HIV-1 and HIV-2 prevalence and associated risk factors among postnatal women in Harare, Zimbabwe

J. H. HUMPHREY[1]*, K. J. NATHOO[2], J. W. HARGROVE[3], P. J. ILIFF[2],
K. E. MUTASA[3], L. H. MOULTON[1], H. CHIDAWANYIKA[3], L. C. MALABA[2],
L. S. ZIJENAH[2], P. ZVANDASARA[2], R. NTOZINI[3], C. D. ZUNGUZA[4],
B. J. WARD[5], and the ZVITAMBO Study Group†

[1] *Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, USA*
[2] *University of Zimbabwe Faculties of Medicine and Science, Harare, Zimbabwe*
[3] *ZVITAMBO Study Group, Harare, Zimbabwe*
[4] *Harare City Health Department, Harare, Zimbabwe*
[5] *Research Institute of the McGill University Health Center, Montreal, Quebec, Canada*

SUMMARY

Studies of antenatal women form the predominant source of data on HIV-1 prevalence in Africa. Identifying factors associated with prevalent HIV is important in targeting diagnostic services and care. Between November 1997 and January 2000, 14 110 postnatal women from Harare, Zimbabwe were tested by ELISAs reactive to both HIV-1 and HIV-2; a subset of positive samples was confirmed with assays specific for HIV-1 and HIV-2. Baseline characteristics were elicited and modelled to identify risk factors for prevalent HIV infection. HIV-1 and HIV-2 prevalences were 32·0% (95% CI 31·2–32·8) and 1·3% (95% CI 1·1–1·5), respectively; 4% of HIV-1-positive and 99% of HIV-2-positive women were co-infected. HIV-1 prevalence increased from 0% among 14-year-olds to >45% among women aged 29–31 years, then fell to <20% among those aged >40 years. In multivariate analyses, prevalence increased with parity, was lower in married women than in single women, divorcees and widows, and higher in women with the lowest incomes and those professing no religion. Adjusted HIV-1 prevalence increased during 1998 and decreased during 1999. Age modified the effects of parity, home ownership and parental education. Among older women, prevalence was greater for women who were not homeowners. Among younger women, prevalence increased with parity and low parental education. None of these factors distinguished women co-infected with HIV-2 from those infected with HIV-1 alone. Prevalent HIV-1 infection is associated with financial insecurity and weak psychosocial support. The ZVITAMBO study apparently spanned the peak of the HIV-1 epidemic among reproductive women in Harare.

INTRODUCTION

Women attending antenatal clinics (ANCs) form the predominant source of data on HIV-1 prevalence in Sub-Saharan Africa [1]. These data are important in themselves because young women make up a large proportion of the total adult population and because the epidemic among antenatal women directly

---

* Author for correspondence: Dr J. H. Humphrey, 615 N. Wolfe Street, Room 2043, Baltimore, MD 21205, USA.
(Email: jhumphrey@zvitambo.co.zw)
† Members of the ZVITAMBO Study Group are listed in the Appendix.

influences the current and future epidemic among children. ANC prevalence data also provide useful estimates of prevalence among the general population, after adjustment for factors such as age, parity, education, housing, marital status and occupation [2, 3].

Zimbabwe is a country of 12 million people, one-third of them urban dwellers, at the epicentre of the HIV-1 epidemic in Africa. HIV-1 prevalence among women attending antenatal clinics in Harare, the capital city, was 18% (±1·2%) and 30·4% (±1·3%) in two research studies conducted in 1990 [4] and 1995 [5], respectively, and was 30·0% [95% confidence interval (CI) 26·9–33·2, $n = 841$], 30·6% (95% CI 27·8–33·5, $n = 1043$), 24·2% (95% CI 21·7–26·8, $n = 1097$) and 20·2% (95% CI 17·9–22·7, $n = 1118$) in the Harare sites of the national sentinel surveys conducted by the Ministry of Health and Child Welfare in 2000, 2001, 2002, and 2004, respectively [6].

As part of ZVITAMBO, a clinical trial of post-partum vitamin A supplementation among 14 110 women and their neonates enrolled between November 1997 and January 2000 in Harare [7–10], we examined maternal prevalence of HIV-1 and HIV-2 infections. HIV-2 prevalence has been estimated in Harare in only one previous study among all women presenting for first booking between May 1994 and June 1995 at one of four ANCs which also served as recruitment sites for ZVITAMBO. Mothers were tested for HIV by Capillus test, confirmed by Western Blot, ELISA, and an HIV rapid test. Of the 1168 women in that study, 355 (30·4%) were HIV-1-positive and 88 (7·5%) were HIV-2-positive; all HIV-2- infected women were co-infected with HIV-1. Distinguishing the infections can be important because virulence and horizontal and vertical transmission are lower for HIV-2 relative to HIV-1 [11]. Co-infection may also require modified antiretroviral therapy [12]. Changes over time in the contribution of HIV-2 infection to total HIV prevalence may, therefore, have important public health implications. Since standard HIV ELISA tests react to the presence of both HIV-1 and HIV-2 antibodies, we incorporated strain-specific ELISAs into our testing protocol.

The second objective of the current analysis was to identify risk factors of prevalent HIV-1 and HIV-2 infection. Given the usually long and debilitating course of HIV, characteristics associated with prevalent infection may reflect the effects of living with the infection rather than pre-existing characteristics associated with an increased risk of acquiring HIV.

Nevertheless, the identification of factors associated with HIV prevalence is important in describing sub-groups most affected by the disease that should be targeted for diagnosis, counselling, and care.

METHODS

As part of the ZVITAMBO trial, 14 110 postnatal mothers and their neonates were enrolled within 96 h of delivery at one of 14 maternity clinics and hospitals in greater Harare between 25 November 1997 and 30 January 2000. During the recruitment period, all deliveries at the sites were documented and reviewed for eligibility. Mother–baby pairs were eligible if neither had an acutely life-threatening condition, the baby was a singleton with birth weight $\geqslant 1500$ g, and the mother planned to stay in Harare after delivery. Eligible mothers were invited to enrol following written informed consent. Women refusing consent were asked to provide maternal age and parity, and infant birth weight, without any personal identifiers.

At recruitment, each mother provided information for herself and her husband (or father of the child), on education and occupation, and her age, parity, religion, housing, and family income. Income in Zimbabwe dollars was inflation-adjusted and converted to US dollars. Maternal serum or plasma was tested for HIV at baseline by two ELISA tests (HIV 1.0.2 ICE: Murex Diagnostics, Edenvale, South Africa, and GeneScreen HIV 1/2: Sanofi Diagnostics Pasteur, Johannesburg, South Africa) run in parallel. Discordant pairs of ELISA test results were resolved by Western blot assay (HIV Blot 2.2: Genelabs Diagnostics SA, Geneva, Switzerland), interpreted according to the manufacturer's guidelines. Sensitivity and specificity of the two ELISAs were assessed among 477 specimens; the gold standard was the concurrent ELISA or Western blot result. Respectively, sensitivity and specificity were 98·9% and 100% for Murex, and 100% and 97·2% for Gene-Screen. Mothers were followed up at 6 weeks, 3 months, and then three-monthly for 12–24 months. Women testing HIV-positive at baseline were re-tested at their next visit by Murex VK57 and Murex GE22 assays, which specifically detect HIV-1 and HIV-2, respectively.

Data analysis was performed using STATA 8.0 (StataCorp, College Station, TX, USA) and SAS 8.2 (SAS Institute Inc., Cary, NC, USA). Logistic regression was used to investigate the association between baseline factors and each of the HIV infections.

All maternal baseline demographic variables were added singly into the model, starting with the variable that accounted for the greatest proportion of the variance in univariate analysis. Variables were considered statistically significant at the $\alpha = 0.05$ level; variables that were rejected at a given step were re-tested at each stage when a further variable was added to the model. In particular, once the final model had been selected, each variable that had been previously rejected was retested to ensure that it still had no significant effect. Potential effect modification by maternal age was modelled by logistic regression; interaction terms were considered significant at the $\alpha = 0.10$ level.

The Medical Research Council of Zimbabwe, The Medicines Control Authority of Zimbabwe, The Johns Hopkins Bloomberg School of Public Health Committee on Human Research, and the Montreal General Hospital Ethics Committee approved the study.

RESULTS

The 14 110 enrolled women comprised ~20% of the nearly 70 000 deliveries occurring during the recruitment period at our recruitment sites (Fig. 1). Of the 48 808 mother–infant pairs considered for enrolment, 31% were excluded. Of these, 40% failed to meet medical or birth weight inclusion criteria, 56% had their primary residence outside Harare, and 4% were excluded for other reasons. Women failing to meet medical inclusion criteria were presumably more likely to have been HIV-infected than those enrolled, since HIV-positive women are at greater risk of pregnancy-related morbidity and adverse obstetric outcome [13, 14]. However, women excluded based on residence may have been at lower risk of HIV since prevalence in 2000 was 29.8% in rural compared to 34.6% in urban areas of Zimbabwe [15]. Among the 33 576 eligible women, 19 867 (59%) did not consent to enrol, but agreed to provide their age and parity, and their infant's birth weight. Compared with women enrolled in the study, non-consenting women were ~1 year younger [24.3 years (95% CI 24.2–24.3) vs. 24.5 years (95% CI 24.4–24.6)] and their babies were ~10 g heavier [2997 g (95% CI 2991–3004) vs. 2973 g (95% CI 2965–2980)], but had the same mean parity (2.07, 95% CI 2.05–2.09). Thus, although some aspects of our eligibility criteria and consent procedures might have produced an underestimate and others an overestimate of the underlying
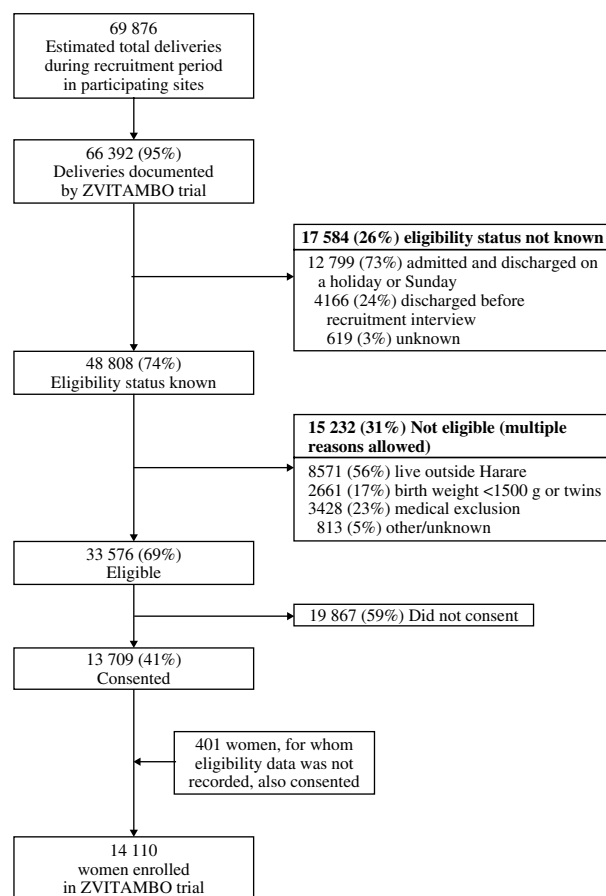
prevalence, on balance, the prevalence in the ZVITAMBO cohort is likely to have been generally comparable to that of all antenatal women in Harare in 1998–1999.† A study in eastern Zimbabwe conducted in 1998–2000 found that HIV prevalence of ANC attendees closely approximated that among all women aged 15–44 years [16], indicating that the ZVITAMBO data is also likely to reflect the general population of sexually active women in Harare during 1998–1999.

Prevalence of HIV-1 and HIV-2 infection

Of the 14 110 enrolled women, 53 (0.4%) mothers were indeterminate for HIV-1 or HIV-2 infections at delivery and were excluded from further analyses. Of the remainder, 4495 (32.0%, 95% CI 31.2–32.8) were HIV-positive and 9562 (68.0%) were HIV-negative. HIV-positive women were older, of higher parity,

Fig. 1. Flow of participants through study.

---

† Indeed, our estimate did not significantly differ from the prevalence measured in Harare sites in the 1999–2000 MoHCW antenatal sentinel survey (30.0%, 95% CI 26.9–33.2) [6].

Table 1. *Characteristics of HIV-1 positive and negative mothers and their husbands (figures in parentheses indicate the 95% confidence interval in each case)*

|	HIV status		
	Positive	Negative	P
Mean age of mothers (years)	25·6 (25·4–25·7)	24·1 (24·0–24·2)	<0·0001
Mean parity	2·27 (2·24–2·31)	1·97 (1·95–2·00)	<0·001
Mean family income (US$/month)	111·2 (106·5–115·9)	115·5 (112·3–118·7)	0·14
Mother married (%)	89·9 (89·0–90·8)	92·9 (92·4–93·4)	<0·001
Mother lives in own house (%)	9·8 (9·0–10·7)	10·7 (10·1–11·3)	0·12
Mother <8 years schooling (%)	19·7 (18·5–29·9)	17·3 (16·6–18·1)	0·001
Father <8 years schooling (%)	11·3 (10·4–12·2)	8·9 (8·3–9·5)	<0·001
Father a professional (%)	14·1 (13·1–15·2)	17·5 (16·7–18·3)	<0·001
Mother states no religion (%)	5·9 (5·2–6·6)	4·6 (4·2–5·1)	0·002
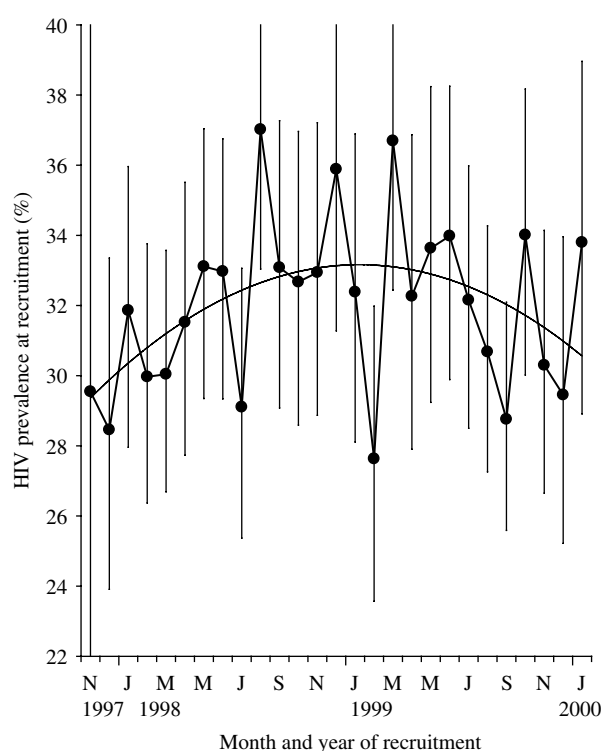
Fig. 2. HIV-1 prevalence among women enrolled in the ZVITAMBO trial as a function of month of recruitment. Error bars indicate the 95% confidence interval about the estimate. Function fitted: Prevalence (%) = 29·1 + 0·552t – 0·0188t^2 where t is the month of recruitment. Standard errors for linear and quadratic coefficients 0·23 and 0·008, respectively. $P < 0·05$ (t test) in each case.

had lower family incomes, and were less likely to be married, to own their own house, have a stated religion, or be married to a professional man (Table 1). They and their husbands were also less likely to have been to high school.

Among the 10 653 women recruited during the first 21 months of recruitment (November 1997 to July 1999) who provided an unequivocal HIV result at baseline, 3444 (32·3%) tested positive and 2353 (68·3%) of these were available for confirmation by virus-specific assays. Of these, 94 (4·0%, 95% CI 3·2–4·9) were co-infected with HIV-2, and one woman had isolated HIV-2 infection, yielding an overall HIV-2 prevalence of 1·3% (95% CI 1·1–1·5). Thus, by the time of our recruitment period in late 1997 to early 2000, the prevalence of HIV-2 infection had declined substantially from the 7·6% reported in 1995, and uni-infection with HIV-2 was virtually nil. None of the baseline characteristics measured were significantly different among women infected with both viruses compared to women infected with HIV-1 only. Given these results, we discontinued strain-specific testing and subsequent baseline-positive women were confirmed at their next blood drawing using GeneScreen.

Univariate risk factor analyses

Month of recruitment

Baseline HIV prevalence increased significantly from November 1997 to the end of 1998, then decreased significantly until January 2000 (Fig. 2).

Age

Maternal median and mean ages were 23·4 and 24·6 years (95% CI 24·5–26·6), respectively. HIV prevalence increased with maternal age from 0% among 14-year-olds to >45% among women aged 29–31 years, and then fell rapidly with age to <20% among women aged >40 years (Fig. 3). Both the

linear and quadratic effects of age were significant (Table 2).

Parity

HIV prevalence had a nonlinear relationship to parity (Table 2), and the interaction term between age and parity was significant indicating that the effect of age on HIV prevalence differed for women of different parities. Thus, for parous-1 and parous-2 women, HIV prevalence increased linearly with age and was consistently ~10% higher among the parous-2 women across all ages (Fig. 4). For parous-3 women, prevalence and age were independent. For women with a parity of ⩾4, prevalence decreased at the high end of the age range.

Marital status

In univariate analyses, HIV prevalence tended to be higher among single women (this difference reached significance in multivariate analysis, see below) and was significantly higher among divorced and widowed women compared to married women (Table 2).

Religion

Prevalence was higher among women who stated that they had no religion or that they were Roman Catholic compared to other religions (Table 2).

Education and occupation

Prevalence was higher if either the mother or her husband had <8 years of school education and was lowest where either parent had clerical or professional jobs; it was highest when either worked as a vendor. Women whose husbands were vendors or unemployed were about twice as likely to be HIV-1 positive as those whose husbands held clerical positions; the odds ratios were 1·9 (95% CI 1·6–2·3) and 2·1 (95% CI 1·6–2·7), respectively.

Housing and income

HIV-1 prevalence tended to be lower among women who owned their home compared to those who did not. There was no significant variation in prevalence among women with family incomes ⩾US$18/month (data not shown). However, the 3% of women living in families with incomes below this level were significantly more likely to be HIV-infected (Table 2). Poverty levels increased during 1999: the proportion of women living in households earning
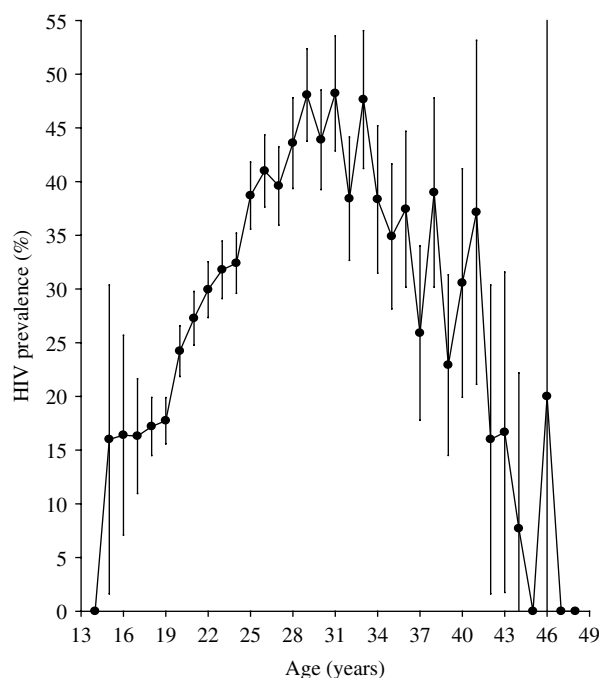
Fig. 3. The relationship between age and HIV-1 prevalence among new mothers in maternity clinics in Harare between November 1997 and January 2000. Error bars indicate the 95% confidence interval about the estimate.

<US$18/month doubled from 2·3% of the women recruited between November 1997 and February 1999, to 4·6% of the women recruited between March 1999 and January 2000, and the 25th percentile monthly income fell from US$54 to US$39/month between these two periods, respectively.

Recruitment site

Prevalence tended to be higher among women delivering at Harare Hospital, a tertiary referral hospital, compared to those delivering at municipal clinics of the Harare City Health Department (Table 2). Women recruited at peripheral sites outside the city limits exhibited the highest prevalence; 62% of these women were from Chitungwiza, which exhibited higher prevalence than Harare in other studies [17].

The only demographic variable collected that was not significantly associated with HIV prevalence in univariate analysis was travel time to the nearest health-care facility.

Multivariate analyses

In the final multivariate model, both linear and quadratic effects of recruitment month remained (Table 2), suggesting that prevalence significantly

Table 2. *Regression analyses of risk factors for prevalent HIV infection among postpartum women enrolled in ZVITAMBO trial*

Variable		*n*	Univariate	Multivariate
			Odds ratio (95% confidence interval)	
Recruitment month	Linear	14 057	1·41 (1·08–1·83)	1·56 (1·18–2·06)
	Quadratic		0·87 (0·78–0·97)	0·84 (0·75–0·94)
Age (years)	Linear	14 017	1·72 (1·62–1·82)	1·23 (1·12–1·36)
	Quadratic		0·991 (0·99–0·992)	0·99 (0·99–1·00)
Parity	Linear	14 057	2·58 (2·34–2·86)	3·11 (2·47–3·93)
	Quadratic		0·88 (0·87–0·90)	—
Age × parity		14 017		0·96 (0·96–0·97)
Marital status	Married	13 198	1	1
	Single	638	1·11 (0·94–1·32)	1·36 (1·13–1·63)
	Divorced	174	2·04 (1·51–2·75)	1·54 (1·13–2·11)
	Widowed	100	5·28 (3·42–8·15)	3·97 (2·53–6·23)
Mother's religion	Non-Catholic	10 643	1	1
	Catholic	2709	1·13 (1·04–1·24)	1·19 (1·09–1·31)
	None	705	1·32 (1·12–1·54)	1·31 (1·11–1·55)
Mother's education	Secondary	11 515	1	1
	<Secondary	2542	1·17 (1·07–1·28)	1·58 (0·99–2·53)
Age × mother's education		14 017	—	0·98 (0·97–1·00)
Father's education	Secondary	12 701	1	1
	<Secondary	1356	1·30 (1·16–1·46)	2·27 (1·25–4·13)
Age × father's education		14 017	—	0·97 (0·95–0·99)
Father's occupation	Professional	2306	1	1
	Other	11 751	1·29 (1·17–1·42)	1·22 (1·10–1·35)
Mother's housing	Own	1464	1	1
	Other	12 593	1·10 (0·98–1·23)	0·43 (0·24–0·77)
Age × mother's housing		14 017		1·04 (1·02–1·06)
Family income (US$/month)	⩾18	10 332	1	1
	<18	357	1·59 (1·29–1·98)	1·45 (1·16–1·82)
	Missing	3378	1·20 (1·10–1·30)	1·18 (1·08–1·29)
Recruitment site	Harare clinics	4101	1	1
	Harare hospital	7982	1·07 (0·99–1·17)	1·14 (1·04–1·24)
	Peripheral clinics	1974	1·15 (1·02–1·29)	1·21 (1·07–1·37)

increased, and then decreased, during the enrolment period. The linear and quadratic effects of age also remained in the final model. Interaction terms with age were not significant for paternal occupation, month and site of recruitment, income, and religion, but maternal age did significantly modify the effects of housing, maternal and paternal education, marital status, and parity. With inclusion of the interaction term between age and parity, the quadratic effect of parity was no longer significant. To facilitate interpretation of these interactions, multivariate analyses were applied separately to mothers who were older or younger than 23 years, the approximate median

maternal age. Thus, the odds of being HIV-infected increased with parity among women aged <23 years [adjusted OR (aOR) 1·58, 95% CI 1·42–1·77], but not for women aged ⩾23 years (aOR 1·01, 95% CI 0·97–1·06). In contrast, the odds of infection was more weakly associated with not owning one's home among women aged <23 years compared to older mothers (aOR 1·05, 95% CI 0·83–1·34, and aOR 1·33, 95% CI 1·14–1·54, respectively). Thus, high parity was associated with being HIV-infected among younger but not older women, while not owning one's home was more strongly associated with HIV infection among older compared to younger women.
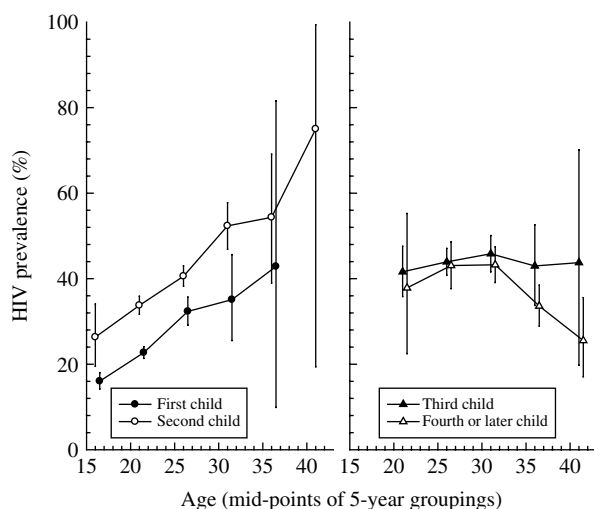
Fig. 4. The relationship between age and HIV-1 prevalence among new mothers in maternity clinics in Harare between November 1997 and January 2000; the effect of parity. Error bars indicate the 95% confidence interval about the estimate.

Similarly, the odds ratio for being HIV-infected among women who had <8 years of school education compared to those with more education was 1·12 (95% CI 0·95–1·33) and 0·96 (95% CI 0·83–1·10) among women aged <23 years and ⩾23 years, respectively. The same pattern was seen for the husband's education (OR 1·22, 95% CI 0·95–1·56, and OR 0·98, 95% CI 0·84–1·15), among women who were aged <23 years and ⩾23 years, respectively. Although these effects of education were not significant when the data were stratified on age, the interaction terms using continuous data were significant (Table 2). Marital status is presented in Table 2 as four categories to illustrate the differences in risks associated with the three unmarried categories (divorced, single, and widowed). The interaction term between marital status entered as a dichotomous variable (married/not married) and maternal age was significant (data not presented). To explore this further, multivariate analyses were applied separately to mothers who were older or younger than 23 years, and showed that the risk associated with being divorced, single, and widowed was lower, similar, and higher for younger compared to older women, respectively (data not presented). Extreme poverty remained strongly associated with HIV-1 prevalence in the multivariate analysis: the 3% of the sample of women living in families earning <US$18/month were nearly 40% more likely to be HIV-1-positive compared to women in higher income households.

However, as in the univariate analysis, HIV-1 prevalence did not vary with income among the great majority of women living in households with incomes higher than this cut-off.

DISCUSSION

Among women enrolled into the ZVITAMBO trial, HIV-1 prevalence significantly increased between November 1997 and December 1998, and then significantly declined by 18% from 35·9% to 29·5% between December 1998 and December 1999. Given the relatively short time period of this study (26 months), these data provide only preliminary evidence of declining HIV prevalence among antenatal women in Harare. However, these data are consistent with other recent reports from Harare and elsewhere in the country which together document that HIV-1 prevalence has declined dramatically in Zimbabwe since around 2000 [6, 18, 19].

HIV-2 prevalence was markedly lower in this study (1·3%) than the 7·6% estimated in 1996 [5]; the proportion of HIV-1-infected people who were co-infected with HIV-2 declined from 25% to 4% over the same period. HIV-2 may be at a replicative disadvantage during dual infection [20], such that HIV-2 viral load and transmissibility declines as the HIV-1 infection progresses. Since both viruses share the same general risk factors for transmission, the maturation of the HIV-1 epidemic in Zimbabwe may have decreased the success of the simultaneous HIV-2 epidemic, leading to lower numbers of both singly HIV-2-infected and dually infected people. The programmatic implication of the very low HIV-2 prevalence we observed is that diagnostic algorithms used for clinical or public health monitoring purposes in Zimbabwe do not need to incorporate strain-specific ELISA assays.

As well as apparently spanning the peak of the HIV-1 epidemic among pregnant women in Harare, the ZVITAMBO study was carried out against a backdrop of sharply declining family incomes during 1999. Booking fees for public-sector antenatal services increased by 43% in July 1998 and by a further 60% in July 1999. Lower income women may thus have opted to deliver outside the health-care system in disproportionately higher numbers than better-off women during 1999 compared to 1998. However, the worsening economy is unlikely to have biased the decline in prevalence observed in ZVITAMBO data during 1999 for two reasons. First, the effect of

recruitment month on prevalence remained highly significant after adjusting for income in the multivariate model. Second, low income was only associated with increased HIV-1 prevalence among women with the lowest 3% of incomes.

Risk factors associated with HIV prevalence

As in two previous studies of HIV prevalence among antenatal women in Harare [4, 5], we found that prevalence was higher among single compared to married women and among women who rented homes compared to those who owned them. However, the association between HIV prevalence and maternal age and parity differed across the three studies. In 1990, prevalence was highest among teenagers and parous-1 women [4]. In 1994–1995, prevalence was highest among women aged 20–25 years and increased with parity [5]. In ZVITAMBO, peak HIV prevalence occurred among women aged 29–33 years. Thus, the age group of peak prevalence was initially among the youngest women for whom incidence is highest, but became older as the epidemic matured. The fact that different age cohorts of women will have become sexually active and had children at different stages of the epidemic probably also explains the maternal age effect modification of parity on HIV prevalence. Among parous-1 and parous-2 women, HIV prevalence increased linearly with age as would be expected since age reflects duration of exposure to a disease of long duration (Fig. 4). The higher overall level in the parous-2 compared to parous-1 women may reflect a more extended period of sexual activity or a younger sexual debut in the former, both of which are associated with higher risk of HIV acquisition [21, 22]. The decline in prevalence after age 30 years among parous-4 women, may reflect that parous-4 women >30 years probably had their sexual debut earlier than parous-4 women <30 years, and probably had it about 10–20 years prior to ZVITAMBO enrolment – before the HIV epidemic in Harare began (around 1980) or became severe (around 1995). If, at that time, these women married, they will have been at reduced risk compared to younger parous-4 women since married women are at substantially lower risk of acquiring HIV compared to single women [9]. Moreover, a large proportion of women who *were* infected would have died [23] or been of reduced fertility [24–26] by the time of ZVITAMBO. By contrast, parous-4 women in their 20s, presumably began having sex at a younger age

and/or during the early to middle 1990s when HIV prevalence was high. Moreover, a smaller proportion of them would have died or become infertile compared with older women of the same parity. Parous-3 women lie between the extremes seen in women of lower and higher parity and, for them, HIV prevalence is independent of age.

What stands out in these analyses is that women who are HIV infected are more likely than HIV-negative women to be unmarried, have not completed secondary school education, rent or live with extended family rather than own their home, be unemployed, have a husband with fewer years of schooling and a non-professional occupation, and have an extremely low family income and no religious affiliation. In short they are less financially secure and have less psychosocial support.

We describe elsewhere the risk factors for HIV-1 incidence among the 9562 women in ZVITAMBO who were HIV-1 negative at baseline [9]. Although being unmarried and unemployed were both associated with increased HIV incidence, the other factors associated with *acquiring* HIV infection were quite different to those associated with *being* HIV infected. In contrast to the risk factors for prevalent HIV, no category of income, nor of women's or her husband's education, was associated with HIV incidence. Likewise, women who were professionals were at increased risk of HIV incidence, as were those whose husbands were in professional or clerical positions. Conversely, women whose husbands had professional occupations were the least likely to have prevalent HIV infection (Table 2).

These differences may be partly because the two groups of HIV-infected women became infected during different calendar times: most women HIV-positive at baseline acquired their infections in the early to mid-1990s, while incident cases acquired their infection during follow-up between 1998 and 2001. However, the differences may also reflect the impact of HIV infection on socio-economic status. Thus, while HIV strikes across a broad swathe of socio-economic strata, those infected are subsequently more likely to fall into extreme poverty and insecure employment, be less able to secure or maintain home ownership, more likely to divorce or become widowed, and less likely to complete formal education. This pattern has important implications for programmes providing antiretroviral therapy and other health care for HIV-infected people. HIV-positive people in Zimbabwe are likely to have lost

much of their social and financial support network during the course of their illness. If they are to access, comply with, and benefit from treatment, psycho-social and economic support must be fully integrated within care programmes.

APPENDIX. The ZVITAMBO Study Group

Members, in addition to the named authors are: Edmore Marinda, Florence Majo, Mary Ndhlovu, Ellen Piwoz, Lidia Propper, Philipa Rambanepasi, Naume Tavengwa, Michael Mbizvo, Andrea Ruff, Agnes Mahomva, Faith Mzengeza.

DECLARATION OF INTEREST

None.

REFERENCES

1. **Zaba B, Boerma T, White R.** Monitoring the AIDS epidemic using HIV prevalence data among young women attending antenatal clinics: prospects and problems. *AIDS* 2000; **14**: 1633–1645.
2. **WHO.** Evaluation of a national AIDS programme. A methods package.1. Prevention of HIV infection. WHO/GPA/TCO/SEF/94.1. 1994. WHO: Geneva.
3. **UNAIDS/WHO/MEASURE Evaluation/USAID.** National AIDS programmes: a guide for monitoring and evaluation. Draft, 1999. Geneva: UNAIDS/WHO.
4. **Mahomed K, et al.** Seroprevalence of HIV infection amongst antenatal women in greater Harare, Zimbabwe. *Central African Journal of Medicine* 1991; **37**: 322–325.
5. **Mbizvo MT, et al.** Trends in HIV-1 and HIV-2 prevalence and risk factors in pregnant women in Harare, Zimbabwe. *Central African Journal of Medicine* 1996; **42**: 14–21.
6. **Mahomva AI, et al.** HIV prevalence and trends from data in Zimbabwe, 1997–2004. *Sexually Transmitted Infection* 2006; **82** (Suppl. I): i42–i47.
7. **Malaba LC, et al.** Effect of postpartum maternal or neonatal vitamin A supplementation on infant mortality among infants born to HIV-negative mothers in Zimbabwe. *American Journal of Clinical Nutrition* 2005; **81**: 454–60.
8. **Humphrey JH, et al.** Effects of a single large dose of vitamin A, given during the postpartum period to HIV-positive women and their infants, on child HIV infection, HIV-free survival, and mortality. *Journal of Infectious Diseases* 2006; **193**: 860–871.
9. **Humphrey JH, et al.** HIV incidence among post-partum women in Zimbabwe: risk factors and the effect of vitamin A supplementation. *AIDS* 2006; **20**: 1437–1446.
10. **Iliff PJ, et al.** Early exclusive breastfeeding reduces the risk of postnatal HIV-1 transmission and increases HIV-free survival. *AIDS* 2005; **19**: 699–708.
11. **Reeves JD, Doms RW.** Human immunodeficiency virus type 2. *Journal of General Virology* 2002; **83**: 1253–1265.
12. **Rodes B, et al.** Viral response to antiretroviral therapy in a patient co infected with HIV Type 1 and Type 2. *Clinical Infectious Diseases* 2005; **41**: e19–e21.
13. **Graham WJ, Newell M.-L.** Seizing the opportunity: collaborative initiatives to reduce HIV and maternal mortality. *Lancet* 1999; **353**: 836–839.
14. **Temmerman M, et al.** Infection with HIV as a risk factor for adverse obstetrical outcome. *AIDS* 1990; **4**: 1087–1093.
15. **Ministry of Health and Child Welfare.** National survey of HIV and syphilis prevalence among women attending antenatal clinics in Zimbabwe, 2002, pp. 1–38, 2002.
16. **Gregson S, et al.** Study of bias in HIV-1 surveillance data in a high contraceptive prevalence population in sub-Saharan Africa. *AIDS* 2002; **16**: 643–652.
17. **Ministry of Health and Child Welfare.** *Zimbabwe National HIV and AIDS Estimates*. Zimbabwe: MOHCW, 2003.
18. **UNAIDS.** Evidence for HIV decline in Zimbabwe: a comprehensive review of the epidemiological data, pp. 1–45, 2006.
19. **Gregson S, et al.** HIV decline associated with behaviour change in Eastern Zimbabwe. *Science* 2006; **311**: 664–666.
20. **Sarr AD, et al.** HIV-1 and HIV-2 dual infection: lack of HIV-2 provirus correlates with low CD4+ lymphocyte counts. *AIDS* 1998; **12**: 131–137.
21. **Duncan ME, et al.** First coitus before menarche and risk of sexually transmitted disease. *Lancet* 1990; **335**: 338–340.
22. **Pettifor AE, et al.** Early age of first sex: a risk factor for HIV infection among women in Zimbabwe. *AIDS* 2004; **18**: 1435–1442.
23. **Casella JF.** Time from HIV-1 seroconversion to AIDS and death before widespread use of highly-active

anti-retroviral therapy. A collaborative analysis. *Lancet* 2005; **335**: 1131–1137.

24. **Gregson S, Zaba B, Garnett GP.** Low fertility in women with HIV and the impact of the epidemic on orphanhood and early childhood mortality in sub-saharan Africa. *AIDS* 1999; **13**: S249–S257.

25. **Gray RH, *et al.*** Population-based study of fertility in women with HIV-1 infection in Uganda. *Lancet* 1998; **351**: 98–103.

26. **Terceira N, *et al.*** The contribution of HIV to fertility decline in rural Zimbabwe, 1980–2000. *Population Studies* 2003; **57**: 149–164.