm, 4.12.2003.

VanLandingham, M. J. & Suprasert, S. & Sittitrai, W. & Vaddhanaphuti, Ch. & Grandjean, N., 1993. `Sexual Activity among never-married men in Northern Thailand´, Demography, Vol. 30 (3): 297-313.

VanRensburg, E.J. & Lemmer, H.R. & Joubert, J.J., 1995. `Prevalence of viral infections in Mozambican refugees in Swaziland´, East African Medical Journal, Vol. 72 (9): 588-590.

Vlahov, David & Galea, Sandro, 2002. `Urbanization, urbanicity, and health´, Journal of Urban Health: Bulletin of the New York Academy of Medicine, 79 (4, Supplement 1): 1-12.

Walker, N. & Grassly, N.C., Garnett, G.P. & Stanecki, K.A. & Ghys, P.D., 2004. `Estimating the global burden of HIV/AIDS: what do we really know about the HIV pandemic? The Lancet, Vol. 363, : 2180-2185.

Walker, N. & Stover, J. & Stanecki, K.A. & Zaniewski, A.E. & Grassly, N.C., & Garcia-Calleja, J.M. & Ghys, P.D., 2004.`The workbook approach to making estimates and projecting future scenarios of HIV/AIDS in countries with low level and concentrated epidemics´, Sexually Transmitted Infections, Vol. 80 (Supplement I): i10-i13.

Ward, Jeanne & Brewer, Jessica, 2004. `Gender-based violence in conflict-affected settings: overview of a multi-country research project´, Forced Migration Review, Vol. 19: 26-29.

Wax, Emily, 2001.`A brutal legacy of Congo war: extent of violence against women surfaces as fighting recedes´, Washington Post Foreign Service, Saturday, October 25th of 2003: A01, http://www.washingtonpost.com/ac2/wp-dyn?pagename=article&contentId=A14059-2003Oct24&notFound=true, 19.11.2004.

Weiss, H.A. & Quigley, M.A. & Hayes, R.J., 2000. `Male circumcision and risk of HIV infection in sub-Saharan Africa: a systematic review and meta-analysis´, AIDS (14): 2361-2370.

Whiteside, A. & Hickey, A. & Ngcobo, N. & Tomlinson, J., 2003. `What is driving the HIV/AIDS epidemic in Swaziland, and what more can we do about it? final report prepared for the National Emergency Response Committee on HIV/AIDS (NERCHA) and United Nations Programme on HIV/AIDS (UNAIDS), April 2003, http://www.sarpn.org.za/documents/d0000706/index.php, 31.12.2004.

Williams, Ada & Martin, Veronika, 2002. `The next war in Angola: combating HIV/AIDS´, Refugees International online Bulletin, 07/30/2002, http://www.refugeesinternational.org.cgi-bin/ri/bulletin?bc=00433, 25.6.2004.

Wollants, E. & Schoenenberg, M. & Figueroa, C. & Shor-Posner, G. & Klaskala, W. & Baum, M.K., 1995.`Risk factors and patterns of HIV-1 transmission in the El Salvador military during war time´, AIDS, 9 (11): 129-130.

Zierler, Sally & Krieger, Nancy, 1997. `Reframing women´s risk: social inequalities and HIV infection´, Annual Review of Public Health, Vol. 18: 401-436.

Reports:

Amnesty International (AI), April 05th of 2004. `Rwanda: marked for death: rape survivors living with HIV/AIDS in Rwanda´, AI Index: AFR 47/007/2004. http://www.reliefweb.int/library/documents/2004/ai-rwa-06apr.pdf, 15.1.2005.

British Broadcasting Company (BBC) News, July 26th of 2003. `AIDS `economic catastrophe´ looms´, http://news.bbc.co.uk/go/pr/fr/-/2/hi/business/3089227.stm, 26.6.2003.

Central Intelligence Agency (CIA), 2003. `World Factbook 2003´, http://www.cia.gov/cia/download.html, 15.7.2003.

Fischer Weltalmanach, 2001.`Zahlen, Daten, Fakten 2001´, von Baratta, Mario (ed.), Fischer Taschenbuchverlag, Frankfurt am Main 2001.

Government of South Africa, 2001.`Presidential AIDS Advisory Panel Report: a synthesis report of the deliberations by the panel of experts invited by the President of the Republic of South Africa, the Honourable Mr. Thabo Mbeki, March 2001 (the international panel met on two occasions in Pretoria (May 6th - 7th of 2000) and in Johannesburg (July 3rd – 4th of 2000)). http://www.polity.org.za/html/govdocs/reports/aids/aidspanel.htm, 15.1.2005.

Human Rights Watch, 1995. `Rape in Somalia´,
http://www.hrw.org/about/projects/womrep/General-28.htm, 16.11.2004

Interagency Coalition on AIDS and Development (ICAD), August 2001. `HIV/AIDS and displaced people´,
http://www.icad-cisd.com/pdf/publications/e_displaced.pdf, 11.3.2004.

International Crisis Group (ICG), April 2nd of 2002. `Myanmar: the HIV/AIDS crisis´, International Crisis Group Asia, Briefing, Bangkok/Brussels,
http://www.crisisweb.org//library/documents/report_archive/A400601_02042002.pdf, 11.3.2004.

International Crisis Group (ICG), June 19th of 2001. `HIV/AIDS as a security issue´, International Crisis Group, Washington/Brussels,
http://www.crisisweb.org/home/index.cfm?id=1831&l=1, 3.12.2003.

International Organization for Migration (IOM), 2003. `Mobile populations and HIV/AIDS in the Southern African region: recommendations for action: desk review and bibliography on HIV/AIDS and mobile populations, May 2003,
http://www.sarpn.org.za/documents/d0000365/index.php, 31.12.2004.

Panos Institute, 1992. `Panos Dossier: the hidden cost of AIDS: the challenge of HIV to development´, The Panos Institute, London: Panos Publications Ltd.

Physicians for Human Rights, 2002. `War-related sexual violence in Sierra Leone: a population-based assessment, Physicians for Human Rights, Boston, Washington DC 2002,
http://www.phrusa.org/research/sierra_leone/report.html, 11.3.2004.

Stockholm International Peace Research Institute (SIPRI) 2004. `Yearbook 2004: armament, disarmament and international security´.
http://editors.sipri.se/pubs/yb04/about01.html, 15.1.1005.

The Economist, May 24th of 2003. `Special report civil wars: the global menace of local strife´, 367 (8325):23-25.

United Nations Programme on HIV/AIDS (UNAIDS), August 2004. `Questions & Answers II: basic facts about the AIDS epidemic and its impact´,
http://www.unaids.org/Unaids/EN/Resources/questions_answers.asp#II, 11.11.2004.

United Nations Programme on HIV/AIDS (UNAIDS), July 2002. `Fact Sheet 1: HIV/AIDS and security´.
http://www.unaids.org/en/in+focus/topic+areas.asp, 1.6.2004.

United Nations Programme on HIV/AIDS (UNAIDS), July 2002. ` Fact Sheet 2: HIV/AIDS and conflict´.
http://www.unaids.org/en/in+focus/topic+areas.asp, 1.6.2004.

United Nations Programme on HIV/AIDS (UNAIDS), July 2002. `Fact Sheet 3: HIV/AIDS and uniformed services´.
http://www.unaids.org/en/in+focus/topic+areas.asp, 1.6.2004.

United Nations Programme on HIV/AIDS (UNAIDS), July 2002. `Fact Sheet 4: HIV/AIDS and peacekeeping´.
http://www.unaids.org/en/in+focus/topic+areas.asp, 1.6.2004.

United Nations Programme on HIV/AIDS (UNAIDS), 2001. `Together we can: leadership in a world of AIDS´.
http://www.unaids.org/Unaids/EN/In+focus/Topic+areas/Leadership.asp, 15.1.2005.

United Nations Programme on HIV/AIDS (UNAIDS), 1998. `UNAIDS Point of View: AIDS and the military´, May.
http://www.unaids.org/en/in+focus/topic+areas.asp, 1.6.2004.

United Nations Programme on HIV/AIDS (UNAIDS), 1997. `Point of View: refugees and AIDS´, April.
http://www.unaids.org/en/in+focus/topic+areas.asp, 15.1.2005

United Nations Programme on HIV/AIDS (UNAIDS), 1997. `Technical Update: refugees and AIDS´, September.
http://www.unaids.org/html/pub/publications/irc-pub04/refug-tu_en_pdf.pdf

United Nations Programme on HIV/AIDS (UNAIDS), December 2004. `AIDS Epidemic Update-December 2004´,
http://www.unaids.org/wad2004/EPI_1204_pdf_en/EpiUpdate04_en.pdf, 27.1.2005.

United Nations Programme on HIV/AIDS/World Health Organization (UNAIDS/WHO), July 2004. `Epidemiological Fact Sheet- 2004 Update: Mozambique´.
http://www.who.int/emc-hiv/fact_sheets/All_countries.html, 15.1.005.

United Nations Programme on HIV/AIDS/World Health Organization (UNAIDS/WHO), 2004. `Report on the Global AIDS Epidemic´.
http://www.unaids.org/bangkok2004/report.html, 15.1.2005.

United Nations Programme on HIV/AIDS/World Health Organization (UNAIDS/WHO), 2002. `Report on the Global HIV/AIDS Epidemic 2002´,
http://www.unaids.org/EN/resources/publications/corporate+publications/report+on+the+global+hiv_aids+epidemic+2002+.asp, 19.12.2004.

United Nations Programme on HIV/AIDS/World Health Organization (UNAIDS/WHO), 2002. `AIDS Epidemic Update – December 2002´,
http://www.unaids.org/html/pub/Publications/IRC-pub03/epiupdate2002_en_pdf.pdf, 15.1 2005.

United Nations Programme on HIV/AIDS/World Health Organization (UNAIDS/WHO) Working Group on Global HIV/AIDS/STI Surveillance, 2000. `Guidelines for second generation HIV surveillance´, WHO/CDS/CSR/EDC/2000.5-UNAIDS/00.03,
http://www.who.int/hiv/pub/surveillance/en/cds_edc_2000_5.pdf, 2.1.2005

United Nations Development Programme (UNDP), 2004. `Human Development Report 2004´,
http://hdr.undp.org/reports/global/2004, 17.8.2004.

United Nations Economic Commission for Africa (UNECA), 2000. ` Lessons Africa has learnt in 15 years of responding to HIV/AIDS´, working paper, African Development Forum 2000, http://www.uneca.org/adf2000/theme2making.htm, 3.12.2003.

United Nations High Commissioner for Refugees (UNHCR)/Save the Children-UK, 2002. `Note for implementing and operational partners on sexual violence and exploitation: the experience of refugee children in Liberia, Guinea and Sierra Leone´, http://www.db.idpproject.org/Sites/idpSurvey.nsf/wViewCountries/0E391018D7DCD5BDC1256C39002AFC4D, 5.12.2003.

United Nations Children's Fund (UNICEF), July 2002. `HIV/AIDS and children affected by armed conflict´.
http://www.unicef.org/publications/pub_hiv_armedconflict_en.pdf, 15.1.2005.

United Nations (U.N.), December 2004. `A more secure world: our shared responsibility´, report of the high-level panel on threats, challenges and change, United Nations Department of Public Information, New York,
http://www.un.org/secureworld/report2.pdf, 20.12.2004

United Nations (U.N.), 2001. `Fact Sheet: global crisis - global action: AIDS as a security issue´, prepared for the United Nations Special Session on HIV/AIDS, June 25th – 27th of 2001, New York, http://www.un.org/ga/aids/ungassfactsheets/pdf/fssecurity_en.pdf, 15.10.2004.

United Nations Population Division (UNPD), Department of Economic and Social Affairs, 2003. ` World Urbanization Prospects: the 2003 revision´,
http://www.urbancentre.utoronto.ca/pdfs/elibrary/UN_World_Urbanization_Trend.pdf, 15.7.2004.

United Nations Population Division (UNPD), Department of Economic and Social Affairs, 2002. ` World Population Prospects: the 2002 revision´,
http://www.un.org/esa/population/publications/wpp2002/wpp2002annextables.PDF, 19.12.2004.

United Nations (U.N.) Press Release AFR/640 OP/865, June 6th of 2003. `Escalating conflict in Liberia threatens health of millions across West Africa, UNFPA warns´,
http://www.un.org/News/Press/docs/2003/afr640.doc.htm (4.12.3003).

United Nations (U.N.) Statistics Division, 2004. `Millennium Indicators´,
http://millenniumindicators.un.org/unsd/mi/mi_goals.asp, 10.7.2004.

UNWire, July 26th of 1999. `Health: conflict helps limit spread of HIV/AIDS´,
http://www.unfoundation.org/unwire/, 15.8.2001.

United States Agency for International Development (USAID), 1999. `Angola and HIV/AIDS´,
http://63.107.122.20/documents/738_Angola.PDF, 13.12.2003

United States (U.S.) General Accounting Office, 2001. ` HIV/AIDS and U.N. Peacekeeping: United Nations faces challenges in responding to the impact of HIV/AIDS on peacekeeping operations´, report to the Chairman, Committee on International Relations, House of Representatives: 24-27, December 2001 (GAO-02-194),
http://www.usip.org/pubs/specialreports/sr75.html, 3.12.2003.

United States Institute of Peace (USIP), 2001. `Special report AIDS and violent conflict in Africa´,
http://www.usip.org/pubs/reports.html, 15.09.2003

United States Institute of Peace (USIP), 2001. `Plague upon plague: AIDS and violent conflict in Africa´, current issues briefing transcript, United States Institute of Peace,
http://www.usip.org, 3.12.2003.

United States National Institute of Health, National Cancer Institute, 2003. `Theory at a glance: a guide for health promotion practice´,
http://cancer.gov/aboutnci/oc/theory-at-a-glance/page3, 4.1.2005.

World Bank, 2004. `World Development Indicators 2004´,
http://www.worldbank.org/data/wdi2004/tables/table1-6.pdf, 15.7.2004.

World Health Organization (WHO), 2004. `World Health Report 2004´.
http://www.who.int/whr/2004/en/09_annexes_en.pdf, 15.7.2004.

World Health Organization (WHO), 2000. `World Health Report 2000´,
http://www.who.int/whr/previous/en/, 15.1.2005.

Online Sources:

http://www.stat.ufl.edu/~winner/mar5621/mar5621.doc, 28.1.2005.

http://www.nd.edu/~rwilliam/stats2/l11.pdf, 28.1.2005;

http://www.fordham.edu/economics/vinod/multiple-regression.doc, 28.1.2005.

http://www.unaids.org/Unaids/EN/Resources/Epidemiology/, 25.1.2005.

http://www.reliefweb.int/w/RWB.NSF/0/7B5CA92DF08CC0F085256E1A006F98A9?OpenDocument, 6.1.2005.

http://apus.wiwi.hu-berlin.de/~sigbert/diplom/zanter/node10.html, 3.1.2005.

http://www.sowi.uni-mannheim.de/lehrstuehle/lessm/veranst/stat1/folien/folien_5.pdf, 3.1.2005.

http://www.politik.uni-mainz.de/kai.arzheimer/Lehre-Stata/Deskriptive-Dateien/frame.html#slide0001.html, 3.1.2005.

http://www.belarus-botschaft.de/de/presse41_2002_de.htm, 18.12.2004.

http://www.staatenderwelt.de/staaten/estland/estland.htm, 18.12.2004.

http://www.swasiland.de/c_swazis.asp?content=sprache_religion.html, 18.12.2004.

http://www.vietnam-freunde.net/seite01/html/religion___glauben_in_vietnam.html, 18.12.2004.

http://www.auswaertiges-amt.de/www/de/laenderinfos/laender/laender_ausgabe_html?land_id=190&type_id=2, 18.12.2004.

http://www.itl.nist.gov/div898/handbook/pmc/section5/pmc52.htm, 7.12.2004.

http://gsbwww.uchicago.edu/computing/research/SASManual/qc/chape/sect11.htm, 7.12.2004.

http://www.unaids.org/Unaids/EN/Resources/Epidemiology/EPI_SoftwareTools.asp, 6.12.2004.

http://www.unwire.org/UNWire/20031027/449_9787.asp, 19.11.2004.

http://www.comminit.com/hivaids/change_theories.html, 29.10.2004.

http://www.ticino.com/usr/opadlina/new/ph/epi-ges.htm, 29.10.2004.

http://www.etr.org/recapp/theories/hbm/Resources.htm, 29.10.2004.

http://www.tcw.utwente.nl/theorieenoverzicht/Theory%20Clusters/Health%20Communication/Health_Belief_Model.doc/, 29.10.2004.

http://www.icaso.org/docs/newsletters/january99.html, 24.10.2004.

http://www.rnw.nl/humanrights/zimbabwe/html/jaipur.html, 24.10.2004.

http://www.rnw.nl/humanrights/zimbabwe/html/whiteside.html, 24.10.2004.

http://www.icaso.org/docs/newsletters/january99.html, 20.10.2004.

http://www.iss.co.za/Pubs/ASR/10No4/Fourie.html, 19.10.2004.

http://www.worldbank.org/data/wdi2004/tables/table1-6.pdf, 15.7.2004.

http://www2.chass.ncsu.edu/garson/pa765/regress/htm, 22.8.2004.

http://millenniumindicators.un.org/unsd/mi/mi_goals.asp, 10.7.2004.

http://millenniumindicators.un.org/unsd/mi/mi_worldregn.asp, 10.7.2004.

http://www.worldbank.org/aids-econ/toolkit/intro.htm, 25.6.2004.

http://www.ifst.org/aids.htm, 25.6.2004.

http://www.rho.org/html/hiv_aids_overview.htm, 25.6.2004.

http://www.worldbank.org/data/countryclass/classgroups.htm, 13.7.2003.

http://www.unaids.org/security/Issues/conflict.html, 16.6.2003.

http://www.smh.com.au/news/specials/intl/aids/aids16.html, 1.6.2001.