Paper II

Marked HIV prevalence declines in higher educated young people: evidence from population-based surveys (1995–2003) in Zambia

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Objectives: Higher educational attainment has been associated with a greater risk of HIV infection in sub-Saharan Africa. We investigated change over time in HIV prevalence by educational attainment in the general population.

Methods: The data stem from serial population-based HIV surveys conducted in selected urban and rural communities in 1995 ($n = 2989$), 1999 ($n = 3506$) and 2003 ($n = 4442$). Analyses were stratified by residence, sex and age-group. Logistic regression was used to estimate age-adjusted odds ratio of HIV between low (≤ 4 school years) and higher education (≥ 8 years) for the rural population and between low (≤ 7 school years) and higher education (≥ 11 years) for the urban population.

Results: There was a universal shift towards reduced risk of HIV infection in groups with higher than lower education in both sexes among urban young people [odds ratio (OR), 0.20; 95% confidence interval (CI), 0.05–0.73] in men and (OR, 0.33; 95% CI, 0.15–0.72) in women. A similar pattern was observed in rural young men (OR, 0.17; 95% CI, 0.05–0.59) but was less prominent and not statistically significant in rural women. In age 25–49 years, higher educated urban men had reduced risk in 2003 (OR, 0.43; 95%CI, 0.26–0.72) but this was less prominent in women.

Conclusions: The findings suggested a shift in the association between educational attainment and HIV infection between 1995 and 2003. The most convincing sign was the risk reduction among more educated younger groups where most infections can be assumed to be recent. The changes in older groups are probably largely influenced by differential mortality rates. The stable risk among groups with lower education might also indicate limitations in past preventive efforts.

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Introduction

In less than two decades, HIV has been transformed to an international emergency and a developmental disaster with social impacts as devastating as any war [1]. One such well documented impact has been on both the supply and demand of education in sub-Saharan Africa in particular [2,3]. In the earlier part of the HIV epidemic, higher educational attainment, socio-economic status and travel was associated with a greater risk of infection [4–8]. However, some studies in Zambia and Uganda have reported a shift towards reduced risk differentials amongst higher educated younger antenatal women [6,7,9,10]. This is of interest because knowledge, behaviour and behavioural change may be linked to educational level in that ability to understand and act on health promotional messages as well as attitudes that can influence one's health are increased[6,9,11].

Although economic and lifestyle changes that accompany educational attainment may have been associated with behaviours that increased the risk of HIV transmission,

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this relationship may dissolve as the epidemic spreads in any given population [6]. Highly educated groups may be the first to respond positively to preventive messages. However, educational attainment is still seen as a risk factor in some population sub-groups suggesting that there may be an interplay of several factors in this association [9]. Hence, the relationship between educational attainment and HIV infection and other associated factors should be investigated in order to understand the dynamics that might be associated with the HIV epidemic and how different groups respond.

In this study we investigate change over-time in the association between educational attainment and HIV infection in the general population in Zambia.

Methods

Population and sampling procedures

The data stem from serial population-based HIV surveys conducted in 1995, 1999 and 2003 in Kapiri Mposhi ($n = 892$, 1545 and 1861)) and Chelstone ($n = 2097$, 1961 and 2581) using stratified random-cluster sampling method. The detailed methods and major findings of the baseline survey conducted in 1995 and 1999 have been reported elsewhere [7,10]. The Zambian Census Population mapping system was used to establish the sampling frame which consisted of 24 standard enumeration areas (SEAs) with 2786 households in Chelstone and 26 SEAs (5225 households) in Kapiri Mposhi. These SEAs defined the primary sampling unit (clusters) of the study. Using 'probability proportional to size', 10 SEAs in Chelstone and five SEAs in rural Kapiri Mposhi were initially (1995) selected for this study. The numbers of SEAs in rural Kapiri Mposhi were increased to 10 in 1999 and 2003.All household members aged 15–59 years who lived in the selected clusters were listed and invited to participate in the study.

Data collection

In the sampled clusters (SEAs), a personal structured interview was carried out with all eligible and willing household members in order to collect information on education, sociodemographic characteristics and risk behaviours. Records on participants who were not found in the subsequent survey included date of leaving if migrated or date of death, whichever was applicable. The second part of the interview involved HIV testing using saliva.

Laboratory investigation

In the 1995 survey, all saliva samples were tested using Gacelisa HIV 1 & 2 (Welcome Diagnostics, Dartford, Kent, UK) and in addition, 450 randomly selected samples were tested using Bionor HIV-1 & 2 (BIONOR AS, Skien, Norway) magnetic particle assay following modifications for saliva. The two test kits showed a 99.8% agreement. The accuracy of Gacelisa was validated based on paired saliva and serum samples collected from 494 antenatal clinic attendees, and both sensitivity and specificity were 100% [12]. In the 1999 and 2003 follow-up surveys, samples were tested using Bionor HIV 1 & 2. Once collected, these specimens were stored in a central place and then transported once a week for testing at a national reference laboratory (University Teaching Hospital, Lusaka, Zambia).

Analysis

Statistical Package for Social Sciences Version 11.5 for Windows (SPSS, Chicago, Illinois, USA) and Epi-Info version 6.04 (Centres for Disease Control and Prevention, Atlanta, Georgia, USA) were used for overall and trend analyses respectively. Intercooled Stata version 8 (College Station, Texas, USA) was used to calculate confidence limits of all odds ratios (ORs) taking into account the cluster effect among the SEAs in the regression analysis. Educational level was measured using number of formal school years a respondent attained excluding night school and adult education years [13]. All analyses were stratified by age, sex and residence (rural versus urban). Prevalence was standardized for age using the Zambian census (2000) as standard population. Multivariate logistic regression was used to estimate age-adjusted odds ratios showing contrasts between lower and higher education. All logistic regression results were adjusted for age as a continuous variable in the 15–24 years category and for age group in the 25–49 years category. The rural versus urban proportions of people with ≥ 11 school years were 8 versus 32%, 6 versus 43% and 11 versus 55% in 1995, 1999 and 2003, respectively. Similarly, those with ≤ 4 school years were 27 versus 5%, 32 versus 5% and 28 versus 4% in 1995, 1999 and 2003, respectively. In view of these distribution differentials, when examining the effect of education on risk, the rural area was categorized into lower primary (0–4 years), upper primary (5–7 years) and secondary (≥ 8 years) education. However, the urban area was categorised into primary (0–7 years), junior secondary (8–10 years) and senior secondary ($\geq$ 11years). However, for rural–urban comparisons, only the 0–7 years, 8–9 years and $\geq$ 10years grouping was employed (see Fig. 1). Interactions were sought using the likelihood ratio test. Model diagnostics were evaluated using the maximum likelihood estimation (MLE) and the Hosmer–Lemeshow goodness-of-fit. All the variables in the study namely education, marital status, mobility (measured as travel frequency out of residential area), employment status and religion and age or age groups were included in the model since they were few.

Ethics

The survey protocol received clearance from the National AIDS Research Council and the University of Zambia Research and Ethics Committee. In addition, participation

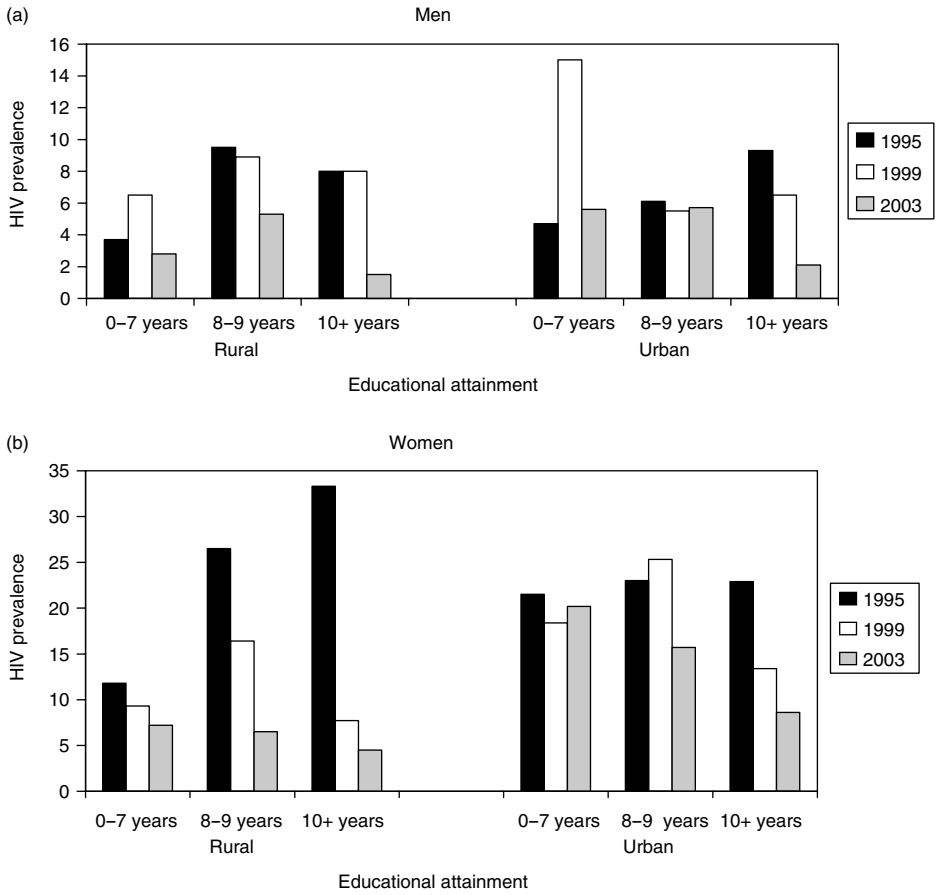

Fig. 1. Patterns of HIV prevalence in Kapiri Mposhi (rural) and Chelstone (urban) among young people, 15–24 years: 1995–2003. *P* value for linear trend among groups with ≥ 10 school years: (a) men: rural, $\chi^2 = 2.75$, $P = 0.09$ and urban, $\chi^2 = 15.5$, $P = 0.000$; (b) women: rural, $\chi^2 = 7.37$, $P = 0.0006$ and urban, $\chi^2 = 22.2$, $P = 0.000$. Age-adjusted odds ratio and 95% confidence interval of infection for young people with ≥ 10 school years, with 0–7 years as reference in 2003 (a) men: rural, 0.53 (0.1, 2.78) and urban, 0.33 (0.13, 0.86); (b) women: rural, 0.55 (0.24, 1.3) and urban, 0.28 (0.16, 0.48).

in the population-based HIV survey was based on informed consent except in 2003 when written consent was used also. Respondents were counselled and informed that the testing was purely for research purposes and was to be handled anonymously. However, the respondents who showed interest to know their status were offered voluntary counselling and testing (VCT) and a blood specimen was collected for serum-based HIV testing. In the 1995 and 1999 surveys, respondents were offered VCT either at home or at the clinic depending on their preference. However in 2003, VCT was only provided at home in light of acceptability findings from earlier studies [12,14,15].

Results

Participation and distribution

Participation in the 1995 and 1999 population-based surveys has already been published [7,10]. In 2003, the total number of adults listed as rural residents were 2705 (1301 men; 1404 women) and urban residents were 4086 (1861 men; 2225 women). Non-participation in testing was due to absence or refusals. Those absent (mostly either at school, in hospital or travelled away temporarily) were 25.5% (334) of the rural men, 11.8% (166) in rural women, 28.3% (527) among urban men and 14% (311) in urban women. Of the *de facto* eligible population among rural men, rural women, urban men and urban women, saliva refusal rates were 3.5, 5.6, 9.8 and 11.1%, respectively. The respondents that did not have complete information on education (9.1% rural men, 8.6% rural women, 8.7% urban men and 7.2% urban women) were excluded in the final analysis. This reduced the total number of respondents with all required information to 4442 in 2003, whereas it was 2989 and 3506 in 1995 and 1999 and respectively. The mean age in years was 32.5 for rural men and 26.9 for urban men, whereas it was 30.9 and 26.2 for rural and urban women, respectively. Educational attainment differed significantly between rural and urban areas by sex. The pooled mean urban–rural difference was 3.97 [95% confidence interval (CI)

Table 1. Age-specific rural/urban distribution of the means and median years of schooling in age 15–49 years, 1995–2003.

		School years				
		Rural		Urban		
Sex	Year	Mean (95%CI)	Median (IQR)	Mean (95%CI)	Median (IQR)	Urban–rural difference in means (95%CI)
Men	1995	6.69 (6.36, 7.02) $n = 398$	7.0 (4–9)	9.98 (9.97, 10.18) $n = 801$	10.0 (8–12)	3.29 (2.94, 3.66)
	1999	6.65 (6.40, 6.90) $n = 652$	7.0 (4–9)	10.72 (10.53, 10.92) $n = 743$	12 (9–12)	4.07 (3.76, 4.39)
	2003	7.30 (7.06, 7.55) $n = 813$	7.0 (5–9)	11.12 (10.94, 11.29) $n = 1041$	12 (9–13)	3.82 (3.52, 4.11)
	All years	6.94 (6.78, 7.09) $n = 1863$	7.0 (4–9)	10.65 (10.54, 10.76) $n = 2585$	11 (9–12)	3.7 (3.53, 3.89)
Women	1995	5.77 (5.48, 6.07) $n = 495$	6.0 (2–7)	8.64 (8.45, 8.83) $n = 1296$	9.0 (7–10)	2.86 (2.51, 3.22)
	1999	5.15 (4.94, 5.36) $n = 893$	5.0 (2–7)	9.21 (9.04, 9.38) $n = 1218$	9.0 (7–12)	4.06 (3.79, 4.32)
	2003	5.82 (5.62, 6.02) $n = 1048$	6.0 (3–7)	10.10 (9.95, 10.26) $n = 1540$	10.0 (8–12)	4.28 (4.03, 4.53)
	All years	5.57 (5.44, 5.69) $n = 2436$	6.0 (3–7)	9.37 (9.26, 9.47) $n = 4054$	9.0 (7–12)	3.80 (3.63, 3.96)

IQR, interquartile range; CI, confidence interval.

3.85–4.09] school years. The rest of the distribution by education is illustrated in Table 1.

HIV infection trends

In the age group 15–49 years, there was a universal shift towards declining prevalence of HIV infection in respondents with higher education during this period, as illustrated in Table 2. Prevalence declined from 30.2 to 11.7% in urban men (trend, $P < 0.01$), 34.3 to 17.5% in urban women (trend, $P < 0.01$) and 29.7 to 17.3% in rural women (trend, $P < 0.01$), but showed a less pronounced decline in rural men (18.1 to 15.3%, trend, $P < 0.01$). In sharp contrast, prevalence remained stable or even increased in lower educated groups. In 2003, urban populations with higher education had lower odds of infection than lower educated groups in men (OR, 0.45; 95% CI, 0.27–0.74) and in women (OR, 0.65; 95% CI, 0.42–0.98).

The overall pattern of reduced prevalence among groups with higher education was more evident in the age group 15–24 years. In the urban area, prevalence changed in higher educated young women from 21.2% in 1995, 16.1% in 1999 to 8.5% by 2003 (trend, $P < 0.01$). In the same period, prevalence changed from 10.9% in 1995, 6.9% in 1999 to 1.4% by 2003 in young men (trend, $P < 0.01$). By 2003, higher educated groups had reduced odds of infection than lower educated groups: OR, 0.33 (95% CI, 0.15–0.72) in young women and OR, 0.20 (95% CI, 0.05–0.73) in young men. In the rural area the pattern was similar. In higher educated young women, prevalence declined from 28.1% in 1995 to 5.6% by 2003 (trend, $P < 0.01$) and from 8.9% in 1995 to 3.2% by 2003 (trend, $P < 0.01$) in young men, see Table 2.

Out-of-school urban young women aged 15–19 years were at higher risk than those still in school in 1995 (OR, 3.3; 95% CI, 1.46–7.46), but this was less pronounced later in 1999 (OR, 2.1; 95% CI, 0.95–4.5) and in 2003 (OR, 2.1; 95% CI, 0.98–4.48). There were no significant differences in HIV risk between in-school and out-of-school respondents' among urban young men and rural respondents (both sexes). This predominantly declining pattern was also observed in age group 25–49 years. In urban women with ≥ 11 school years, prevalence changed from 45.6% in 1995; 39.9% in 1999 to 29.0% by 2003. However, in the group with ≤ 7 school years, prevalence remained stable; 27.3% in 1995, 26.9% in 1999 and 31.6% by 2003. Among urban men in this age group, higher educated groups had lower odds than lower educated groups; OR, 0.43 (95% CI, 0.26–0.72) for those with ≥ 11 school years and OR 0.53(95%CI 0.27–1.00) for those with 8–10 school years. However, higher educated groups remained at higher odds of infection than groups with less schooling for both sexes in the rural area.

In order to compare rural and urban HIV prevalence pattern differences in young people aged 15–24 years, one educational grouping (0–7, 8–9 and ≥ 10 school years) was used and Fig. 1 illustrates this. Prevalence patterns over time were similar in rural and urban populations, remaining stable in lower educated groups but declining in higher educated groups. In respondents with ≥ 10 school years, prevalence declined from 8.0% (2/25) to 1.5% (1/68) in rural young men, 9.3% (15/62) to 2.1% (9/428) in urban young men, 33.3% (5/15) to 4.5% (2/44) in rural young women and 22.9% (43/188) to 8.6% (43/497) in urban young women between 1995 and 2003. In 2003, urban young people with ≥ 10 school years had lower odds of infection than those with 0–7 school years: OR, 0.28 (95% CI, 0.16–0.48) for young women, and OR, 0.33 (95% CI, 0.13–0.86) for young men. In the rural area, although higher educated groups had reduced odds of infection compared with lower educated respondents, it was not statistically significant: OR, 0.55 (95% CI, 0.24–1.3) for young women and OR, 0.53 (95% CI, 0.10–2.78) for young men. The proportion of infected young women remained higher in urban than rural areas, pooled OR, 2.30 (95% CI, 2.02–2.63). The bulk of the infected group consisted of those aged 20–24 years who had significantly higher odds of infection for men (OR, 1.75; 95% CI, 1.20–2.54) and for

Table 2. Changes in the association between years of schooling and HIV prevalence in age 15–49, 1995–2003; results of multivariate logistic regression.

				1995		1999		2003	
Age group (years)	Location	Sex	School years	Prevalence (*n*)	OR[a] (95%CI)	Prevalence (*n*)	OR[a] (95%CI)	Prevalence (*n*)	OR[a] (95%CI)
15–24	Urban	Men	0–7 years	4.7% (5/106)	1.00	15.0% (9/60)	1.00	5.6% (4/71)	1.00
			8–10 years	5.5% (11/199)	1.31 (0.21, 8.2)	5.3% (9/171)	0.38 (0.13, 1.1)	5.5% (11/200)	1.72 (0.66, 4.47)
			≥ 11 years	10.9% (14/128)	1.81 (0.37, 8.8)	6.9% (14/201)	0.37 (0.12, 1.16)	1.4% (5/351)	**0.20 (0.05, 0.73)**
		Women	0–7 years	21.5% (59/274)	1.00	18.4% (32/174)	1.00	20.2% (35/173)	1.00
			8–10 years	23.8% (66/277)	1.27 (0.89, 1.79)	20.0% (50/250)	1.18 (0.56, 2.48)	13.7% (33/240)	0.75 (0.36, 1.56)
			≥ 11 years	21.2% (31/146)	0.53 (0.28, 1.01)	16.1% (35/217)	0.61 (0.25, 1.49)	8.5% (36/423)	**0.33 (0.15, 0.72)**
	Rural	Men	0–4 years	0.0% (0/34)	–	4.2% (3/72)	1.00	6.1% (4/66)	1.00
			5–7 years	5.5% (4/73)	1.00	8.7% (10/115)	2.25 (0.57, 8.8)	0.9% (1/114)	**0.17 (0.05, 0.59)**
			≥ 8 years	8.9% (6/67)	3.02 (0.43, 21.2)	8.6% (7/81)	2.6 (0.63, 10.7)	3.2% (4/125)	1.28 (0.29, 5.59)
		Women	0–4 years	8.5% (5/59)	1.00	9.0% (11/122)	1.00	8.1% (12/149)	1.00
			5–7 years	13.6% (15/110)	1.22 (0.17, 8.5)	9.5% (18/190)	1.32 (0.37, 4.67)	6.5% (12/184)	0.59 (0.23, 1.52)
			≥ 8 years	28.1% (18/64)	3.1 (0.84, 11.5)	14.7% (10/68)	2.59 (1.0, 6.73)	5.6% (7/121)	0.77 (0.28, 2.1)
25–49	Urban	Men	0–7 years	36.2% (21/58)	1.00	25.7% (9/35)	1.00	41.3% (19/46)	1.00
			8–10 years	33.3% (30/90)	0.87 (0.47, 1.59)	23.7% (14/59)	0.76 (0.38, 1.5)	29.8% (20/67)	0.53 (0.27, 1.0)
			≥ 11 years	41.4% (91/220)	1.14 (0.52, 2.52)	37.3% (81/217)	1.64 (0.82, 3.26)	23.5% (72/306)	**0.43 (0.26, 0.72)**
		Women	0–7 years	28.5% (63/220)	1.00	26.7% (47/176)	1.00	34.6% (55/159)	1.00
			8–10 years	41.1% (86/209)	**1.6 (1.12, 2.28)**	42.4% (86/203)	**2.01 (1.17, 3.61)**	36.4% (78/214)	0.97 (0.54, 1.74)
			≥ 11 years	45.6% (77/169)	**2.01 (1.1, 3.77)**	39.9% (79/198)	**1.45 (1.0, 2.27)**	29.0% (96/331)	0.67 (0.52, 1.1)
	Rural	Men	0–4 years	16.0% (8/50)	1.00	13.8% (11/80)	1.00	17.8% (18/101)	1.00
			5–7 years	23.5% (24/102)	1.19 (0.47, 2.99)	19.7% (34/173)	1.17 (0.24, 5.6)	15.1% (30/199)	0.71 (0.34, 1.44)
			≥ 8 years	26.8% (19/71)	1.54 (0.38, 6.21)	16.8% (22/131)	1.03 (0.34, 3.1)	22.6% (47/208)	1.35 (0.61, 2.97)
		Women	0–4 years	12.2% (12/98)	1.00	18.3% (40/219)	1.00	16.1% (33/205)	1.00
			5–7 years	17.0% (17/100)	3.19 (0.43, 23.3)	17.7% (37/209)	0.82 (0.46, 1.47)	18.8% (48/255)	1.39 (0.86, 2.25)
			≥ 8 years	31.3% (20/64)	**7.4 (1.74, 13.8)**	15.3% (13/85)	0.69 (0.21, 2.21)	27.6% (37/134)	**2.31 (1.13, 4.47)**
15–49	Urban	Men	0–7 years	15.9% (26/164)	1.00	8.9% (18/95)	1.00	19.7% (23/117)	1.00
			8–10 years	14.2% (41/289)	0.87 (0.39, 1.9)	10.0% (23/230)	0.51 (0.23, 1.12)	11.6% (31/267)	**0.73 (0.48, 0.99)**
			≥ 11 years	30.2% (105/348)	1.49 (0.67, 3.3)	22.7% (95/418)	0.93 (0.45, 1.9)	11.7% (77/657)	**0.45 (0.27, 0.74)**
		Women	0–7 years	24.6% (122/495)	1.00	22.6% (79/350)	1.00	27.1% (90/332)	1.00
			8–10 years	31.3% (152/486)	**1.45 (1.11, 1.89)**	30.0% (136/453)	**1.68 (1.1, 2.67)**	24.4% (111/454)	0.92 (0.54, 1.56)
			≥ 11 years	34.3% (108/315)	**1.59 (1.16, 2.2)**	27.5% (114/415)	1.33 (0.86, 2.0)	17.5% (132/754)	**0.65 (0.42, 0.98)**
	Rural	Men	0–4 years	9.5% (8/84)	1.00	9.2% (14/152)	1.00	13.2% (22/167)	1.00
			5–7 years	16.0% (28/175)	**1.61 (1.19, 2.16)**	15.3% (44/288)	1.43 (0.41, 4.98)	9.9% (31/313)	0.59 (0.29, 1.18)
			≥ 8 years	18.1% (25/138)	2.27 (0.98, 5.26)	13.7% (29/212)	1.25 (0.46, 3.40)	15.3% (51/333)	1.17 (0.58, 2.38)
		Women	0–4 years	10.8% (17/157)	1.00	14.9% (51/341)	1.00	12.7% (45/354)	1.00
			5–7 years	15.2% (32/210)	1.95 (0.58, 6.54)	13.8% (55/399)	0.95 (0.52, 1.75)	13.7% (60/439)	1.15 (0.66, 2.0)
			≥ 8 years	29.7% (38/128)	**4.44 (1.75, 11.2)**	15.0% (23/153)	1.0 (0.52, 1.93)	17.3% (44/255)	1.87 (0.94, 3.73)

[a]OR denotes odds ratio adjusted for age in age group 15–24 years but for age group in 25–49 and in 15–49 years categories. All confidence intervals (CI) adjusted for clustering effect using Intercooled Stata version 8 with the standard enumeration area (SEA) as clusters. The confidence limits in bold are statistically significant, $P < 0.05$. Sample sizes: $n = 2989$ in 1995; $n = 3506$ in 1999; $n = 4442$ in 2003. Variables included in the model were education, age as continuous variable (15–24), age group, marital status, religion, mobility (travel) and employment status.

women (OR, 3.24; 95% CI, 2.63–4.0) than the 15–19-year-old individuals.

Discussion

The findings have revealed a changing pattern of HIV prevalence by educational attainment, showing a universal shift towards reduced risk of infection in groups with higher education during the period 1995–2003. The most convincing sign was the marked risk reduction among more educated younger groups where most infections can be assumed to have been acquired recently. Furthermore, education uniquely appeared to be an effective preventive factor in reducing the likelihood of HIV infection in both sexes among young people. This is in contrast to what was seen in earlier studies in which higher educational attainment was associated with higher risk of infection and showed a differential picture by gender [4,6,7,10,16,17]. However, this has confirmed findings of a similar shift reported in Uganda and in both young ANC women and the general population in Zambia [6,10,11,18]. The prevalence changes in older groups are probably largely influenced by differential mortality rates [19]. The stable or rising risk in the groups with lower education might also indicate serious limitations in past preventive efforts.

HIV prevalence often is a reflection of the balance between incidence, migration and mortality. The changes noticed in young people aged 15–24 years where mortality is low, is seen as a marker of incidence. The possible explanations for these findings will need to be explored further, but the prevalence decline may suggest a positive response to preventive messages on behaviour change. We are aware that HIV-related intervention programs in Zambia intensified since the early 1990s [20]. Most of these programs were in English and concentrated largely in urban areas. In addition, high profile people like Dr K. D. Kaunda (first republican president of Zambia) joined the fight with enormous vigour. The preventive messages and activities seem to have had a positive impact in reversing HIV risk patterns, initially with higher social groups, and this will hopefully diffuse to lower social groups as proposed in the Diffusion of Innovation theory [21]. This theory states in part that, 'awareness messages and the opinion of leaders are said to influence behaviour change by employing cultural contexts and language codes that are community specific in the campaigns' [21–23]. Lifestyles, cultural practices and communication patterns may significantly differ by educational attainment. However, whenever change happens, it does most probably begin with the higher educated groups. Hence the change we have seen among higher educated groups responding positively to preventive messages in both rural and urban areas may be a stage of progression. These findings further suggest that education may have been a strong factor in reducing HIV risk, supporting the 'education vaccine' view [24,25]. Delaying sexual debut is one such example of behaviour change which is associated with higher educational attainment [26–28]. Education may thus be central in reducing both the risk and vulnerability to HIV infection [5,6,29,30].

Although women have been most affected historically, this study has demonstrated that HIV prevalence drastically declined in higher than lower educated young people in both sexes. The main common feature in these young women and men was similar educational level, suggesting that education may have been the factor behind this reduction in both sexes [31]. Among higher educated young urban women, prevalence declined from 21.2% in 1995 to 8.5% in 2003 and declined from 10.9% in 1995 to 1.4% in 2003 in urban men. In the rural area, the direction of change was similar by sex. In women, prevalence drastically changed from 28.1%, in 1995 to 5.6% by 2003, whereas it changed from 8.9% to 3.2% in higher educated young men, in the same period (Table 2). These changes may be suggesting that policies that aimed at increasing proportion of women enrolling in school and encouraging them to stay on at school may be bearing fruit and need to be strengthened. As a result, this may have improved their requisite capacity to reduce the likelihood of getting infected with HIV. Sadly however, out-of-school adolescents tended to have higher odds of infection than those still at school. Furthermore, out-of-school women had three to four times higher prevalence than out-of-school men in both rural and urban areas. The sex differentials seen here are a reminder of sex-related imbalances seen across the epidemic in the sub-region with women having higher odds of infection than men even across educational groups [31–33]. Explanations for the high prevalence in women are unclear. Possibilities include low enrolment rates of women in school in comparison with their male counterparts, earlier sexual debut for girls and with older men and higher likelihood for women to trade sex for gifts thereby putting them at higher risk [31,34].

Although the education distribution among our respondents reflects the national pattern, non-response may be distributed differently [35]. Differential non-response over time could have biased our estimates but it is difficult to estimate the magnitude and direction of this effect. However, we are convinced that it is unlikely to be an important factor explaining the marked and consistent differential changes. We also feel that the protective effect of education in the young people could even have been under-estimated due to the fact that those not found because of school attendance are less likely to be infected [18]. However, we are persuaded to believe that if this bias was present, its effect was very minimal. We also realize that any effect of higher education on risk of infection is likely to be exerted through mediator factors such as more consistent condom use, lower likelihood of sexually

transmitted infections and less number of sexual partners, as well as increased contraceptive use [5,36]. In view of this, some authors suggest that the relationship between these factors and education ought to be checked in the same population and during the same time as it may provide an opportunity to check the correlation between reported and actual behaviour with prevalence [18,37]. Although this part has not been reported here, it has been addressed in another yet to be published article focusing on sexual behaviour patterns of this population.

The changes observed in the 15–24-year-old respondents suggest that the availability of HIV preventive information was useful in forming their sexual behaviour, as they became sexually active after this critical information became well known [4]. However, in this study it is not possible to draw conclusions on how education does this. Elsewhere, it has been suggested that education does not only work by providing knowledge, fostering attitudes and conferring skills that are useful in reducing both the risks of and people's vulnerability to HIV infection, but that the general impact of education in and of itself is the most significant factor [25]. Finding that higher educated groups especially among the young people are at less risk of infection than lower educated ones suggests that this is the group that responded when interventions were launched in Zambia. This may provide many new challenges and opportunities for prevention. One among many challenges is how to improve preventive programs so as to target the hard-to-reach lower educated and predominantly rural populations [36]. Improving these programs seems logical because unchanging risks observed may be an indication of failed or poorly implemented preventive efforts in particular local settings or populations. Another possible challenge is strengthening existing educational structures in order to improve access for universal basic education among lower educated groups since the presence of education can now be linked to lower risk of HIV [1,38].

Despite these challenges, opportunities seem to open up. In this study changes were most pronounced in the respondents who had attained secondary education. This may have policy implications in that the current focus on universal basic education probably ought to be reshaped by aiming at attaining universal secondary education instead. This will probably arm the young population with a better requisite tool for behaviour change. If this is done, complimented with the addition of appropriate sexual education, which is still missing in the curricula of regular schools, night schools as well as adult education and life-skills centres, the desirable consequence of curbing newer infections may be realised in groups who may not have responded so far [25,39]. Kelly and Coombe noted that 'education and schooling provide almost the only known antidote to HIV infection. Making this antidote universally available implies making appropriate education universally available' [25].

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