A Decomposition of Fertility Differences in HIV-Infected and Uninfected Women in Rural Tanzania

Kristin Bietsch[1], Mark Urassa[2], Milly Marston[3], Raphae Isingo[2], Yusufu Kumogola[2], Baltazar Mntenga[2], Julius Mngara[2], John Changalucha[2], Benjamin D. Clark[3], and Basia Zaba[3]

[1]Princeton University

[2]National Institute for Medical Research, Tanzania

[3]London School of Hygiene and Tropical Medicine

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Abstract

Using a decomposition of Poisson models, this paper explores the reasons for lower fertility observed in HIV positive compared to HIV negative women in the same community in rural northwest Tanzania. Several proximate determinants of fertility are broken down into their contributions to the gap via endowments (structural differences) and coefficients (effect differences). Of the gap between the 123 per 1000 women-years birth rate for the HIV positive population and the

217 per 1000 women-years birth rate for the HIV negative population, 11% can be explained by differences in the characteristics of the subpopulations. Differences in coefficients account for 84%, where the effect of a similar change in a characteristic leads to different effects on the fertility rate, which we interpret as differences in fecundability, the probability of conception over a specific time period. This leaves 5% of the gap unexplained by our proximate determinant variables. Differences in coefficients are strongly evident in reactions to sexually transmitted infection symptoms and coital frequency, while demographic differences in the distributions of age and marital status by sero status also contribute to the fertility rate gap. The large effect of coefficients suggests a reduced fecundability in HIV positive women and their partners which is now easily measurable using decomposition of Poisson models

1 Introduction

The interaction between fertility and HIV has received extensive attention in HIV/AIDS research [7, 9, 8, 10, 20]. This paper employs a statistical model uncommon in fertility research to better explore the fertility-reducing effect of HIV. Using a decomposition technique, we model births over an interval of HIV positive and negative women. By including numerous proximate determinants of fertility in these models, we estimate the effect of multiple determinants on the gap in fertility rates. The determinant can affect rates either through differences in endowments (the structural differences in the population; for example more divorced women in one group than the other) and coefficients (the different effect the same change has for the two

sub-groups; for example, an additional symptom of a sexually transmitted infection may reduce fertility more in one group than the other). This technique offers a detailed view of the fertility reducing effects of HIV as a disease and social phenomenon.

A popular motivation for research on the fertility of infected and uninfected women is the common use of ante-natal clinic data in developing prevalence estimates of HIV for the entire population [9] and how differential fertility may bias these estimates. A high proportion (90%) of women visit ante-natal clinics, and in most countries with low contraceptive prevalence, pregnant women are assumed to be representative of the sexually active population [21]. However, as research has shown, fertility varies by HIV status, and data collected from clinics underestimates the prevalence of the whole female adult population [10]. Thus, decomposition will further the understanding of why a bias may result from using pregnant women as a representative sample of the adult female population.

2 Literature

Understanding the effect of HIV on fertility is essential as women of reproductive age represent a large proportion of the population in sub-Saharan Africa. Patterns have emerged showing lower fertility among HIV infected women compared to uninfected women in the same population [14, 12, 20, 13] While the mechanisms for the fertility divergence have been hypothesized [20, 13], an empirical approach to decompose the proximate determinants of fertility will provide a deeper understanding of the fertility-reducing effects of HIV.

In both developed and developing countries and in both contracepting and non-contracepting populations, HIV infected women have lower birthrates than uninfected women, including after controlling for confounding variables [14, 12]. In the mid-1990s, a 29% reduction in fertility was observed among HIV-positive women in Kisesa, Tanzania [12]. Fertility reduction, found in other sites across sub-Saharan Africa, is not constant across age groups. Fertility of HIV infected women is higher than uninfected women in the age range 15-19, where pregnancy and HIV are highly correlated with early sexual debut and other high risk activities [20, 13]. In all other ages, fertility is higher in the uninfected group, with the divergence increasing with age and epidemic duration. The difference in fertility is greatest between uninfected women and women with AIDS-associated symptoms and in the terminal stages of infection [14, 12].

On an aggregate level, it has been estimated that lower fertility among HIV positive women is associated with a reduction in the total fertility of a population by 0.4% per each percentage point of HIV prevalence in the female population [8, 13, 20]. The HIV epidemic is associated with population level declines in fertility, but this reduction is difficult to disentangle, as many countries are still at the early stages of fertility transitions [10]. Declines in fertility may occur in areas with high HIV prevalence because of lower fertility in HIV positive women, but also because of the protective behavioral changes of HIV negative women in the population [20]. As the epidemic matures, fertility of HIV positive women will be further reduced, so that the difference between infected and uninfected women increases [20]. Also, as infertile HIV positive women succumb to the disease, fertile women will

become a larger proportion of the population and the total fertility rate will rise [10].

Fertility can also affect the progression of HIV as a disease. First, the likelihood of acquiring HIV is significantly higher during pregnancy than during lactation or non-pregnant, non-lactating periods [7]. This is probably caused by the rise in pregnancy-produced hormones. Second, if pregnant women have already contracted HIV, it may accelerate disease progression [8]. Higher-order pregnancies, which may lead to the development of AIDS [20], would create a population where fecund HIV-positive women would have shorter survival times than infecund, causing a decrease in the fertility of the HIV-positive female population.

Conscious efforts to raise fertility appear to be weak [10], as women are found to be concerned about their existing children [10, 20]. Many women already have several children when they learn their status, and in a Zimbabwean study, around half of the HIV positive women interviewed said they wanted fewer additional children and wanted to delay their next birth longer than before they knew their status [10]. These social changes combine with the biological effects of HIV to cause a large reduction in fertility among HIV positive women.

3 Proximate Determinants of Fertility

While numerous proximate determinants have been hypothesized to reduce fertility in HIV positive women, further analysis is needed to estimate their individual contributions. In addition, proximate determinants deemed to

have limited roles in many populations may have larger effects when studying the difference between HIV positive and negative women's fertility.

In 1978, Bongaarts put forward eight direct determinants (biological and behavioral factors) which were influenced by indirect determinants (socioeconomic, cultural, and environmental variables) [4]. The eight proximate determinants identified by Bongaarts include proportion married, contraception, induced abortion, lactational infecundability, frequency of intercourse, sterility, spontaneous intrauterine mortality, and duration of the fertile period [5]. Not all proximate determinants have the same level of effect on fertility. Therefore, while many factors which may affect fertility on a personal level are discussed in Bongaarts work, they are not considered to vary enough across populations to explain fertility differentials between groups [4, 6]. Three of these factors, coital frequency, sexually transmitted infections, and intrauterine mortality may contribute more when examining the fertility differential in sub-population groups.

The difficulties in separating the proximate determinants for HIV infected and uninfected women are the virus's direct effect on women's fecundability, or probability of conception[13] and its indirect effects caused by behavioral responses to the HIV epidemic. The fertility factors proposed by previous research include age, marital status, coital frequency, sexually transmitted infections, fetal loss, and contraceptive use [10, 21, 13, 12].

Age is an important factor determining fertility, with age-specific fertility rates beginning at menarche, rising until the mid-20s, and then declining with age until menopause [11]. Age is associated with changes in fecundability, higher risk of intrauterine mortality at older ages, and lower frequency of

sexual intercourse with the rising ages of both partners [5]. Women who are HIV positive are, on average, older than the HIV negative group, and may have lower fertility in the absence of HIV infection. Therefore, one of the main differences between fertility of infected and uninfected groups could be explained by the differing age distributions.

In areas with high HIV prevalence, marital disruptions become more common because of increases in divorce and widowhood [10, 21, 13]. In the case of widowhood, a woman may not survive her partner for a significant length of time because of her own positive sero status. If a woman does remarry, she may become a second wife to a man who has a large number of previous sexual partners compared to her late husband, and may therefore be at higher risk of contracting HIV. Because coital frequency per woman is lower in polygamous relationships, birth rates will also decline [10]. With the rise of knowledge of the epidemic, divorce rates have risen because of suspicion of partner infection [12, 18] and remarriage rates may decrease because the general population may avoid divorced and widowed partners as a protection against HIV. A final type of separation is short term occasions, for example seasonal migration. This is dangerous in terms of HIV acquisition because of the higher contact with casual partners, and will also lead to lower fertility in periods of partner absence [10].

Bongaarts does not label coital frequency as an important determinant of fertility because of its lack of variation across populations [4]. When studying the effects of HIV on fertility, coital frequency may be a significant factor as many different biological and cultural aspects of the disease lead to decreased coital frequency in the group of infected women [10]. Coital

frequency of couples can be reduced by either partner's health status [10]. Lower coital frequency occurs when either partner begins to show symptoms of HIV associated illnesses [12, 13]. This reduces fertility early in the progression of the disease if their partner's disease is further advanced. The reduction in coital frequency does not account for all reduction in fertility [8], but it provides a valuable comparison between infected and uninfected women.

The final partner-related fertility reduction factor is more difficult to measure than partner presence and coital frequency. Reduction in the quality and quantity of the sperm of HIV positive males may cause a decrease in fecundability [10, 20]. Lower production of spermatozoa is found at the advanced stages of HIV infection of males [8]; this coupled with decreased coital frequency reduces fertility while a partner is present. Coital acts are necessary for reproduction, and HIV, operating through either partner, can reduce coital frequency or remove it all together from the lives of reproductive age women in HIV infected areas.

History of sexually transmitted infections is much more common in HIV positive women [13] and may explain a large portion of the difference in fertility. The effects of sexually transmitted infections (STIs) on fertility, which include sterility and spontaneous intrauterine mortality [4], can be exacerbated by the presence of HIV. STIs interact with HIV to reduce fertility in two ways. First, STIs, which may cause genital lesions, can increase HIV transmission [10] which leads to a decline in fertility through many factors.

Second, many STIs can cause infertility and subfertility. In 1983, Bongaarts and Potter found that variations across populations in intrauterine

mortality were small, though there was a noticeable increase with age of the mother [6]. However, differences in intrauterine mortality may be more evident in groups of the same population, such as when comparing HIV positive and negative individuals. Intrauterine mortality is often hard to detect at early stages of pregnancy, especially since most of it occurs before the woman knows of her pregnancy. Subfertility can be caused by amenorrhea or fetal loss [13], and has been estimated to account for almost half of the variation in fertility between the positive and negative sub-groups of women [10, 13, 20].

Thus the high correlation of HIV and STIs appears to have a dramatic effect on fertility of HIV positive women. This effect could be reduced over time as diagnosis and treatment of STIs become more common or as safe sex practices increase.

HIV positive women are found to have a higher frequency of contraceptive use (including condom use) [12] than their uninfected counterparts. This may be to protect a partner [10] or to space/avoid additional pregnancies. The need to protect one another and greater discussion of sexually transmitted infections in the community opens dialogue between partners, which can increase contraceptive use. Alternative methods, such as the female condom, offer women a means of protection that does not require her partner wearing a condom [10]. Though the woman may already be infected, HIV status is often unknown, and attempts to protect oneself from HIV can result in lower fertility.

While age, marital status, coital frequency, sexually transmitted infections, fetal loss, and contraceptive use have all been examined as possible

explanations for differences in fertility rates of HIV positive and negative women, the following decomposition will allow us to observe the effects that differing levels of each variable can have on fertility rates.

4 Data

Data analyzed in this paper come from the Kisesa Ward in the Mwanza region of northwest Tanzania, where the Tazama Project has collected demographic data and HIV information on the populations of 6 villages since 1994 in order to monitor the response to the HIV epidemic.

As part of the ongoing project, all adults ages 15 and older were invited to participate in HIV testing and a detailed survey from late 2006 to early 2007. Respondents were not informed of their status at the time, but were told to visit the local clinic to receive their test results, making it unknown in this data set who is aware of their serological status. Three household visits were conducted between late 2007 and late 2009, recording new residents (births and in-migrants) and exits of residents (deaths and out-migrants) from the survey area.

Women ages 15 to 44 who attended HIV testing, who reported ever having engaged in sexual intercourse, and who were identified in at least one of the three demographic surveys following testing were included in this analysis for a final sample size of 2770 women. For each woman, exposure time following HIV testing was calculated (the mean was 2 years), as well as the number of children born in the observation window. Based on information provided in the 2006/2007 detailed survey, parity prior to testing, marital

status, coital frequency per year, sexually transmitted infection symptoms, current contraceptive use, and history of miscarriage were calculated.

Table 1 provides HIV prevalence of sub-groups identified in the data. The varying prevalence levels of childbearing following the 2006 serological survey is an example of the striking association between HIV and fertility, prevalence declines for each group with each additional child born in the observation window (from 10.3% for no children, 5.9% for one, 1.4% for two, and 0% for three). Age has a nonlinear relationship with HIV for these women, with the highest rates between 25 and 40 (10.0% for 25-29, 12.0% for 30-34, and 12.4% for 35-39), and lowest in the late teens (1.6%). Divorced and widowed women have the highest levels of HIV prevalence of any marital status (16.5% and 22.6%, respectively). There is also a general trend of increasing HIV prevalence with the increased reporting of symptoms of sexually transmitted infections (symptoms include painful urination, bloody urine, genital discharge, and genital ulcers), prevalence ranges from 7.82% for no symptoms to 20% for all four. Less obvious trends appear with contraceptive use, coital frequency, miscarriage history, and parity.

Table 2 presents summary statistics separately for the HIV positive and negative women included in this paper. The final sample size of 2770 is composed of 233 HIV positive and 2537 HIV negative women. In the observation period following HIV testing, births per 1000 women-years of 217 for the HIV negative women and 123 for the HIV positive are statistically different at the 1% level. Explaining the difference in these rates is the goal of the following decomposition.

The summary statistics found in Table 2 illustrates the demographic dif-

ferences of the HIV negative and positive populations. These differences are striking in age and marital distribution, where the HIV negative population is skewed to the younger ages and is more likely to be either single or monogamous than HIV positive women (both significant at the 1% level). Symptoms of sexually transmitted infections are higher in the HIV positive population, with an average of 0.54 symptoms for HIV positive women and 0.45 for negative women, though this difference is not statistically significant. Contraceptive use, which has been hypothesized to be more prevalent in HIV positive women, is more common in the HIV negative population with 8% of women using any method compared to only 5% of the HIV positive population (significant at the 5% level). This may suggest an age effect in contraceptive use, where younger women (who are more likely to be HIV negative) are more likely to adopt contraceptive use. Coital frequency, while in the hypothesized direction, is not statically different between HIV positive and negative women with an average of 78.4 and 81.1 coital acts per year respectively. Finally, history of miscarriage and parity are not statistically different between groups.

5 Methods

The basic decomposition technique results in the estimation of individual variable contributions to endowments and coefficients. In this case, the effect of endowments describes how the fertility difference would decline if HIV positive and negative women had the same characteristics. The effect of coefficients estimates the different returns the two groups receive from their

characteristics, and how the fertility difference would shrink if HIV positive women had the same returns as HIV negative women.

Decomposition is a popular tool in the statistical analysis, particularly for ordinary least square regression models [3, 15]. Decompositions for other forms of regression models have been developed by numerous authors [1, 2, 16, 17, 19]. A recent contribution includes Powers, Yoshioka, and Yun's Stata command: mvdcmp, which allows for the decomposition of two Poisson regression models [16].

To decompose the difference between HIV positive and HIV negative women, each subpopulation was modeled using as a Poisson count with an outcome of number of children born in the observation window. The models were offset by the log of observation time for each woman. We also include variables for potential determinants of fertility including age, marital status, miscarriage history, contraceptive use, presence of STI symptoms, coital frequency, and parity. Categorical variables are used for age (in five year age groups), marital status (monogamous, polygamous, divorced, widowed, and single), miscarriage history (loss, no loss, or unknown), and current contraceptive use (including modern and traditional methods, modeled as yes or no). Continuous variables include coital frequency per year, number of sexually transmitted infection symptoms (which can range from 0-4), and parity. All control variables are reported by the woman at baseline.

6 Results

Table 3 provides the results of a detailed decomposition of HIV positive and negative sexually experienced women age 15-44 in Kisesa, Tanzania. The results show that 11.3% of the gap in fertility is attributable to the endowments of HIV positive and negative women, while 84.0% is attributable to differences in coefficients.

Age represents a large share of the total difference (27.6%), reflecting both the differences in age structure between the subpopulations and the fecundability at each age (which is illustrated by the high coefficient category). If HIV positive and negative women had similar proportions of their populations in the oldest age group, the gap in fertility rates would decline by 16%. The excess of young women in the HIV negative population is actually masking some of the difference in rates, if HIV positive women had the same proportion of young women, the gap would increase by 18%.

Marital status reflects similar results, though on a smaller scale (11.4%). The endowment effects of single and monogamous act in opposite directions, reflecting the heterogeneity in the HIV negative population composed of low fertility single women and high fertility monogamous women.

Contraceptive use, though low in both subpopulations, is in the opposite direction than the literature predicts and would increase the fertility rate differences between HIV positive and negative population by 25% if HIV positive women had the same use and response as HIV negative women. A possible explanation for this is that women may not know their HIV status, and are therefore not using contraception as a reaction to their status.

Sexually transmitted infections constitute a large amount of the percentage gap attributable to coefficients (49.4%). This would suggest that symptoms of sexually transmitted infections reduce fecundability more in HIV positive women than HIV negative, while the difference in fertility is not explained by the number of symptoms itself.

The effect of coital frequency (28.1%) is also seen in its effect on characteristics (27.1%), signifying a reduction in fecundability per act of coitus, which could be caused by both women and their partners. The effects of previous miscarriages and number of children at testing account for a small fraction of the fertility difference in HIV positive and negative women (6.1% and 0.40% respectively).

4.7% of the gap in the rates is attributed to difference in the constants of the models for the HIV positive and negative populations. This suggests that the current variables leave 4.7% of the difference in rates unaccounted for. We may be unable to ever fully explain gaps in fertility, as some biological and behavioral measures may be difficult or impossible to measure.

7 Discussion

The results found in the decomposition of fertility rates for HIV positive and negative women offer a detailed explanation for lower fertility observed in HIV positive population. Endowment effects such as age and marriage distributions align with current literature on the demographic differences between the subpopulations. The benefit of decomposition is that it allows for differences in the coefficients to contribute to the explanation of the

difference in rates. Returns to symptoms of sexually transmitted infections and coital frequency demonstrate the lower fecundability of the HIV positive population. The effect of HIV on fecundability may come through several pathways, both for males and females, including subfertility and infertility. This would also suggest that reducing STI infection in the general population would increase future fertility of women, especially those infected with HIV.

The extent of the difference in rates attributed to the coefficients suggests that many variables affect HIV positive and negative women differently, making it difficult to explain differences in birth rates via normal data analysis. Effects on fecundability have traditionally not been invoked when comparing fertility rates. Therefore, by employing this decomposition we have allowed for fecundability to play a more prominent role in fertility research.

8 Conclusion

Decomposition between two rates using a Poisson model allows for a more detailed explanation of fertility difference in subpopulations. The fertility gap between HIV positive and negative women is largely explained by the differences in coefficients, suggesting a reduction in fecundability in the HIV positive population. Age, marriage, STI symptoms, and coital frequency all contribute to the fertility gap found between HIV positive and negative women in Kisesa, Tanzania.

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Table 1: HIV Prevalence in Sexually Experienced Sub-Populations of Women 15-44 in Kisesa, Tanzania, 2006

Variable	HIV Prevalence (%)
Children Born in Observation Period	
0	10.3
1	5.9
2	1.4
3	0
Age (Years)	
15-19	1.6
20-24	5.6
25-29	10.1
30-34	12.0
35-39	12.4
40-44	7.1
Marital Status	
Monogamous	7.7
Polygamous	8.2
Divorced	16.5
Widowed	22.6
Single	4.4
Contraceptive Use	
No Current Use	8.7

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Table 1 – continued from previous page

Variable	HIV Prevalence (%)
Current Use	5.3
Coital Frequency	
0	12.9
<Once per Month	6.7
<Once per Week	8.0
<Twice per Week	7.3
<Thrice per Week	8.6
>Thrice per Week	9.5
STI Symptoms Number	
0	7.8
1	9.4
2	10.6
3	6.7
4	20.0
Miscarriage History	
Loss	8.4
Maybe	6.3
None	8.7
Previous Parity	
0	5.9
1	9.6
2	9.3

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Table 1 – continued from previous page

Variable	HIV Prevalence (%)
3	10.6
4	9.5
5	8.2
6	9.9
7	9.1
8 or More	3.4

Table 2: Summary Statistics of Ever Sex HIV Positive and Negative Women age 15-44 at Baseline Survey, Kisesa, Tanzania, 2006

X	HIV-Positive	HIV-Negative	Significant
N=2770	233	2537	
Exposure Time (Years)	462.37	5276.68	
Children Born in Observation	57	1144	
Births per 1000 Person Years	123.3	216.8	***
Age (Years)			***
15-19	0.02	0.12	
20-24	0.13	0.21	
25-29	0.24	0.20	
30-34	0.25	0.17	
35-39	0.21	0.14	
40-44	0.14	0.16	
Marital Status			***
Married Monogamous	0.58	0.63	
Married Polygamous	0.13	0.13	
Divorced	0.17	0.08	
Widowed	0.06	0.02	
Single	0.07	0.14	
Contraceptive Use	0.05	0.08	*
Coital Frequency (Per Year)	78	81	
STI Symptoms Count	0.54	0.45	
Miscarriage History			
Loss	0.17	0.17	
Unknown	0.07	0.09	
None	0.76	0.74	
Previous Parity	3.45	3.60	

*** p<0.01, ** p<0.05, * p<0.1

Table 3: Decomposition of Poisson Regressions of Proximate Determinants of Fertility (Percent Contribution), Kisesa, Tanzania, 2006

	Total	Endowments	Coefficients
Age (total)	**27.6**	**11.4**	**16.2**
15-19	15.8	18.1	-2.3
20-24	16.5	17.1	-0.6
25-29	-3.5	-9.0	5.5
30-34	-3.7	-6.6	3.0
35-39	7.3	8.0	-0.6
40-44	-4.8	-16.0	11.3
Marriage (total)	**11.5**	**5.4**	**6.1**
Monogamous	14.9	10.3	4.6
Polygamous	-2.3	0.6	-2.8
Divorced	4.6	0.0	4.6
Widowed	1.0	5.7	-4.7
Single	-6.7	-11.1	4.4
Miscarriage History (total)	**6.1**	**0.8**	**5.4**
Previous Loss	-5.2	0.0	-5.2
Unknown	1.6	0.2	1.3
No Loss	9.8	0.5	9.3
Contraceptive Use	**-24.4**	**-10.8**	**-13.7**
STI Symptoms	**46.0**	**-3.4**	**49.4**
Coital Frequency	**28.1**	**1.0**	**27.1**
Parity at Baseline	**0.4**	**6.9**	**-6.5**
Total	95.3	11.3	84.0
Constant (Unexplained)	4.7		4.7

Total may differ from E+C due to rounding