Fertility trends in sub-Saharan Africa: Has HIV contributed to fertility stalls?

—Preliminary version—

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Abstract

Fertility transition in sub-Saharan Africa has been different than elsewhere, showing slow pace, halts and reversals in fertility decline since the early 2000s. Likewise, it is the region of the world hardest hit by HIV. HIV is associated with both fertility-enhancing and fertility-reducing behaviors, and the net effect on fertility depends on which effect dominates. Our goal is to better understand the mechanisms through which HIV may have affected fertility changes over the last decades and whether they are connected to fertility stalls. Our analyses are conducted at three levels, regional, cross-country, and country level, focusing on countries with strong and moderate evidence of fertility stalls. We use information from the World Bank data repository, UNAIDS, and the Demographic and Health Surveys. We reconstruct fertility rates according to HIV status and analyze changes over time. Our results suggest that unambiguous cases of fertility stalls are mostly detected in countries of high HIV prevalence. In these countries, there was an unusually rapid decline in fertility during the 1990s and then a slowdown beginning in the mid-2000s. Fertility of HIV-positive women has contributed to fertility stalls, but it is the fertility of HIV-negative women that explains them to a greater extent. In Zimbabwe, fertility-enhancing behaviors during the stall period, such as increased sexual activity, low rates of contraceptive use, reduced length of postpartum infecundability, and high demand for children, together with high rates of antiretroviral treatment among pregnant women, have counter-balanced fertility-reducing behaviors.

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Introduction

Reports identify the first cases of infections by Human Immunodeficiency virus (HIV) and deaths by Acquired Immunodeficiency Syndrome (AIDS) between the mid-1970s and the early-1980s, formerly recognized as cases of a rare lung infection, Pneumocystis carinii pneumonia, and an unusually aggressive cancer, Kaposi's Sarcoma (Centers for Disease Control, 1981; Hymes et al., 1981). By the late 1980s, official reports indicated about 140,000 confirmed AIDS cases in 145 countries, although the World Health Organization (WHO) estimated up to 400,000 cases globally (WHO, 1989). During the 1990s, the number of cases increased rapidly worldwide, especially in Africa, to the point of declaring HIV/AIDS a global epidemic. By the end of the decade, WHO announced that AIDS was the fourth cause of death worldwide and the leading cause in sub-Saharan Africa (SSA): An estimated 33 million people were living with HIV and 14 million people had died from AIDS since the start of the epidemic (WHO, 1999).

From the 2000s, increased access to drugs and advances in research allowed to better treat AIDS and reduce HIV infections, including mother-to-child transmission (UNAIDS, 2017, 2020b). In addition, after including the reversal of the spread of HIV as a specific goal in the United Nations' Millenium Development Goals (MDG), a greater flow of funds was directed to combat the epidemic, especially to SSA. By 2015, the United

Nations announced that the MDG target related to halt and reverse the spread of HIV was met nine months ahead schedule (UNAIDS, 2015). Currently, more than half of the global population living with HIV are receiving antiretroviral treatment (Editorial Lancet HIV, 2017). Also, significant decreases in HIV infections have been registered in SSA, especially among young women and girls (UNAIDS, 2017).

Since HIV is a sexually transmitted disease, it has a direct effect on fertility. On the one hand, HIV is associated with fertility-reducing factors such as less sexual exposure —less sexual activity and more marital dissolution—, increased contraceptive use —especially condom use—, induced abortion, increased secondary infertility, increased incidence of amenorrhea, lower desired fertility, and increased fetal loss (Agyei-Mensah, 2006; Bankole et al., 2011, 2014; Brocklehurst & French, 1998; Chirgwin et al., 1996; Hoffman et al., 2008; Johnson et al., 2009; Magadi & Agwanda, 2010; Moultrie & Timaeus, 2003; Porter et al., 2004). For instance, a comparative study in 12 SSA countries shows that in the 1990s fertility was lower among HIV-infected women than among uninfected women, and that for each percentage point of increase in HIV prevalence, total fertility declined in 0.37% (Lewis et al., 2004). In addition, other studies have found evidence of lower fertility among HIV-infected women (Chen & Walker, 2010; Gemmill et al., 2018; Gregson et al., 2002; Kongnyuy & Wiysonge, 2008; Lewis et al., 2004; Marston et al., 2017; Zaba & Gregson, 1998).

On the other hand, HIV has also been associated with fertility-enhancing behaviors. First, there is evidence of the intervals between births of infected women becoming shorter as breastfeeding may be reduced and, second, the desire to have more children increases in response to increased child mortality —replacement births— (Houle et al., 2016; Magadi & Agwanda, 2010; Westoff & Cross, 2006). Lower access to contraception, as resources may be directed away from Family Planning programs, may also lead to higher fertility (Bongaarts, 2008; Fuchs & Goujon, 2014; Romaniuk, 2011; Ross et al., 2004; Westoff & Cross, 2006). As a result, the lack of access to contraceptives and early initiation of sexual activity have led to a higher risk of infection in adolescents and higher fertility rates among those infected compared to those not infected (Chen & Walker, 2010; Gregson et al., 2002; Lewis et al., 2004; Marston et al., 2017; Zaba & Gregson, 1998). The net fertility outcome would depend on the balance between fertility-enhancing and fertility-reducing behaviors. Thus, a halt in fertility decline could occur in those countries where the positive effects outweigh the negative effects (Magadi & Agwanda, 2010). The effect may also vary over time, partly as a results of changes in access to treatments and risks of infections.

In terms of fertility transition in SSA, the late 1970s and early 1980s saw the first signs of declining fertility rates, and by the late 1990s the fertility transition was ongoing in most SSA countries (Sánchez-Páez & Schoumaker, 2020). However, literature has identified cases of halts and reversals in fertility decline in several countries since the early 2000s (e.g., Bongaarts, 2008; Garenne, 2008; Howse, 2015; Schoumaker, 2019; Shapiro & Gebreselassie, 2008; Westoff & Cross, 2006). However, the list of countries and periods with fertility stalls over the past 20 years vary depending on the method used to define the stall and the source of information (Sánchez-Páez & Schoumaker, 2020; Schoumaker, 2019). Although recent evidence shows that fertility stalls are not as common as thought, they are not as exceptional either (Schoumaker, 2019). In most cases, the stalls have started in the early and mid-2000s (Schoumaker, 2019), coinciding with the democratization of HIV treatments.

Prevention in mother-to-child transmission increases the possibility of bearing more children. This possibility may result in a net increase in fertility. For instance, women infected with HIV in South Africa are increasing their fertility as they now have access to effective treatments (Moultrie et al., 2008). Some studies show that the fertility stalls in rural South Africa (Houle et al., 2016) and in Kenya (Magadi & Agwanda, 2010; Westoff

& Cross, 2006) can partly be explained by replacement births by infected women who have previously lost a child to HIV. Also, in Kenya (Magadi & Agwanda, 2010) and South Africa (Garenne et al., 2007) HIV has contributed to stalled fertility through reduced breastfeeding and birth intervals. On the other hand, a study conducted at the regional level in SSA discusses that the decline in life expectancy of young adults associated with HIV/AIDS may have triggered a drop in investments in education and human capital, leading to fertility stalls in SSA countries with high HIV prevalence, as the trade-off between quantity and quality of children becomes biased towards the former (Gori et al., 2020).

Little research has focused on the role of HIV on fertility stalls in SSA. We find evidence arguing for a contribution of HIV to fertility stagnation in Kenya (Magadi & Agwanda, 2010; Westoff & Cross, 2006) and South Africa (Garenne et al., 2007; Houle et al., 2016), whereas in Ghana the evidence does not support it (Agyei-Mensah, 2006). At the light of the lack of and mixed evidence, our goal is to identify the extent to which HIV has contributed to fertility stalls in SSA countries. We aim to better understand the mechanisms through which HIV may have affected fertility changes over the last decades and whether they are connected to fertility stalls. By using macro- and micro-level data, we analyze fertility and HIV trends, changes in sexual activity, contraceptive use, length of postpartum infecundability, demand for children, and HIV knowledge, focusing on periods of fertility stalls. We examine differences by HIV status to assess whether infected women behave differently from uninfected women.

We combine cross-country analyses and a case study in Zimbabwe to identify possible links between HIV/AIDS and fertility stalls. We find that fertility stalls are more likely to have occurred in countries with high HIV/AIDS prevalence, and that they occur once the prevalence in HIV/AIDS has started decreasing. We argue that the spread of HIV-AIDS first contributed to a more rapid fertility decline, both because of the lower fertility of HIV-positive women and their increasing share in the population, and because of changes in behavior among both HIV-positive and HIV-negative women. Afterwards, with a better knowledge of ways of transmission and prevention, improved knowledge of HIV status, and greater availability of treatments —and the lower risks of mother-to-child transmission—, fertility behavior changes and leads to a slower decrease or an increase in fertility among both HIV-infected and HIV-uninfected women, contributing to fertility stalls. The decrease in the prevalence of HIV/AIDS also means the TFR tils towards the higher fertility of HIV-positive women.

Data and methods

To address our objective, we conduct our analyses at three levels: regional, cross-country, and country-level. At the regional level, we first analyze fertility and HIV trends. We use yearly estimates for SSA of TFR 15-49 from the World Bank Open Data repository (World Bank, 2020) and HIV prevalence among women 15-49 from UNAIDS (UNAIDS, 2020a). We exclude from our analyses countries with a reported HIV prevalence of <0.1. Second, we examine the trends from 37 SSA countries by grouping them according to the evidence of fertility stalls. We use the criteria defined by Schoumaker (2019) to group the countries in strong/moderate evidence and weak/no evidence[1]. Then, we compute the regional weighted average for each group, where

[1]Countries with strong evidence of fertility stalls are Botswana (2006–2018), Cameroon (1998–2011), Congo (2005–2011), Kenya (1998–2003), Namibia (2007–2013), Zambia (2002–2007), and Zimbabwe (2005–2015). Countries with moderate evidence of fertility stalls are Benin (2001–2006), Burkina Faso (1993–1999), Côte d'Ivoire (1999–2012), Gabon (2000–2012), Malawi (2000–2004), Nigeria (1990–2008), Rwanda (2000–2005), South Africa (1998–2016), and Tanzania (1999–2004). Remaining countries are classified as weak or no evidence of fertility stalls. Although Madagascar (1992—1997) has been classified as a

the weights are the total population of women 15-49 (United Nations, 2020) and the number of HIV-infected women 15-49 (UNAIDS, 2020a), respectively.

Table 1: Demographic and Health Surveys (DHS) including HIV testing in countries of evidence of strong and moderate fertility stalls included in the sample.

		Fertility stalls	
Country	DHS with HIV tests	Period	Evidence
Burkina Faso	2003, 2010	1993-1999	Moderate
Cameroon	2004, 2011, 2018	1998-2011	Strong
Côte d'Ivoire	2005, 2011	1999-2012	Moderate
Gabon	2012	2000-2012	Moderate
Kenya	2003, 2008	1998-2003	Strong
Malawi	2004, 2010, 2015	2000-2004	Moderate
Namibia	2013	2007-2013	Strong
Rwanda	2005, 2010, 2014	2000-2005	Moderate
South Africa	2016	1998-2016	Moderate
Tanzania	2007, 2011	1999-2004	Moderate
Zambia	2007, 2013, 2018	2002-2007	Strong
Zimbabwe	2005, 2010, 2015	2005-2015	Strong

At the cross-country level, we focus on the countries with strong and moderate evidence of fertility stalls. We use 25 Demographic and Health Surveys (DHS) (DHS Program, 2020) from 12 countries[2] (see Table 1) to compute fertility rates by HIV status. For doing so, we merge HIV testing records to DHS women records. Then, we use the Stata function `tfr2` (Schoumaker, 2013) to reconstruct fertility trends by HIV status from birth histories going back up to 12 years. Although DHS allow going further back in time, doing so could result in biased TFR estimates, as HIV-positive women who gave birth may no longer be alive at the time of the survey. We use the 12-year cut-off since it is the average time of survival of a HIV-positive person without treatment after seroconversion (May & Ingle, 2011), so by using the 12-year cut-off it is more likely to include most infected women when reconstructing fertility trends even in the scenario in which they would not have been treated to extend their lifespan. Then, we pool all estimates and use locally weighted smoothing (LOESS) to average the reconstructed yearly fertility rates. From the smoothed fertility trends, we analyze the effect of HIV on fertility by using the population-attributable change (PAC) in total fertility (Lewis et al., 2004; Zaba & Gregson, 1998) and TFR ratio, i.e. the ratio of the fertility of HIV-positive women to the fertility of HIV-negative women. Regarding PAC, assuming that TFR of all women in the absence of HIV is approximately the same as that observed in HIV-uninfected women (Lewis et al., 2004), it is computed as

$$PAC = \frac{TFR - TFR_{HIV-}}{TFR_{HIV-}} \tag{1}$$

case of moderate evidence, it has been ruled out from the analysis since HIV prevalence was lower than 0.1 until 2012.

[2]Benin, Botswana, and Nigeria are excluded as their DHS do not include HIV testing. Gabon, and Namibia are only considered for pooled estimations, but excluded from cross-country comparisons, as only one survey is available in each case and the reconstructed country-trends according to HIV status are volatile. Congo is excluded, as it is not possible to calculate fertility rates according to HIV status. Burkina Faso is considered for the pooled analysis, but excluded from the country analysis as its stall period is at an early stage of the fertility transition.

Unfortunately, no country, except one, has DHS with HIV testing collected during the complete period identified as of stalled fertility. Therefore, at the country level we focus on Zimbabwe, for which there is complete information for the period 2005–2015. We present the results for all women and by HIV status, with emphasis on the stall period. We first examine the knowledge of HIV. Then, we analyze the changes in demand for children and fertility. Third, we inspect the changes in sexual activity, contraceptive use, and postpartum infecundability. Fourth, we conduct a decomposition analysis to estimate the proportion to which changes in fertility trends is attributable to fertility changes by HIV status and HIV prevalence:

$$
\begin{aligned}
TFR^{t2} - TFR^{t1} = {} & \left(TFR^{t2}_{HIV+} - TFR^{t1}_{HIV+}\right) \times \left[\frac{p^{t1}_{HIV+} + p^{t2}_{HIV+}}{2}\right] \\
& + \left(TFR^{t2}_{HIV-} - TFR^{t1}_{HIV-}\right) \times \left[\frac{p^{t1}_{HIV-} + p^{t2}_{HIV-}}{2}\right] \\
& + \sum_{i=1}^{K} \left(p^{t2}_{i} - p^{t1}_{i}\right) \times \left[\frac{TFR^{t1}_{i} + TFR^{t2}_{i}}{2}\right]
\end{aligned}
\tag{2}
$$

Change in total fertility (TFR) between time 1 ($t1$) and time 2 ($t2$) is decomposed in three components: fertility changes among HIV-positive women (TFR_{HIV+}), fertility changes among HIV-negative women (TFR_{HIV-}) and changes in the composition of the population (p_i) due to changes in HIV prevalence. We use an indirect approach to estimate the share of the population of women ages 15-49 by HIV status. Since TFR at the country level is a weighted average of TFR by HIV status, it is possible to retrieve the weights from the fertility trends by HIV status. Shares of HIV-positive women (p_{HIV+}) and HIV-negative women (p_{HIV-}) at a given time are computed as

$$
\begin{aligned}
p^{t}_{HIV+} &= \frac{TFR^{t} - TFR^{t}_{HIV-}}{TFR^{t}_{HIV+} - TFR^{t}_{HIV-}} \\
p^{t}_{HIV-} &= 1 - p^{t}_{HIV+}
\end{aligned}
$$

Results

Regional analysis

Figure 1 displays the trends of TFR and HIV prevalence for women aged 15-49 in SSA. Black and blue vertical dashed lines show the highest TFR and HIV prevalence in the trend, respectively. HIV prevalence increased from 2.4% in 1990 to its highest level, 5.9%, in 2002. From there, the trend has smoothly decreased to 5% in 2019. In absolute terms, the number of women of ages 15-49 living with HIV increased from 2.7 million in 1990 to 12.8 million in 2019.

Fertility in SSA has been steadily declining from the early 1980s. Table 2 presents the pace at which TFR has decreased over the past 40 years. At first glance, fertility has declined at a similar rate during the two decades before and after 2000, which is when the highest level of HIV prevalence was reached, at a rate of about one birth per woman over the two decades. However, the pace of decline was greater during the

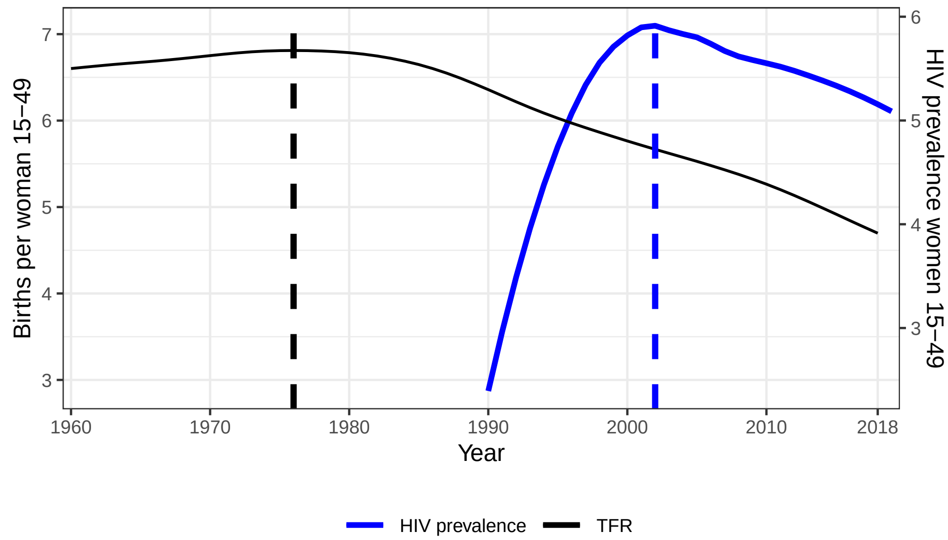

Figure 1: Trends of TFR 15-49 and HIV prevalence 15-49 in sub-Saharan Africa.

1990s, especially between 1990 and 1995, concurring with the significant increase in HIV prevalence. TFR decreased by 0.43 births per woman in the 1980s, then the pace of decline increased to 0.59 births per woman in the 1990s. During the early 2000s, TFR fell at a slower rate, 0.24 births per woman, which is lower than that of the late-1980s. Then, since the 2010s, the TFR has accelerated the rate of decline.

Table 2: Variations of TFR 15-49 and HIV prevalence 15-49 by n-year periods.

	TFR		HIV prevalence	
Period	From	Δ	From	Δ
20-year periods				
1980-2000	6.786	-1.022		
2000-2018	5.764	-1.067		
10-year periods				
1980-1990	6.786	-0.427		
1990-2000	6.358	-0.594	2.431	3.456
2000-2010	5.764	-0.500	5.887	-0.329
2010-2018	5.264	-0.567	5.558	-0.401
5-year periods				
1980-1985	6.786	-0.137		
1985-1990	6.648	-0.290		
1990-1995	6.358	-0.331	2.431	2.375
1995-2000	6.027	-0.263	4.805	1.082
2000-2005	5.764	-0.236	5.887	-0.115
2005-2010	5.528	-0.264	5.772	-0.213
2010-2015	5.264	-0.346	5.558	-0.151

Table 3: Changes in TFR 15-49 and HIV prevalence of women 15-49 by evidence of fertility stalls.

	Strong/moderate fertility stalls				Weak/No fertility stalls			
	TFR		HIV prevalence		TFR		HIV prevalence	
Period	From	Δ	From	Δ	From	Δ	From	Δ
30-year period								
1990-2018	6.003	-1.535	2.795	4.361	6.819	-1.843	2.403	0.815
10-year periods								
1990-2000	6.003	-0.720	2.795	5.024	6.819	-0.458	2.403	1.577
2000-2010	5.283	-0.343	7.819	-0.164	6.361	-0.683	3.979	-0.455
2010-2018	4.941	-0.472	7.654	-0.498	5.678	-0.702	3.524	-0.307
5-year periods								
1990-1995	6.003	-0.456	2.795	3.385	6.819	-0.201	2.403	1.258
1995-2000	5.547	-0.264	6.180	1.639	6.618	-0.257	3.660	0.319
2000-2005	5.283	-0.157	7.819	-0.073	6.361	-0.324	3.979	-0.269
2005-2010	5.126	-0.186	7.746	-0.092	6.037	-0.359	3.710	-0.186
2010-2015	4.941	-0.289	7.654	-0.179	5.678	-0.428	3.524	-0.172

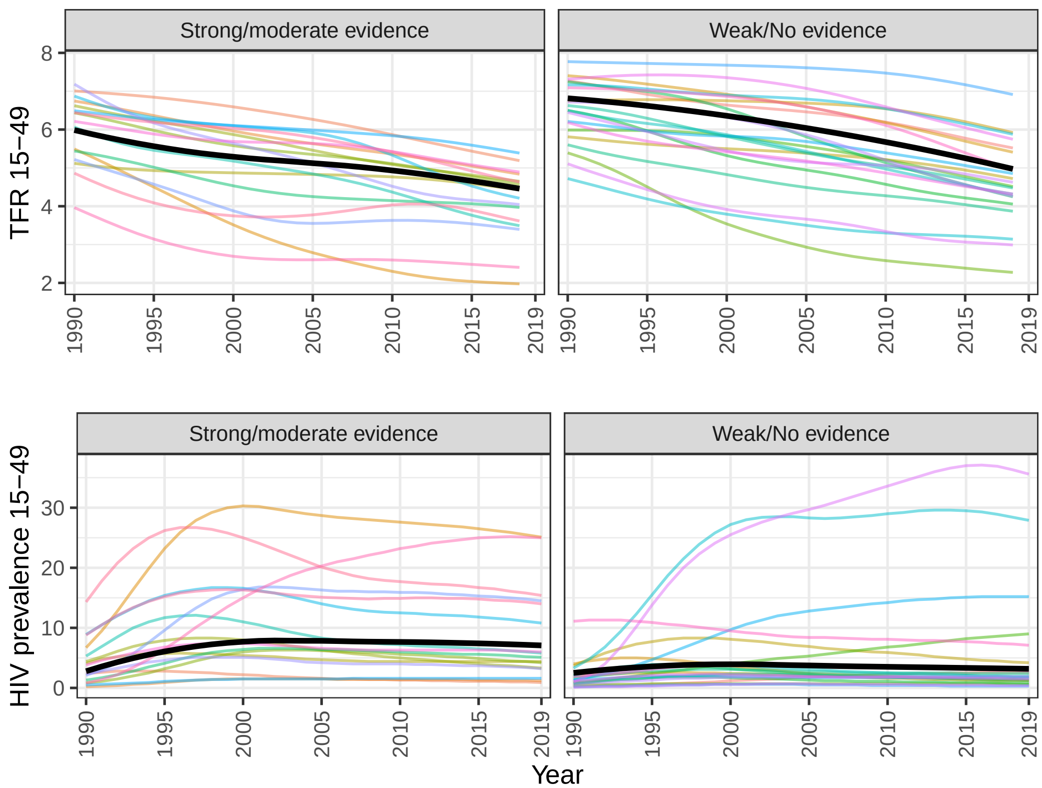

Figure 2: Trends of TFR 15-49 and HIV prevalence 15-49 in sub-Saharan Africa by evidence of fertility stalls.

Figure 2 presents fertility and HIV prevalence trends of SSA countries grouped by evidence of fertility stalls. There are clear differences between both groups. Countries with strong and moderate evidence of fertility stalls have, on average, lower fertility and higher HIV prevalence than those of weak or no evidence.

Exceptions are Eswatini and Lesotho, which are high-prevalence countries with low fertility and no evidence of fertility stalls. When analyzing the rates at which fertility has changed over the last decades (see Table 3), we find, first, that during the strong increase in HIV prevalence in the 1990s, the countries with evidence of strong and moderate fertility stalls decreased fertility 1.6 times faster than countries of weak or no evidence. Second, since the 2000s, fertility decline slowed down in countries of strong and moderate evidence, while the pace of decline increased in countries of weak or no evidence. Third, fertility decline in countries of weak and no evidence of fertility stagnation became increasingly rapid in each 5-year period from 1990-1995 to 2010-2015. In countries of strong and moderate evidence, the highest rate of decline was recorded in 1990-1995, 0.46, and slowed down until 2000-2005, 0.16, after which a slight increase began.

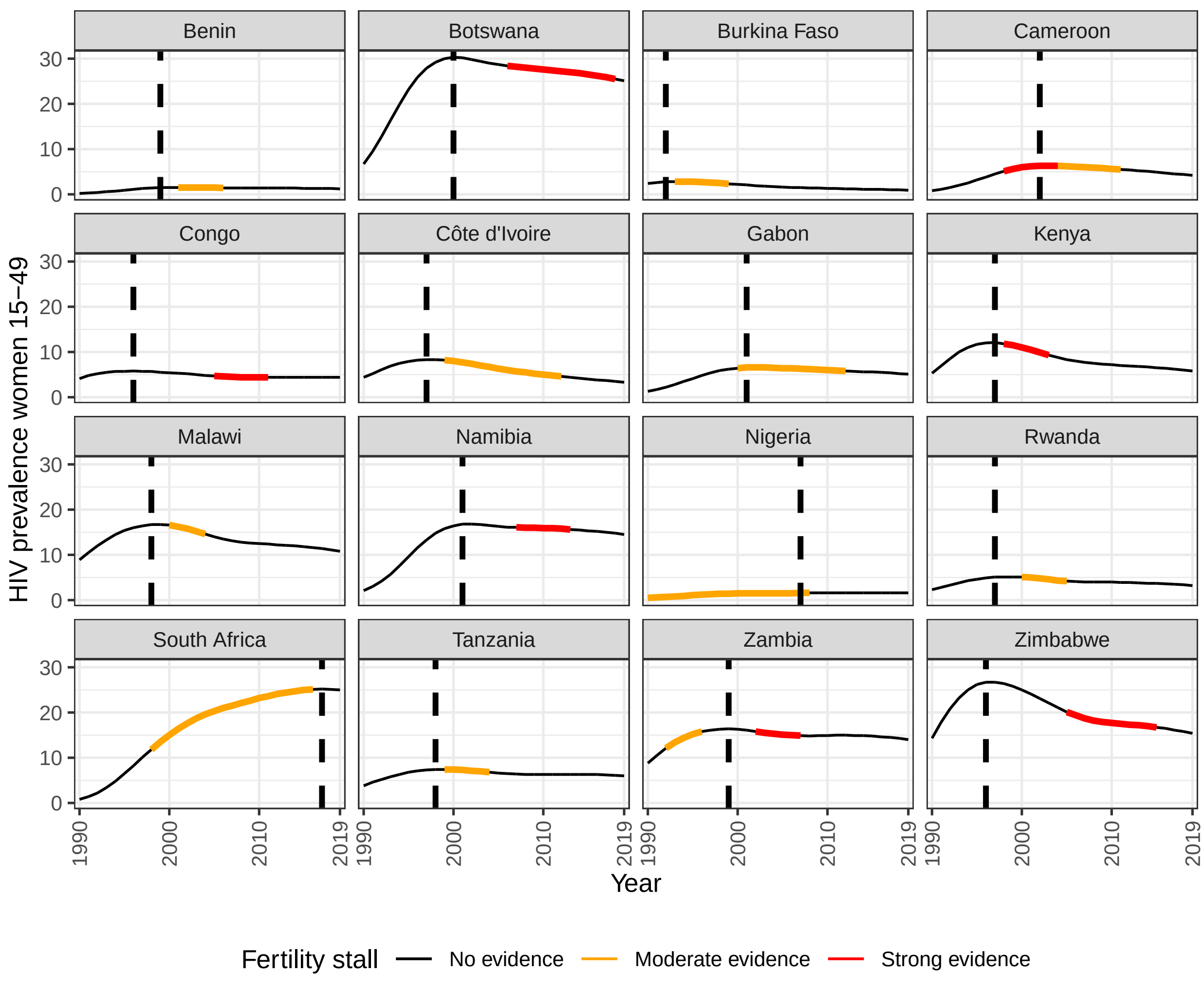

Figure 3: Trends of HIV prevalence 15-49 and fertility stall periods in countries of strong and moderate evidence.

Cross-country analysis

When we overlap the stall periods on the HIV prevalence trends in countries with evidence of strong and moderate fertility stagnation (see Figure 3), we observe that the stalls mostly occur after the peak in the HIV trend. Exceptions are Nigeria, where HIV levels are low, and South Africa, where fertility has been already low since the 1990s and HIV prevalence has been increasing constantly. Note that neither country corresponds to a case of strong evidence. In countries where there is strong evidence, most of the stall periods begin, first, after some decline in HIV prevalence and, second, from the mid-2000s onwards. Also, in general terms, these countries have higher HIV prevalence than those of moderate evidence. The exceptions are Congo, which has a prevalence more comparable to that of countries with moderate evidence, and Malawi and South Africa, which have prevalence levels similar to those of strong evidence.

From the DHS, we present in the upper panel of Figure 4 the reconstructed fertility trends according to HIV status for countries of strong and moderate evidence. As expected, all-women fertility is lower than the SSA average as it was identified that this group of countries had lower fertility than those of weak or no evidence of fertility stalls. Fertility of HIV-positive women and HIV-negative women has decreased overall since the 1990s. On average, in both cases fertility fell 2.3 births per woman over the last three decades (see Table 4). However, the pace of decline over time varies from HIV-positive women to those HIV-negative. During the 1990s, the decrease in fertility of infected women is 2.4 times faster than that of uninfected women. In the following decade, the fertility of uninfected women increases the pace of decline, almost similar to that of infected women. Since 2010, the rate of fertility decline for HIV-positive women is 0.2 births per woman slower than for HIV-negative women. Note that fertility of non-seropositive women mostly represents all-women fertility.

Table 4: TFR 15-49 changes by HIV status in countries of strong and moderate evidence of fertility stalls from reconstructed TFR trends.

	HIV-positive		HIV-negative		All women	
Period	From	Δ	From	Δ	From	Δ
30-year period						
1991-2018	5.155	-2.276	6.267	-2.274	6.136	-2.368
10-year periods						
1991-2000	5.155	-0.808	6.267	-0.344	6.136	-0.391
2000-2010	4.346	-0.882	5.924	-1.104	5.745	-1.153
2010-2018	3.465	-0.585	4.819	-0.826	4.591	-0.823
5-year periods						
1991-1995	5.155	-0.370	6.267	-0.121	6.136	-0.137
1995-2000	4.785	-0.438	6.146	-0.222	5.999	-0.254
2000-2005	4.346	-0.431	5.924	-0.610	5.745	-0.619
2005-2010	3.915	-0.450	5.313	-0.494	5.125	-0.534
2010-2015	3.465	-0.383	4.819	-0.544	4.591	-0.556

Lower panel of Figure 4 displays the population-attributable change in total fertility from the pooled sample in countries of strong and moderate evidence of fertility stalls. Between 1990 and 2018, PAC shows that fertility in the presence of HIV went from 2% lower than the case in the absence of HIV to about 6% lower. Over the same period, a weighted linear regression of PAC on HIV prevalence reveals that for each percentage increase in HIV prevalence in women of ages 15–49, there is a population-attributable decline in TFR of 0.41% (95% CI: 0.11% – 0.71%). PAC trend shows a slowdown in the mid-2000s, which coincides with most periods of stalled fertility. From TFR ratio, we find an increasing gap between the TFR of seropositive and non-seropositive women during the 1990s, which is mainly explained by the faster decline in fertility of the former. During the mid-2000s the ratio increases as the decrease in fertility of HIV-uninfected is faster. Since the end of the 2000s, the ratio has stabilized slightly above 0.7.

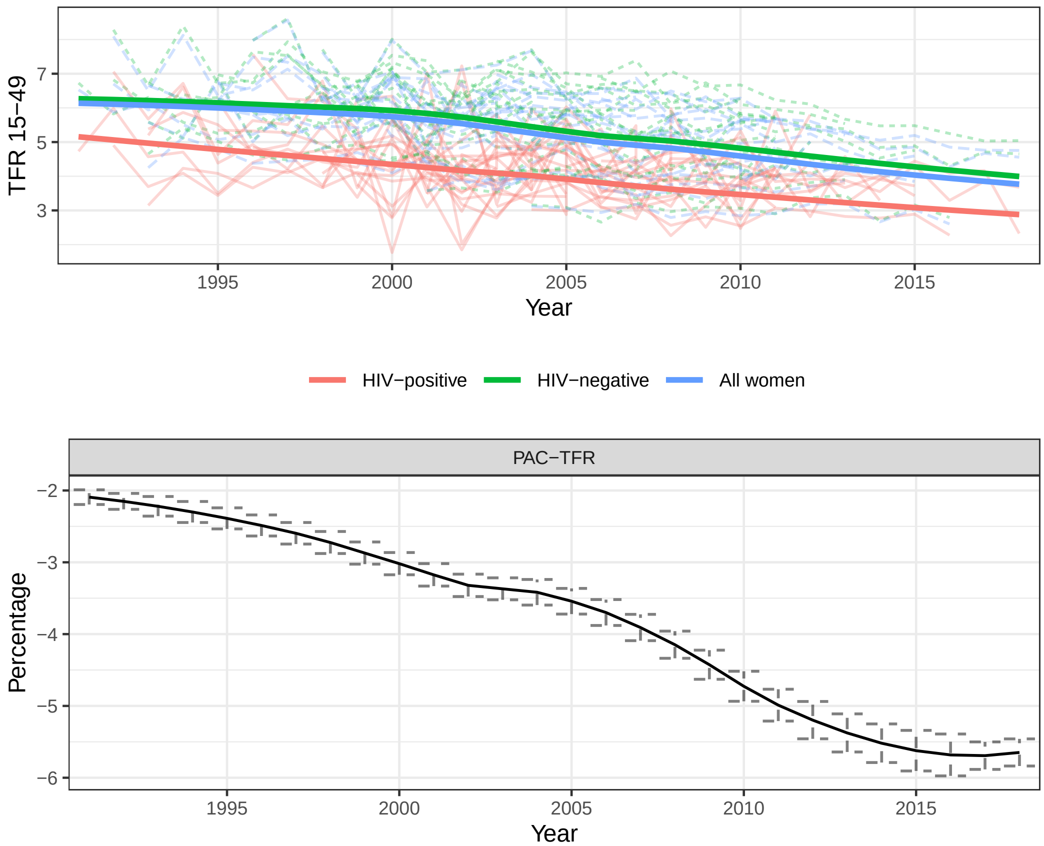

Figure 4: Reconstructed TFR trends by HIV status and Population-attributable change (PAC) in total fertility in countries of strong and moderate evidence of fertility stalls.

Figure 5 displays the reconstructed fertility trends according to HIV status by country and evidence of fertility stalls. Overall, it should be highlighted that stalls are clearly visible among HIV-negative women and that although trends among HIV-positive women may have contributed to the stalls, the contribution is quite limited, especially in countries of moderate evidence. For this latter group of countries, fertility of HIV-positive women has decreased overall; although we observe some reversals in fertility decline during the periods classified under the stalled category in each country. From there, fertility decrease has resumed. We observe a similar pattern in fertility of uninfected women in Malawi and Rwanda. Overall, fertility has declined since the mid-1990s although with halts during periods identified as of stagnant fertility. In contrast, estimates show stalled fertility among non-seropositive women in Côte d'Ivoire, South Africa, and Tanzania since 2000. The reconstructed trends show the opposite case in countries with strong evidence of stagnation. Fertility of HIV-uninfected women has decreased overall in Cameroon, Kenya, and Zambia, but with halts or increases in periods already identified as of stalled fertility. Then, the decline has resumed.

Except for Zambia, fertility of seropositive women is at a plateau since about 2000. Not only do the upward trends coincide with periods of stalled fertility, but current fertility levels resemble those of the mid-1990s.

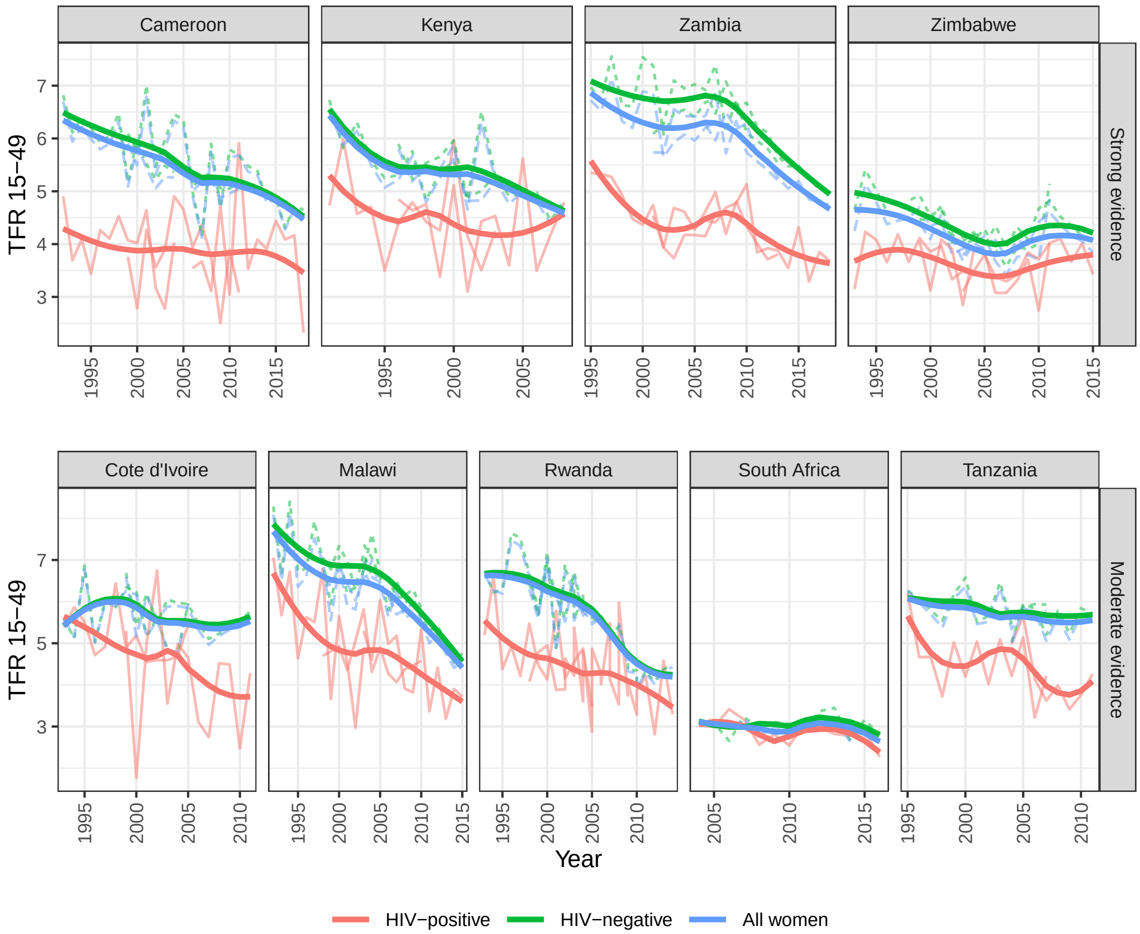

Figure 5: Reconstructed fertility trends by HIV status in countries of strong and moderate evidence of fertility stalls.

From Figure 6, PAC shows that HIV has contributed to fertility decline in countries of strong evidence during the 1990s, especially in high-prevalence countries such as Zambia and Zimbabwe, where HIV prevalence was well above 15% of the female population aged 15-49 years. However, since 2000 the population-attributable change in total fertility has diminished, which means that fertility has become more similar to that of a scenario without HIV. Except for Zambia, PAC in recent years is higher than in the 1990s. We observe slight increases in PAC coinciding with stall periods. In Cameroon, it has increased since 1998, which is when the stall began. In Zambia, we find also an increase from 2002, especially during the stall period 2002-2007. Although fertility decline resumed in Cameroon and Zambia, PAC has still been increasing. In Zimbabwe, PAC increased during the 1990s and then it stabilized until 2010. Since then, it has increased two percentage points. In Kenya, we observe an increase in PAC in the late-1990s, which was the start of the stall period, and a decline afterwards until 2003, which is when the stall ended. Regarding TFR ratio (computed from results presented in Figure 5), it is converging sharply towards one in Kenya, which means that fertility of HIV-infected and HIV-uninfected is similar. In Cameroon, Zambia, and Zimbabwe, the ratio has increased smoothly since 2000, which is explained by the decline in fertility of non-seropositive women and the stalled fertility of those seropositive. In all of these countries, estimates of TFR ratio are well above the average TFR ratio computed from results presented in Figure 4, meaning that the gap is narrowing in countries with strong evidence, in contrast to countries with moderate evidence.

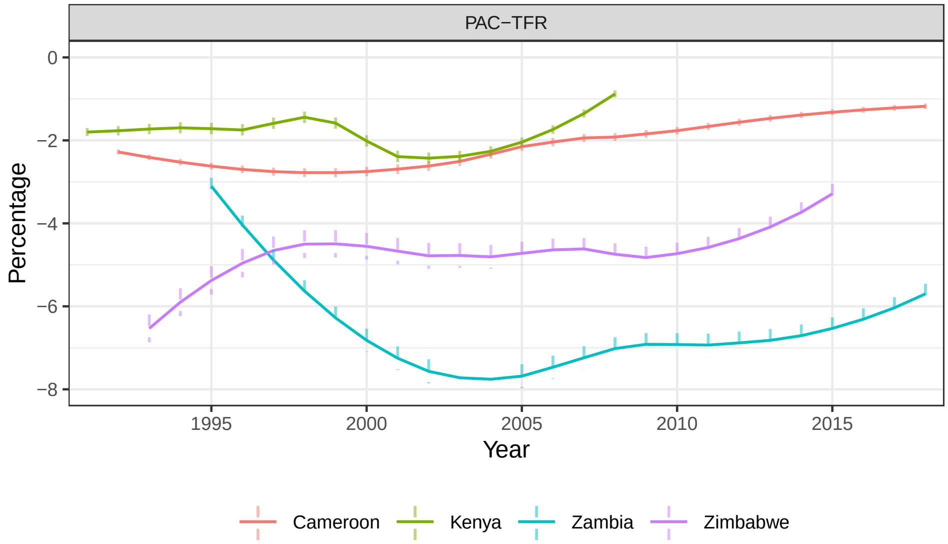

Figure 6: Population-attributable change (PAC) in total fertility in countries of strong evidence of fertility stalls.

Country level analysis

When analyzing the specific case of Zimbabwe, we observe that women are now more aware of mother-to-child transmission of HIV than they were before. Although more than 90% of women know that HIV can be transmitted from mother to child, during the 1990s knowledge was limited to transmission during pregnancy (see Figure 7). Currently, more than 80% of women know that transmission is also possible during delivery and breastfeeding. The level of knowledge is similar among women according to HIV status (see Figure 8), i.e., both seropositive and non-seropositive women are equally aware of the risks of transmission and has been so since 2005. Knowledge about medications that prevent HIV transmission has increased dramatically in recent years, with a slightly higher level among infected women, which also coincides with the period of stalled fertility in Zimbabwe. We believe that this may have contributed to increased fertility, as the fear of mother-to-child transmission has decreased and makes infected women more likely to seek treatment and prevent, among others, fetal loss.

Antenatal care has contributed to knowledge and self-awareness of HIV. Figure 9 displays the proportion of women who received HIV counseling for those who gave birth in the 24 months prior to the survey. We do not observe clear differences by HIV status. We find increased levels of knowledge linked to antenatal care during the stall period in both groups of women, and this could affect fertility as women are now more likely to be aware of their status —and maybe their partner's—. On the one hand, HIV-negative women may be more likely to be ready to have children than before, while on the other hand, those HIV-positive may seek help earlier to prevent mother-to-child transmission. From 2005 to 2015, the proportion of women who were

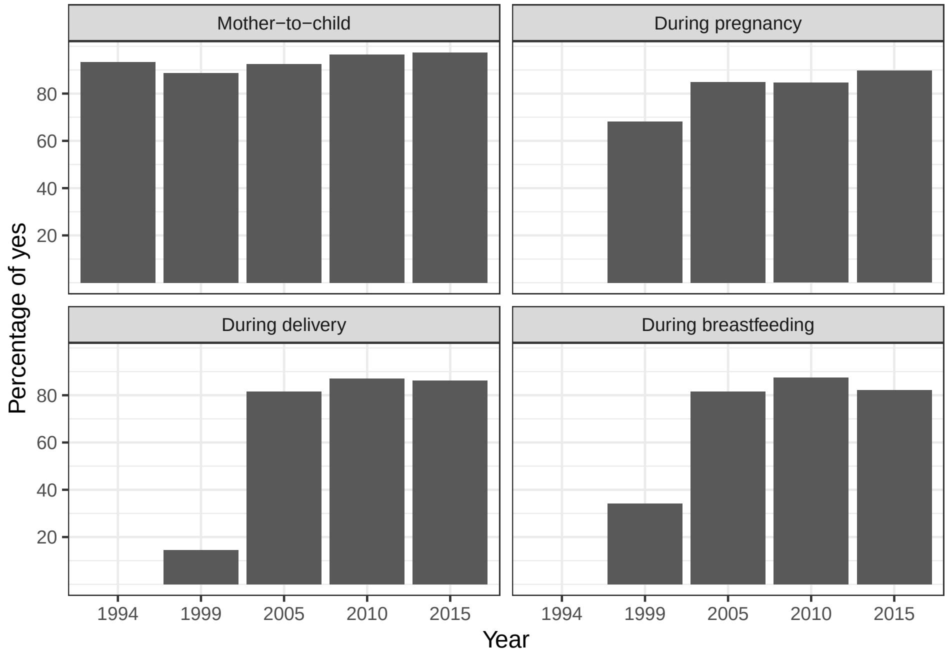

Figure 7: Knowledge of HIV mother-to-child transmission in Zimbabwe.

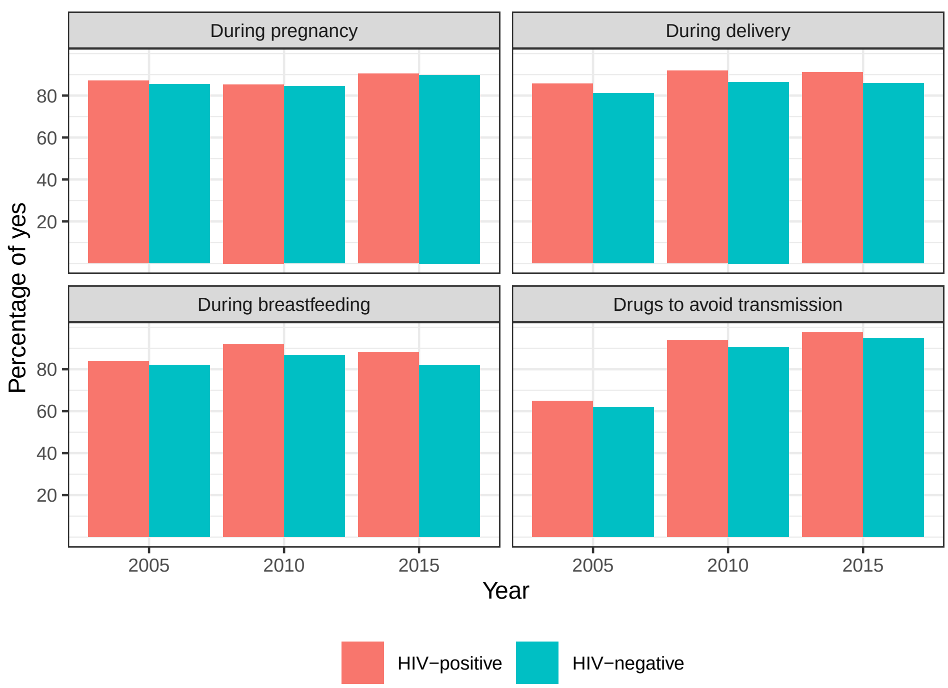

Figure 8: Knowledge of HIV mother-to-child transmission by HIV status in Zimbabwe.

offered HIV testing more than doubled. Of those women, 30% were tested for HIV in 2005, while virtually all women in 2015. On average, more than 90% of women got the results; therefore, they know their HIV status. Currently, 91% of HIV-infected pregnant women in Zimbabwe receive antiretroviral treatment to prevent mother-to-child transmission, while it was 30% in 2010 (UNAIDS, 2020a).

Figure 9: HIV testing linked to antenatal care by HIV status in Zimbabwe.

As a result, increased knowledge and awareness of HIV, together with the development of drugs to prevent mother-to-child transmission, has made it possible to increase the number of births among HIV-positive women. The left panel of Figure 10 shows changes in fertility rates and ideal family size (IFS), which is the desired number of children, from 1988 to 2015. IFS has decreased along with TFR until 2005. In Zimbabwe, current TFR and IFS are at similar levels, at 4 children per woman. Regarding HIV status (see right panel of Figure 10), infected and uninfected women have comparable IFS, which would suggest that their HIV status would not have affected their desired number of children. Non-seropositive women have higher TFR than those seropositive, although the differences have narrowed in recent years. Fertility rates of HIV-positive women have slightly increased during the stall period, while they have been stagnated in the case of HIV-negative women.

To better understand the effect of HIV on fertility stalls, we analyze the fertility dynamics, such as the changes in sexual activity, contraceptive use, and postpartum infecundability, during the stall period according to HIV status. Upper panel of Figure 11 shows the trends of sexual activity from 1994 in Zimbabwe. We consider a woman as sexually active following the definition proposed by Bongaarts (2015), that is, if she is married or, in the case of unmarried women, if she is pregnant, amenorrheic, in postpartum abstinence, using contraceptives or has reported sexual activity in the last month. Sexual activity fell three percentage points until 2005 and since then it has increased to nearly the same levels as in the mid-1990s. According to HIV status, seropositive women has increased sexual activity by almost four percentage points since 2005, while

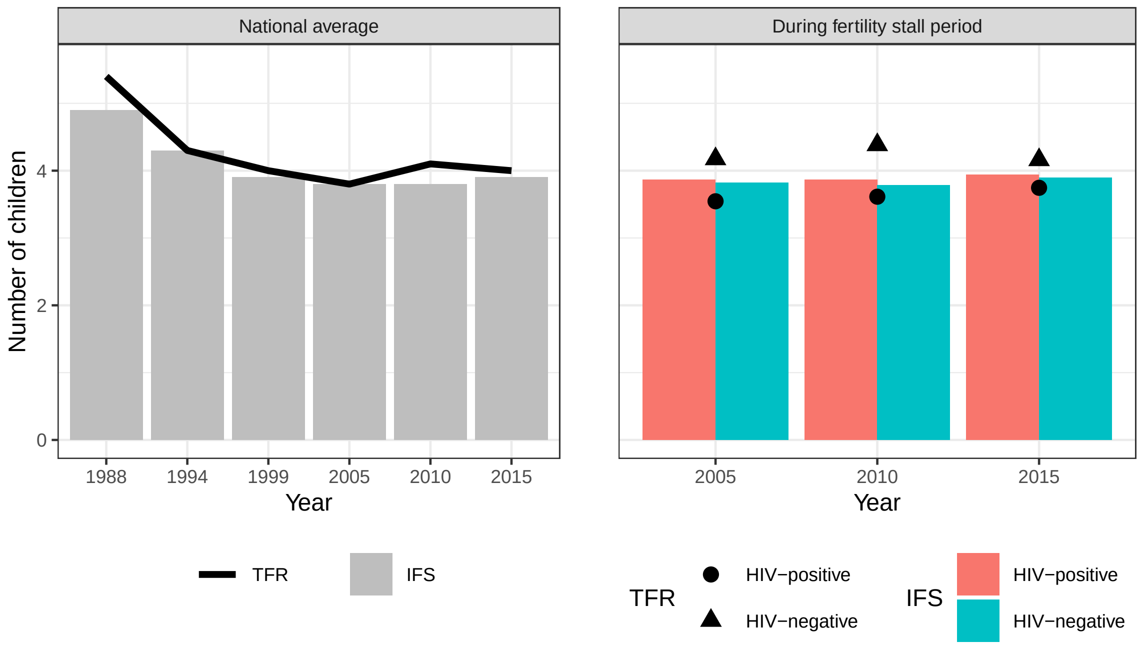

Figure 10: Ideal family size (IFS) and TFR by HIV status in Zimbabwe.

non-seropositive women, in one percentage point. Nevertheless, the positive effect on fertility of the increase in sexual activity may be counter-balanced by the increase in contraceptive use. We observe a rapid increase of nearly 20 percentage points in contraceptive use over the last two decades, with a slowdown during 2005-2010 (middle panel of Figure 11). In the case of non-seropositive women, contraceptive use decreased slightly in 2005-2010 and then increased some eight percentage points; while for seropositive women, contraceptive use increased ten percentage points during 2005-2015. Overall, contraceptive prevalence in Zimbabwe remains low, with one in two women using contraceptives. The lower panel of Figure 11 presents the trends of the mean number of months of postpartum infecundability by HIV status. As seen, the mean duration remained invariant from 1994 to 2005. Since then, the length has decreased, on average, one month for HIV-negative women and two months for those HIV-positive. For the first group of women, the decrease is mainly associated with a shorter duration of breastfeeding of about two months, while for the second group, the decline is explained by a shorter duration of both postpartum abstinence and amenorrhea of about two months in each case.

Results of the decomposition of fertility trends in Zimbabwe are presented in Figure 12. TFR is computed over the five years before the surveys. It suggests that between the 2005 and 2010 surveys (2003–2008 period), fertility decreased among both HIV-positive and HIV-negative women but HIV prevalence decreased as well. As a result, the share of HIV-negative women increased and, as they have higher fertility, the weighted average of the TFR tilted towards increased fertility; therefore, HIV prevalence contributed to higher fertility levels through a composition effect. In contrast, in the most recent period, 2008–2013, the increase in total fertility was driven by the increase in fertility of both HIV-positive and HIV-negative women, while the contribution of HIV prevalence remained stable. Apparently, part of the stall was thus initated by changes in composition of the population —the share of women with higher fertility increased because of the decrease in HIV prevalence— but afterwards, the change is explained by a change in behavior among all women.

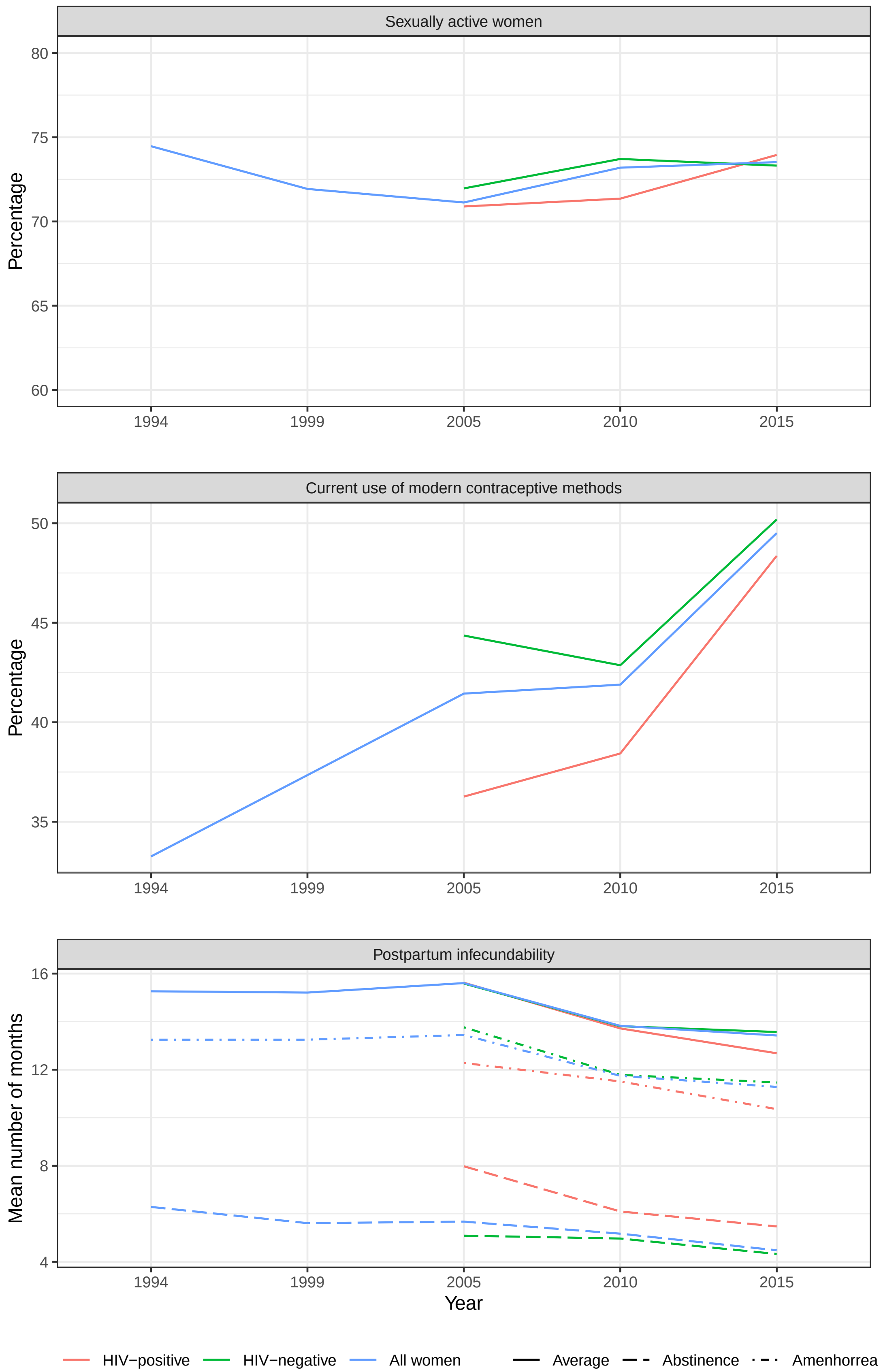

Figure 11: Changes in sexual activity, use of modern contraception and postpartum infecundability by HIV status in Zimbabwe.

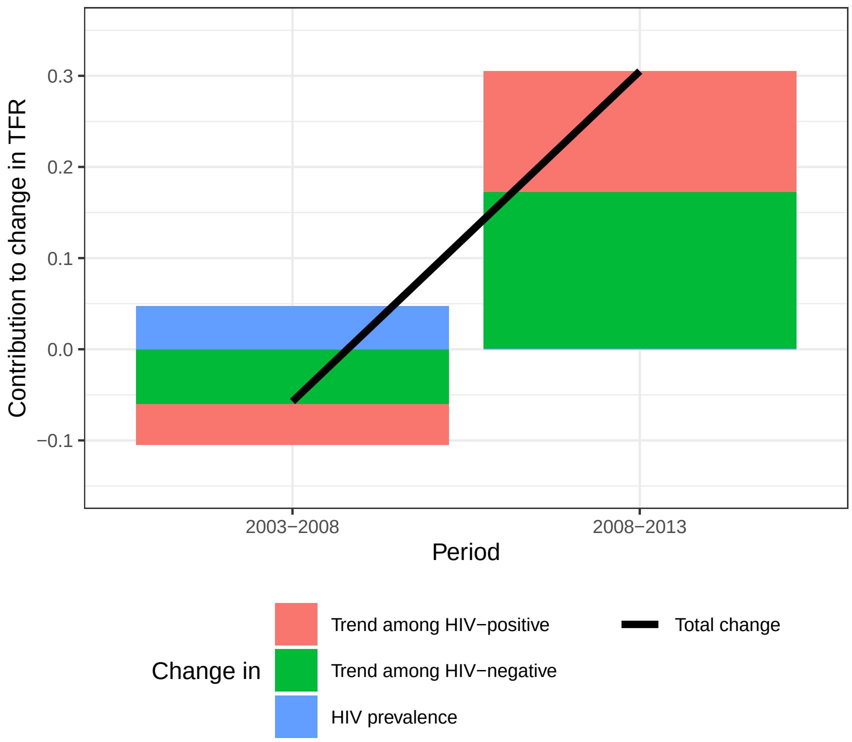

Figure 12: Decomposition of fertility trends in Zimbabwe.

Discussion

In this research we have analyzed the link between HIV and fertility stalls in SSA from three levels, regional, cross-country, and country level. Overall, HIV has contributed to fertility decline; however, prevalence levels and rapid rates of increase in HIV prevalence may have affected fertility differently across SSA. At the regional level, we have observed diverse fertility patterns between countries depending on the levels of HIV prevalence. First, fertility in high-prevalence countries tended to decrease fertility faster during the 1990s than those of lower HIV prevalence. Second, when HIV reached its peak in 2001-2002, the pace of fertility decline in high-prevalent countries slowed to levels comparable to those before the arrival of HIV/AIDS, while the pace of decline increased in low-prevalent countries. Third, this period of slowdown in the mid-2000s coincides with the beginning of most events of stalled fertility in SSA (Schoumaker, 2019). Fourth, all countries that have been included in the category of strong and moderate evidence of fertility stalls are among the countries with the highest HIV prevalence in SSA. We observe a different impact of HIV on fertility depending on levels of prevalence. It appears that the more a country is affected by HIV, the stronger the evidence of halts and reversals in fertility decline. Exceptions are Eswatini and Lesotho, where fertility was already low when the disease arrived.

Cross-country analysis focus on countries with strong and moderate evidence of stalls. First, we have found lower fertility among HIV-positive women than among those HIV-negative, consistent with previous research (Chen & Walker, 2010; Gemmill et al., 2018; Gregson et al., 2002; Kongnyuy & Wiysonge, 2008; Lewis et al., 2004; Marston et al., 2017; Zaba & Gregson, 1998). Second, fertility of both groups of women has decreased overall, although at a different pace: it was much faster among seropositive women during the 1990s but the opposite has been the case since 2000. Third, we observe a more rapid decline in fertility among HIV-negative women in the mid-2000s, which is when the stall periods begin, while the pace of decline remain invariant among those HIV-positive. Fourth, we observe fertility stalls among HIV-negative women in the periods classified as of stalled fertility in each country. In contrast, fertility stalls among infected women are observed mostly in countries of strong evidence, which may suggest that although fertility of HIV-positive women has contributed to fertility stalls —but to a lesser extent in countries of moderate evidence—, fertility of HIV-negative women mainly accounts for stalls. The decrease in the prevalence of HIV/AIDS, by changing the composition of the population, also contributed to the stall. The decrease in funding for family planning programs, precisely to combat HIV, may have contributed to the increase in fertility among uninfected women (Askew et al., 2017; Bongaarts, 2008; Casterline, 2017; Fuchs & Goujon, 2014). In countries with strong evidence of fertility stalls, given that HIV-positive fertility is stagnant and that the TFR ratio is converging to 1, one would expect that overall fertility will not decline rapidly in the short term.

From the country-level analysis, the case of Zimbabwe provides interesting findings. First, antenatal care plays an important role in knowledge of ways of transmission and prevention, and self-awareness of HIV. There are no clear differences in knowledge between HIV-positive and HIV-negative women. Second, knowledge and use of drugs to prevent mother-to-child transmission has increased rapidly during the stall period, allowing more children to be born. On the one hand, infected women may carry more pregnancies to term, while, on the other hand, uninfected women may feel more confident to begin childbearing. Third, ideal family size has stalled at about 4 children since 2005, which is the beginning of the stall period in Zimbabwe, with similar levels by HIV status. Fourth, HIV-positive and HIV-negative women match their fertility with the number of children they wish to have, so both groups of women contribute to fertility stalls. Fifth, compared to non-seropositive women, fertility of those seropositive is lower for all age groups, except for adolescents, which has been already observed elsewhere (Chen & Walker, 2010; Gregson et al., 2002; Lewis et al., 2004; Marston et al., 2017; Zaba & Gregson, 1998). Sixth, in contrast to previous evidence on the fertility-reducing behaviors of HIV-infected women, demand for children has not decreased (Bankole et al., 2011; Johnson et al., 2009), sexual activity has increased (Porter et al., 2004), and duration of amenohrrea has decreased (Chirgwin et al., 1996). On the other hand, we do find fertility-reducing behaviors such as the increase in contraceptive use. Regarding fertility-enhancing behaviors, our results are in line with those of previous investigations, such as high desire for having more children and shorter periods of breastfeeding (Houle et al., 2016; Magadi & Agwanda, 2010; Romaniuk, 2011; Westoff & Cross, 2006). During the stall period, we find a decrease in the duration of breastfeeding and postpartum abstinence, still low levels of contraceptive use —despite the significant increase—, and a demand for children that is twice the replacement rate. As a result, fertility-increasing behaviors have counter-balanced the fertility-reducing behaviors and it has led to non-declining fertility in Zimbabwe. Seventh, the stalled fertility of women of both HIV statuses, together with high demand for children, would predict a longer stall period.

The case of Zimbabwe adds to those of Kenya (Magadi & Agwanda, 2010; Westoff & Cross, 2006) and South Africa (Garenne et al., 2007; Houle et al., 2016) where evidence also shows that HIV has contributed to fertility stagnation. It may also have contributed in Cameroon, Namibia, and Zambia but further research is needed. Unfortunately, we have not been able to analyze these countries due to lack of information on HIV status at the time of fertility stalls. Although in many countries HIV testing is included, unluckily these surveys do not necessarily cover the stall periods. Overall, more research is needed since even with the increased emphasis on antiretroviral treatment and other health care services in SSA, their impact on fertility and childbearing have received relatively little attention (Marston et al., 2017; Myer et al., 2007). Another limitation is that we do not know the time since seroconversion, so when we reconstruct fertility trends we assume that a woman infected at the time of the survey has been infected for the past 12 years, which is our cut-off. This may overestimate the fertility rates of HIV-infected women in the case of recently infected women who have given birth before infection and, for the same reason, underestimate that of uninfected women. However, most infections occur between the ages of 15 and 24 years (UNAIDS, 2020a) and usually before the first birth. Most births in SSA occur before age 34, so it is plausible to assume that most births correspond to women who were already infected when they gave birth. For older women, the probability of infection at an older age increases, but fertility at those ages is lower, so the contribution in total fertility is also lower. Therefore, overestimation and underestimation, if any, would be minimal.

From a public policy perspective, despite the progress in reducing HIV prevalence, SSA remains the most affected region in the world. HIV-positive people, especially adolescents, are at greater risk of exclusion and in some contexts continue to be stigmatized. An early infection and unintended pregancy would mean a double burden for them. HIV prevention should focus, first, on education to prevent infection and, second, on guaranteeing everyone the right to health by breaking down barriers that prevent people from receiving essential services (UNAIDS, 2020b). In terms of fertility, SSA has overall the highest levels of unmet need for family planning. Despite needs are greater among HIV-positive women than among HIV-negative women (MacQuarrie, 2015), they are not met, which could be translated into unintended births. Joint programs addressing both HIV and family planning are thus needed in SSA. The efforts made so far have contributed to improving the quality of life of the infected population, but many challenges remain.

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