Monitoring the AIDS epidemic using HIV prevalence data among young women attending antenatal clinics: prospects and problems

Basia Zaba[a], Ties Boerma[b] and Richard White[a]

Objective: To assess the potential of antenatal surveillance data on HIV prevalence in young women as an indicator of trends in HIV incidence.

Design: Review of empirical data and discussion of problems encountered with surveillance systems, illustrated using cohort-component projection models.

Methods: Simple descriptive analyses are presented of prevalence and incidence data, with projection models used to explore aspects of the dynamic relationships between changes in HIV incidence and prevalence in young pregnant women for which empirical data are not yet available. Incidence changes due to change in risk among sexually active, and change in pattern of sexual debut are explored separately, and the resulting prevalence trends in pregnant women under age 25 years, and those expecting their first two births are described.

Results: HIV prevalence levels in young pregnant women categorized by age and by parity have different relationships to recent incidence levels. Age categorized prevalence data provide a reasonable indication of incidence under stable conditions, but may be very misleading if the age pattern of sexual debut changes. Prevalence levels categorized by parity are a reliable guide to incidence in the sexually active, but not necessarily to incidence in the population as a whole.

Conclusions: Ante-natal surveillance systems should categorize prevalence data by both age and parity to aid in the interpretation of underlying incidence levels.

AIDS 2000, **14**:1633–1645

Keywords: HIV prevalence, HIV incidence, sentinel surveillance, population-based surveys, measurement bias

Introduction

Reliable data on HIV incidence are essential for monitoring the spread of HIV and for impact assessment of sexual health interventions. Unfortunately, incidence data are difficult and costly to collect and therefore limited to localized cohort studies. At national and sub-national levels HIV prevalence data will continue to be the main source for monitoring the epidemic and assessing the impact of interventions to reduce transmission. HIV incidence has been estimated from prevalence data collected in a single survey or in multiple survey rounds by making assumptions about constant incidence rates, steady-state conditions, and mortality rates among diseased subjects [1–4].

Prevalence data may be collected in population-based surveys or by monitoring sentinel populations. Women

From the [a]Centre for Population Studies, London School of Hygiene & Tropical Medicine, London, UK and the [b]Carolina Population Centre, School of Public Health, University of North Carolina at Chapel Hill, Chapel Hill North Carolina, USA.

Sponsorship: The study was partly supported by the UK Department for International Development (Knowledge Programme on Reproductive Health) (B.Z.) and the USAID-sponsored MEASURE evaluation project, USAID Cooperative Agreement ®HRN-A-00-97-00018-00 (T.B. and R.W.).

Correspondence to Basia Zaba, Centre for Population Studies, London School of Hygiene & Tropical Medicine, 49-51 Bedford Square, London WC1B 3DP, UK.

Fax 0207-299-4637; e-mail basia.zaba@lshtm.ac.uk

Received: 18 January 2000; revised: 21 March 2000; accepted: 30 March 2000.

attending antenatal clinics are the predominant source of HIV prevalence data in countries with generalised epidemics, especially in sub-Saharan Africa, where many countries have time series of such data [5]. The extent to which HIV trends among antenatal women reflect trends in the general population depends on the severity of a number of biases. If antenatal attendance is high, as in most countries of sub-Saharan Africa, women attending antenatal clinics are likely to be typical of all pregnant women. However, pregnant women may not be representative of the general female population of reproductive ages – partly because of lower fertility in HIV-infected women and partly because of selection for sexual activity [6]. Comparisons of HIV prevalence between antenatal clinic and population-based samples in six African studies showed antenatal women had lower HIV prevalence than the general female population in all age groups except the youngest (under 20 years).

HIV incidence and prevalence trends among young women are of particular relevance for monitoring the AIDS epidemic. First, the dynamics of HIV infection in young women exert a large influence on the overall course of the epidemic, because young women form a high proportion of the total population. Second, incidence rates among young women are often among the highest observed [7,8]. Third, young women may respond more rapidly to interventions with changes in behaviour – for example declines in HIV prevalence among young antenatal women in Uganda have been attributed to behavioural change [9,10]. Finally, the effects of HIV-associated reduction in fertility or increases in mortality are likely to be less pronounced among the recently infected, making estimation issues less problematic. Fewer assumptions are needed to estimate incidence from prevalence and hence the incidence estimates based on younger individuals should be more robust [11].

For these reasons, HIV prevalence among 15–24-year-old women attending antenatal clinics has been selected as a key indicator for monitoring HIV prevention programmes [12,13], with some authors suggesting that surveillance should focus on an even narrower age group, such as 15–19 or 18–21 year olds [10,14]. However, methodologies for converting these antenatal prevalence measures to general population incidence estimates have yet to be developed. A recent development that may facilitate the future estimation of HIV incidence in large samples of pregnant women is the use of a varying sensitivity test on a single sample [15]. At present, however, there is no evidence of the validity of this approach in developing countries.

This article investigates whether and how data on HIV prevalence among young women from antenatal clinics could be used to estimate trends in HIV prevalence and incidence in the general female population. In particular, the advantages and limitations of age-based measures (15–19 and 15–24 years), are assessed. Biases may occur related to age reporting and initiation of sexual activity. Parity-specific HIV prevalence monitoring avoids some of these biases and is found to be most appropriate for assessing prevalence trends among the sexually active. Models are used to assess the robustness of different measures, particularly in rapidly evolving epidemics.

HIV prevalence and incidence by age

Table 1 summarizes data on female HIV prevalence and incidence from selected studies in sub-Saharan Africa. The first panel shows antenatal prevalence data broken down by age group, the second panel shows similar data from population-based studies. Prevalence among young women varies considerably, with larger variation at 15–19 than at 20–24 years. In several antenatal studies (but not in community studies) HIV prevalence exceeds 20% among women aged 15–19 years. The difference in HIV prevalence between women aged 15–19 and 20–24 years is much larger in population-based surveys than in the antenatal data, because at 15–19 years pregnant women are less representative of their age group than at 20–24, as the 15–19 years age group inevitably contains a large proportion of virgins. The median HIV prevalence ratio 15–19/20–24 is 0.71 in the 17 antenatal studies (range, 0.35–1.02), compared with 0.34 in the 10 population-based surveys (range, 0.13–0.83).

In both sets of studies there is a strong linear relationship between HIV prevalence at 15–24 years and prevalence in the reproductive ages as a whole (15-49 years in most studies), as shown in Figure 1a. Regression lines indicate that correlation is stronger in antenatal studies than in population-based studies ($r^2 = 0.96$ and 0.88 respectively), and that the relationship is closer to simple proportionality in antenatal studies (regression intercepts = 0.0 and 1.1 percentage points respectively). This may be due to transient incompatibility of experience in younger and older women in the cross-sectional measures: prevalence levels at ages 15–24 years reflect only recent incidence rates, prevalence in the age range 15–49 years is the result of exposure to historical and recent incidence. The population-based data in Figure 1 are derived from communities with a mixture of old and new, rapidly growing and almost stable epidemics, so differences between age group experience will be quite variable. By contrast, antenatal data generally represent prevalence due to recent infections, since at longer durations of infection women become less fertile and thus less likely to present at antenatal clinics [28].

Table 1. Female HIV prevalence and incidence by age and data source.

		Age group											
		15–19		20–24		15–24		25–49		All ages 15–49			
HIV Prevalence Data source	Years	(%)	n	(%)	n	(%)	n	(%)	n	(%)	n	Exceptions	Source
Antenatal clinic													
Bujumbura, Burundi	1991–1992	12.5	287	19.0	499	16.6	786	17.2	763	16.9	1549		[16]
Nairobi, Kenya	1991–1993	14.4	653	14.1	1466	14.2	2119	13.1	1043	13.8	3162		[14]
Nairobi, Kenya	1994–1997	13.5	821	15.9	1619	15.1	2440	16.2	1226	15.5	3666		[14]
Blantyre, Malawi	1990	22.0	1377	27.3	2111	25.2	3488	20.3	3196	22.9	6684		[8]
Blantyre, Malawi	1993	24.7	543	33.3	979	30.2	1522	30.1	942	30.2	2464		[8]
Blantyre, Malawi	1994–1995	24.1	1691	37.0	2460	31.7	4151	27.9	2770	30.2	6921		[8]
Mangochi, Malawi	1987–1990	7.5	1352	9.6	1092	8.4	2444	8.6	1509	8.5	3953		[17]
Malawi, rural areas	1997	14.7	1301	23.9	2111	20.4	3412	21.2	2246	20.7	5658		[18]
Butare, Rwanda	1989–1991	11.2	276	13.9	1296	13.4	1572	7.9	4093	9.3	5690		[19]
Rwanda, rural areas	1996	11.4	140	16.0	494	15.0	634	15.2	734	15.1	1368	15–44	[18]
South Africa	1996	12.7	2151	18.0	3491	16.0	5642	13.7	5916	14.8	11558		[20]
South Africa	1997	12.8	2107	19.7	3590	17.1	5697	15.2	6058	16.1	11755		[20]
Fort Portal, Uganda	1991–1993	23.3	400	28.0	371	25.6	771	17.7	526	22.4	1297		[10]
Fort Portal, Uganda	1994–1997	13.8	645	25.1	638	19.4	1283	19.2	691	19.4	1974		[10]
Kinshasa, Zaire	1989	2.8	722	8.1	1356	6.3	2078	6.7	2545	6.5	4623		[1]
Kapiriri, Zambia	1994	9.0	1446	15.2	2012	12.6	3458	13.4	2600	12.9	6058	15–44	[21]
Lusaka, Zambia	1994	20.4	1147	32.2	1867	27.7	3014	28.7	2237	28.2	5251	15–44	[21]
Population-based													
Addis, Ethiopia	1994	3.5	510	9.1	326	5.7	836	8.4	812	7.1	1648		[11]
Rwanda, rural areas	1997	8.3	289	10.0	210	9.0	499	13.2	711	11.5	1210		[18]
Kisesa, Tanzania	1994–1995	1.0	692	7.7	663	4.3	1355	8.6	1730	6.7	2041	15–44	[22]
Kisesa, Tanzania	1996–1997	2.5	651	7.7	793	5.4	1444	9.7	2041	7.9	3485	15–44	[22]
Mara, Tanzania	1989–1990	5.1	475	12.6	548	9.1	1023	6.6	1038	7.9	2061		[23]
Masaka, Uganda	1989	4.5	601	21.3	282	9.9	883	11.5	723	10.6	1606	13–44	[24]
Masaka, Uganda	1994	2.5	531	19.4	211	7.3	742	12.8	611	9.8	1353	13–44	[24]
Fort Portal, Uganda	1995	12.3	122	30.3	99	20.4	221	14.1	306	18.4	527		[10]
Kapiri, Zambia	1995–1996	8.2	122	24.6	114	16.1	236	18.9	190	17.4	426	15–39	[21]
Lusaka, Zambia	1995–1996	12.3	391	35.4	311	22.5	702	40.1	509	29.9	1211	15–39	[21]
HIV Incidence		(%)	(PYO)	(%)	(PYO)	(%)	(PYO)	(%)	(PYO)	(%)	(PYO)		
Population-based													
Blantyre, Malawi	1994–1995	6.0	502	4.6	699	5.2	1201	3.2	1101	4.2	2302		[8]
Kigali, Rwanda	1988–1992	4.3	93	3.4	262	3.6	355	3.2	222	3.5	577	ANC	[25]
Dar es Salaam, Tanzania	1992–1995	6.5	169	3.8	737	4.3	906	2.7	1330	3.4	2236	FP	[26]
Kisesa, Tanzania	1994–1996	0.5	754	1.4	816	1.0	1570	0.8	2510	0.8	4068		[22]
Masaka, Uganda	1990–1994	0.7	1849	1.5	658	0.9	2507	0.8	2197	0.8	4704	15–44	[27]
Rakai, Uganda	1994	5.0	81	6.8	73	5.9	154	2.0	149	4.0	303	15–39	[7]

PYO, person–years of observation; ANC, antenatal clinic; FP, family planning.

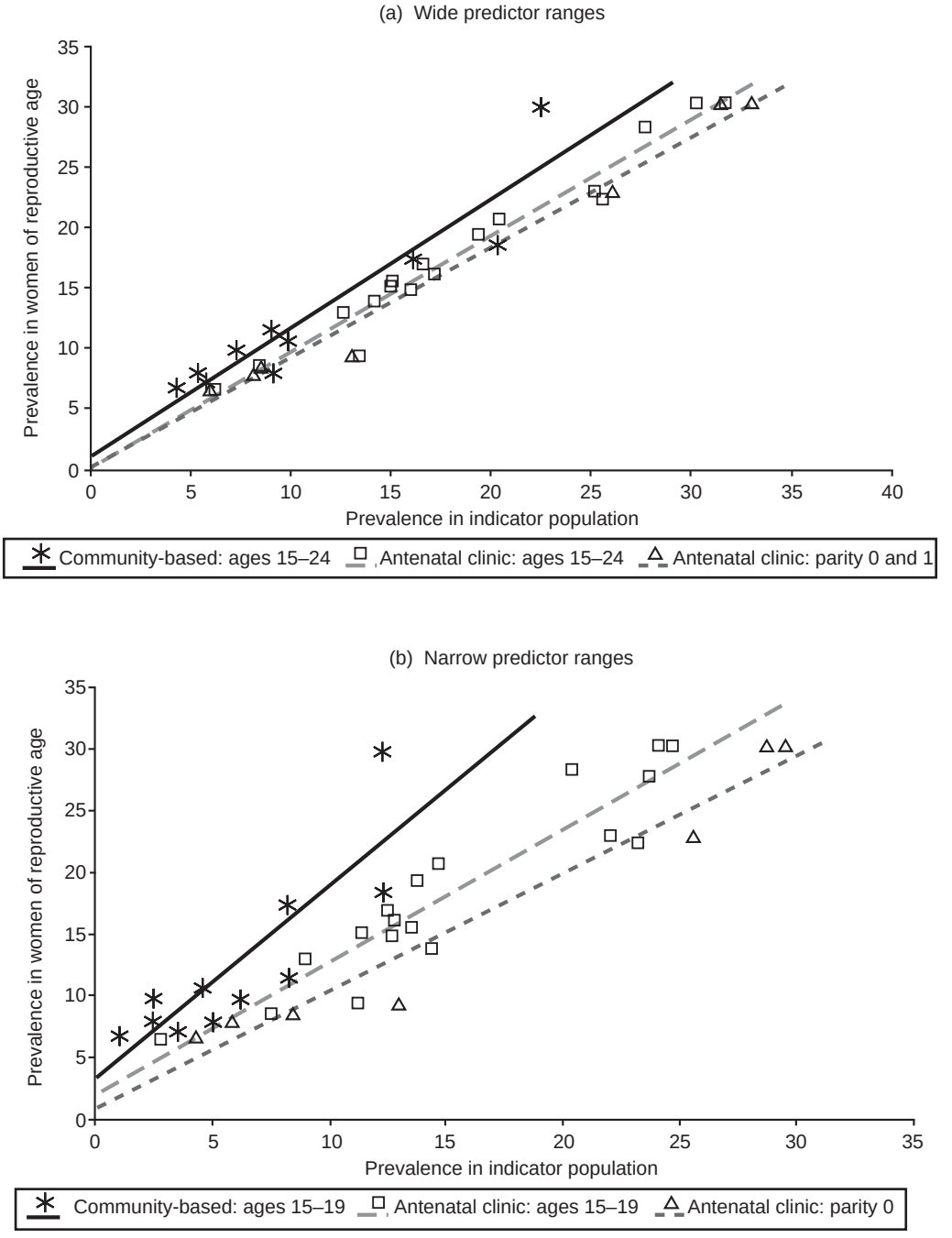

Fig. 1. Prevalence in young women as a predictor for all age prevalence.

Figure 1b shows that prevalence in the 15–19 years age group is not as strongly correlated to all age prevalence ($r^2 = 0.86$ and 0.75 in antenatal and population-based studies respectively) than the corresponding relationships in the 15–24 years age group; furthermore, the relationships are not as close to simple proportionality, with larger regression intercepts (2.0 and 3.4 respectively).

The last panel of Table 1 shows incidence data from the few available longitudinal community-based studies, classified by age as in the first two panels. It is clear that incidence at 15–24 years is appreciably higher than in the population as a whole, but the relationship to all age incidences is reasonably proportionate, with a correlation coefficient close to one ($r^2 = 0.94$) and a small regression intercept (0.3). This relationship is stronger than the corresponding relationship in the 15–19 years age group ($r^2 = 0.88$, intercept 0.7). Direct comparisons between prevalence and incidence meas-

ures for the same population would tell us much about the predictive power of prevalence data from young women as a measure of incidence, but published data are not available – only Blantyre in Malawi has published both antenatal clinic prevalence measures as well as data on incidence.

More than half of all women in the childbearing ages are in the age group 15–24 years (Table 1); the median proportion is 57% (range, 28–67%) in the 17 antenatal-care studies and 53% (range, 41–66%) in the 10 population-based surveys. However considerable variation exists in the proportion of women aged 15–19 years relative to 20–24 years. At one extreme, in Rwanda only 22% of antenatal women in the 15–24 years age-group were aged under 20 years, whereas more than half were under 20 years in Fort Portal, Uganda. Age at first pregnancy is the major determinant of this proportion. As HIV prevalence increases rapidly in the early phase of sexual activity, fluctuations in the age composition within these young age groups will affect the overall prevalence estimate: the younger the sample, the lower the prevalence in the age group. No studies provide data by single years of age, and this does not appear to be a feasible option because of the prohibitive costs of the large samples needed. Hitherto, most surveillance clinics aimed to have samples of about 300–500 women per year in all age groups. World Health Organization [12] recommended a sample size of 3000 women in the age group 15–24 years to monitor HIV trends.

Age mis-statement is common in countries where respondents do not know their exact birth date, with more serious effects if smaller age groups are used. Heaping on age 20 years can be considerable and could have an effect on prevalence monitoring, especially if the 15–19 and 20–24 years age groups are separated. Young pregnant women may report an older age at antenatal clinics, especially if there is popular concern about early and premarital pregnancies because of campaigns aimed at reducing adolescent pregnancy.

The choice of a lower cut-off point for the age range is critical – most antenatal clinics and population-based surveys report data from age 15 years, but in some countries data are reported separately for girls aged under 15 years. In 1977 in South Africa HIV prevalence was 9.5% among 42 pregnant girls aged under 15 years, who constituted only 0.7% of all antenatal women under 25 years [20]. In Malawi in 1977, no pregnant girl under 15 years of age was HIV positive and girls under 15 years accounted for 0.5% of all antenatal women under 25 years of age [29]. Population-based studies including girls aged under 15 years typically found prevalence rates of less than 1% (e.g. Rakai, Uganda [30], and Addis Ababa, Ethiopia [11]), except for Rwanda in 1997, where a 4% prevalence was reported in the 12–14 years age group [18]. Inclusion of girls under 15 years would have a major depressing influence on prevalence in the age group described as 'under 20 years', as most are virgins and because younger girls are more numerous than older ones. Therefore, exclusion of women under 15 years in monitoring HIV prevalence is justified for most populations.

HIV prevalence by parity

Most antenatal clinics routinely collect data on the rank order of the pregnancy and occasionally report such data in HIV sentinel surveillance. Pregnancies can be classified by a woman's parity (number of previous live births) or gravidity (number of previous pregnancies), so it should be possible to obtain HIV prevalence categorized by parity or gravidity. Table 2 shows that women at their first and second pregnancy usually account for slightly less than half of all pregnant women, and that HIV prevalence typically peaks at the second pregnancy. Figure 1a indicates that the proportionate relationship between prevalence in all pregnant women and women experiencing their first or second pregnancy ($r^2 = 0.98$, intercept $= 0.1$) is as strong as the corresponding relationship for prevalence measures based on women aged 15–24 years ($r^2 = 0.96$, intercept $= 0.0$). Figure 1b shows the proportionate relationship between prevalence in women experiencing their first pregnancy and all pregnant women is much stronger ($r^2 = 0.95$, intercept $= 0.9$) than that based on women in the 15–19 years age group ($r^2 = 0.86$, intercept $= 2.0$).

Ideally, pregnancy histories should include previous abortions and stillbirths. However, past abortions are often underreported and since women tend to make their visit to antenatal clinics late in pregnancy (usually in the second trimester) pregnancies that lead to abortions are completely missed. In non-contracepting populations, the parity of a young pregnant woman could be a better measure of sexual exposure than her age, especially if there is wide variation in age at first sex, or if initial sexual contacts are sporadic and infrequent. As a measure of length of exposure to unprotected sex, gravidity would be more precise, as this accounts for fetal losses. However, in view of the reporting problems inherent in classifying women by gravidity, data classified by parity would be more robust.

Population-based sero-surveys may also classify women by parity, although the results are not strictly comparable to antenatal clinic data, since antenatal women are closer to progressing to the next parity than women in a community survey. Also, population-based data in-

Table 2. HIV prevalence and incidence among women by pregnancy or birth order and data source.

HIV prevalence		By gravidity										
		G1		G2–3		G4–5		G6+	All women		Grouping	
Data source	Years	(%)	n	(%)	n	(%)	n	(%)	(%)	n	exceptions	Source
Antenatal clinic												
Blantyre, Malawi	1990	25.6	1560	26.4	2177	22.9	1505	14.4	22.9	6689		[8]
Blantyre, Malawi	1993	28.7	663	33.4	963	30.8	794	21.7	30.2	2775		[8]
Blantyre, Malawi	1994–1995	29.5	2449	37.1	2083	31.6	1125	19.1	30.2	6933	G2, G3+	[8]
Mangochi, Malawi	1987–1990	8.4	1246	8.7	951	5.4	1746		8.5	3943	G4-6, G7+	[17]
Butare, Rwanda	1989–1991	13.0	1351	13.1	1794	5.6	1632	6.2	9.3	5690		[19]
		By parity										
		P0		P1		P2-3		P4+	All women			
		(%)	n	(%)	n	(%)	n	(%)	(%)	n		
Demographic surveillance												
Kisesa, Tanzania	1994	4.3	448	7.1	675	9.2	675	6.7	6.7	2999		[22]
Kisesa, Tanzania	1996	5.8	676	11.1	540	9.6	868	6.6	7.9	2228		[22]

clude women who never progress to the next parity, either because of partnership circumstances or secondary sterility – these women are completely missed in antenatal HIV surveillance.

Modelling time trends in incidence and prevalence

A simple model was constructed to study expected relationships between prevalence of sexually transmitted diseases and incidence measures in young women when incidence changes over time [31]. It predicts prevalence in all women 15–24 years, as well as in pregnant women, and allows us to look at the effects of classifying pregnant women either by age or by parity. Equations formally defining the model are given in the Appendix, and a brief, non-technical explanation of its main features follows.

The simulated population represents females aged 10 to 29 years followed for 25 years from the baseline year, distinguishing single year age groups and time periods. In the following scenarios, the population is assumed to arise from birth cohorts growing at 2% per year, and adult mortality from non-HIV causes is assumed to be 0.5% per year at all ages. Under these initial conditions, successive 5-year age groups differ by about 13%, a structure that is typical of African populations before the advent of HIV. At baseline, HIV prevalence is assumed to be zero, non-zero incidence among the sexually active is assumed to start in the first projection year. HIV incidence among the sexually active is assumed independent of age, but is allowed to vary over time. Incidence is measured as an annual risk for those currently HIV negative, prevalence is a cross-sectional measure with HIV positive and negative in the denominator.

The cumulated risk function describing the age pattern of start of sexual activity is assumed to be a logistic, whose youthfulness can be varied. The default pattern, assumed to operate up to and including the baseline year, assumes a median age at first sex of 15 years. The age pattern of sexual debut is allowed to change over time, but once a woman has become sexually active she remains so until we lose sight of her when she reaches the age of 30 years.

The sexually active are assumed to have a fixed annual risk of giving birth which is independent of age – in the simulations described below this risk is 30%, a reasonable approximation for fecund women under 30 years. The parity distribution of a cohort is therefore determined only by the distribution of the women by time since sexual debut. HIV-infected women are assumed to have the same fertility as uninfected sexually

active women. Median survival time after HIV infection is assumed to be 10 years.

The main input assumption driving the model is the annual rate of HIV incidence among the sexually active, but the pattern of sexual debut, which determines the proportion sexually active by age is also an important determinant of incidence and prevalence. If sexual debut is early, incidence among all women aged 15–24 years is virtually the same as incidence among the sexually active; if it is late, incidence in the general population aged 15–24 years is lower than in the sexually active as there are significant proportions of virgins in this age group.

Various measures of prevalence were computed for this simulated population, as shown in Figure 2. Prevalence measures in pregnant women classified by age or by parity are equivalent to antenatal surveillance measures similarly classified. Parity-based measures extend across the whole of the simulated age range (10 to 29 years), and are not confined to the 15–24 years age group in which population-based incidence and prevalence are measured.

This projection model is used to assess how well trends in HIV prevalence among young antenatal women reflect HIV incidence trends if changes occur in the age at sexual debut or in the risk of HIV transmission. HIV incidence among the sexually active is assumed to rise linearly from year zero, flattening out at 5% per annum in year 5. In scenario 1 (Fig. 2a) it falls linearly from year 15, reaching a level of 2% per annum in year 18. (Incidence in the sexually active population and incidence in the 15–24 years age group coincide so closely in this simulation, that the trend lines appear superimposed throughout.) This pattern of change in incidence among the sexually active could represent the result of behaviour change, but may also be observed in epidemics which reach saturation in high-risk groups.

In scenario 2 (Fig. 2b) incidence among the sexually active remains constant at 5% per annum for the rest of the projection period, but the pattern of sexual debut is assumed to age rapidly between calendar years 10 and 13, so that after year 13, the median age at first sex has risen from 15 to 20 years. The last scenario (Fig. 2c) shows the effect of combining a fall in incidence among the sexually active (as in the first scenario) with a rise in age at first sex (as in the second scenario).

In scenario 1, trends in the population prevalence measure for ages 15–24 years are echoed closely by trends in prevalence among pregnant women aged 15–24 years, and by trends for those experiencing first and second births. Trends for 15–19-year-olds follow a broadly similar pattern, but at a lower overall level. All the measures respond immediately to the decline in incidence rates among the sexually active which starts in year 15. However, there are lags in the responses to the levelling off in incidence, both after the period in which incidence was rising (up to year 5) and after the period of decline (years 15 to 18).

The reason for these lags is that it takes some time for the relevant age or parity groups to fill up with women who have been subject to the new level of risk for the whole of the time that they typically spend in that group. Lags are longer for the 'by parity' grouping of pregnant women – at a constant annual risk of birth of 30% for the sexually active, about half of first births occur within 2 years of the start of sexual activity, but about 10% of second births occur to women who have been sexually active for more than 7 years.

The reason for the more-or-less instant response of all prevalence measures to the drop in risk at year 15, is that the simulation represents a growing population, so the youngest single-year cohort (with lowest HIV prevalence), which enters observation after the fall in risk is numerically larger than the oldest cohort with the highest prevalence which leaves observation. This relationship is tighter for age-based than parity-based measures.

Figure 2b illustrates the second scenario, in which the decline in incidence is due to a rise in the age at sexual debut. The age range in which 98% of the population are assumed to lose their virginity moves from ages 12–17 to 16–24 years, eventually producing a fall in incidence in the age group 15–24 years from 5 to 2%, even though the risk to the sexually active does not change. Although the change in age pattern of sexual debut takes only 3 years to accomplish (as with the fall in HIV risk in the Figure 2a) it takes about 7 years for incidence to fall from 5 to 2%, as this is how long it takes the women who had experienced the earlier, faster rates of sexual debut to leave the 15–24 years age group and be entirely replaced by women who had experienced only the later, slower sexual debut rates. Community-based prevalence responds immediately to the beginning of the incidence fall, and levels out about 5 years after incidence stabilizes.

The parity-based measure stays at its peak value, because it reflects the steady risk experienced by the sexually active – only the sexually active become pregnant, and the distribution of the women experiencing a first birth or second birth by time since first sex does not change, although their age composition may change as sexual debut patterns change. Since prevalence among the sexually active is determined by length of exposure to sexual activity and by transmission risk level, the prevalence by parity curves for pregnant women stay constant.

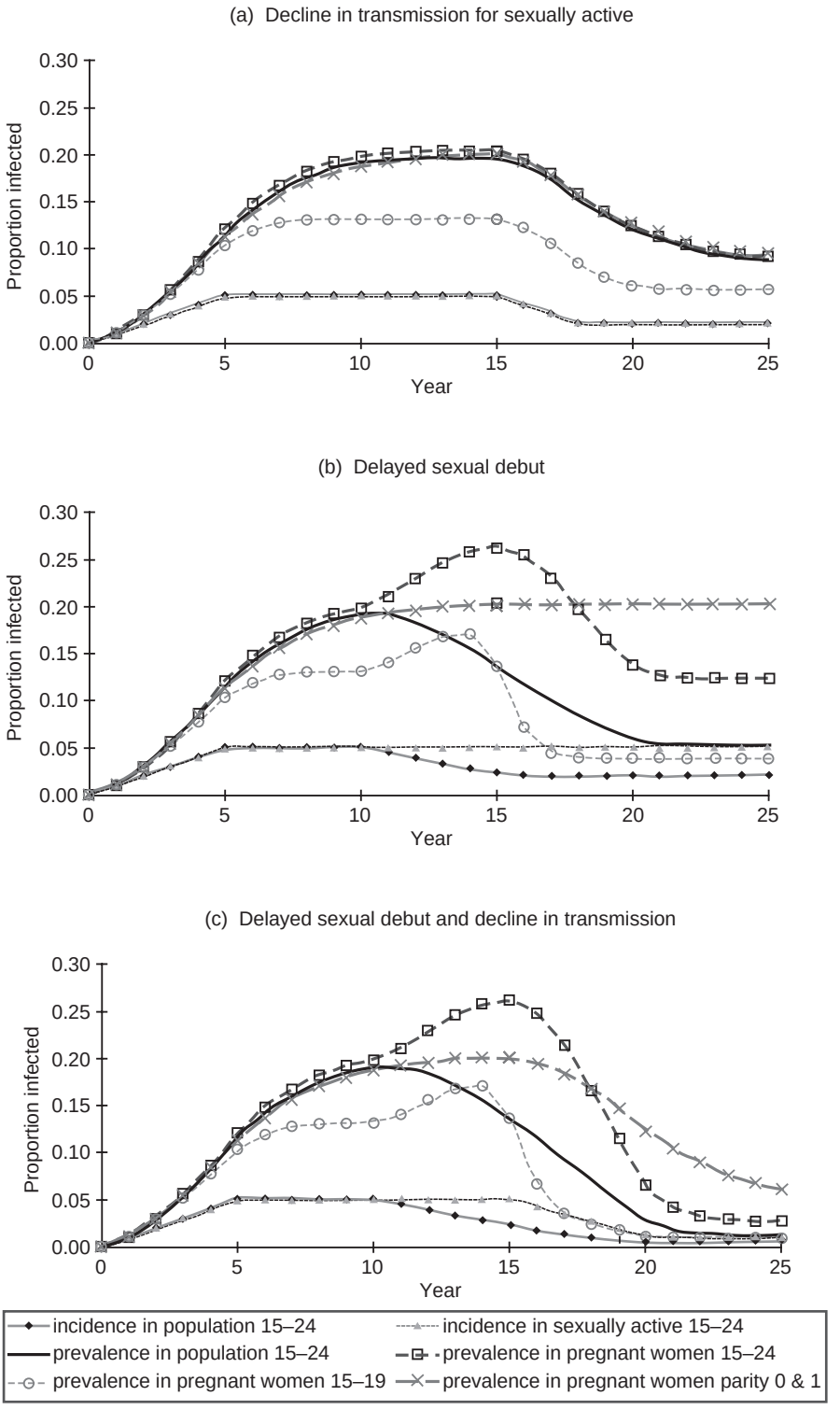

Fig. 2. Prevalence and incidence trends in young women.

Prevalence measures in pregnant women aged 15–24 years actually rise for 4 to 5 years in response to an increase in age at first sex. This counter-intuitive result reflects the fact that in this selected group of pregnant women, the proportion who have been sexually active for a relatively long time initially increases, as incoming cohorts are numerically depleted by lower rates of entry into sexual activity. After some years, those who experienced the earlier pattern of sexual debut cease to dominate the age group, and the distribution of

pregnant women by duration of sexual activity begins to reflect the new pattern of age at first sex. HIV prevalence in pregnant women aged 15–24 years then falls very rapidly, even though incidence in the same age group is levelling off at this stage. The pattern of prevalence change among 15–19-year-old pregnant women is broadly similar to that in 15 to 24-year-old women, but with earlier and steeper rises and falls.

Figure 2c illustrates the situation where both changes occur (sexual debut moves to later ages and the risk for the sexually active is reduced). Overall, prevalence trends in the parity-based data give a truer representation of incidence trends than the age-based data. Changes in prevalence among pregnant women classified by parity consistently lag behind population-based incidence changes in the 15–24 years age group. Trends in the age-based pregnant prevalence measures have an erratic relationship to population-based incidence trends, just as in the previous simulation.

However, the relationship between the peak level of population-based incidence and the corresponding final stable incidence level (5 : 1%) is fairly accurately reflected in the approximate relationship between the peak and final values of the age-based measures for pregnant women (12 : 3% in 15–19-year-olds; 20 : 5% in 15–24-year-olds). In contrast, the peak : final ratio in the parity-based measure (20 : 10%) is too low, although it does reflect the ratio for incidence in the sexually active (5 : 2%).

Figure 3 illustrates the relationship between population-based incidence and prevalence measures in pregnant women incorporating a 3-year time lag, and aggregating data from a wide range of projection scenarios, with different rates of incidence increase and decline, and different lengths of time spent in the peak incidence phase. Clearly, the relationship between the parity-based prevalence measure and incidence in the sexually active (Figure 3b) is tighter than that between the age-based prevalence measure and incidence in the general population (Figure 3a).

These illustrations show that lagged, parity-based measures of prevalence in pregnant women are consistent indicators of incidence in the sexually active population. Short-term trends in prevalence in young pregnant women classified by age can be misleading as indicators of current incidence trends, but if prevalence has been stable for some time, this is a reasonable indicator of stable incidence in the population as a whole. About 5 years of unchanging prevalence in the 15–24 years age group of pregnant women can be taken as evidence of stability, since in this time the 15–19 years age group, whose composition is most likely to be affected by changes in age at sexual debut would experience a complete turnover in membership.

Discussion

Our examination of the empirical data showed that HIV prevalence in antenatal women aged 15–24 years and prevalence in those expecting first and second births have similar proportionate relationships to all age antenatal prevalence, and that these relationships are more robust than corresponding relationships for 15–19-year-olds, or for primigravida, or for community-based measures. More antenatal prevalence data classified by parity are likely to become available in the near future.

The simulations have shown that for antenatal clinic-based HIV prevalence estimates to be useful as indicators of incidence, the age group used should be wide (15–24 years rather than 15–19 years) and that it is useful to collect parity-based information to complement the age-based data as the two data types are subject to slightly different biases. Parity-based indicators are better at reflecting the dynamics of infection in the sexually active population, but age-based measures may be better at portraying stable incidence levels in young women as a whole. Parity-based indicators are therefore particularly useful in the context of 'second generation surveillance' [13] which emphasizes the need to collect data on sexual behaviour to complement data on infections.

Prevalence measures tend to lag behind changes in incidence, but age-based measures in pregnant women may be subject to rapid fluctuations if there are significant changes in age at sexual debut. These fluctuations may give the impression that incidence is rising when it is in fact already falling, or that a rapid decline is occurring when the pace of the incidence decline is quite moderate. Age-classified antenatal prevalence data may exaggerate the height of the peak incidence and prevalence levels in the community, and the pace at which change takes place. However, such biases are transitory, and the stable prevalence level eventually attained in pregnant women aged 15–24 years should be a reasonable guide to the stable incidence level in this age group in the general population. Comparisons with trends in prevalence among women expecting their first and second births could identify spurious transient trends in age-based measures, as parity-based measures should be less affected by changes in age at first sex.

In the simulations we are able to separate the effects of changes in age at sexual debut from changes in risk amongst the sexually active. In real populations behavioural changes of this type may occur together. Furthermore, certain types of behaviour change, such as increased condom use, are likely to alter the level of fertility – this could introduce further non-linearities into the relationship between HIV inci-

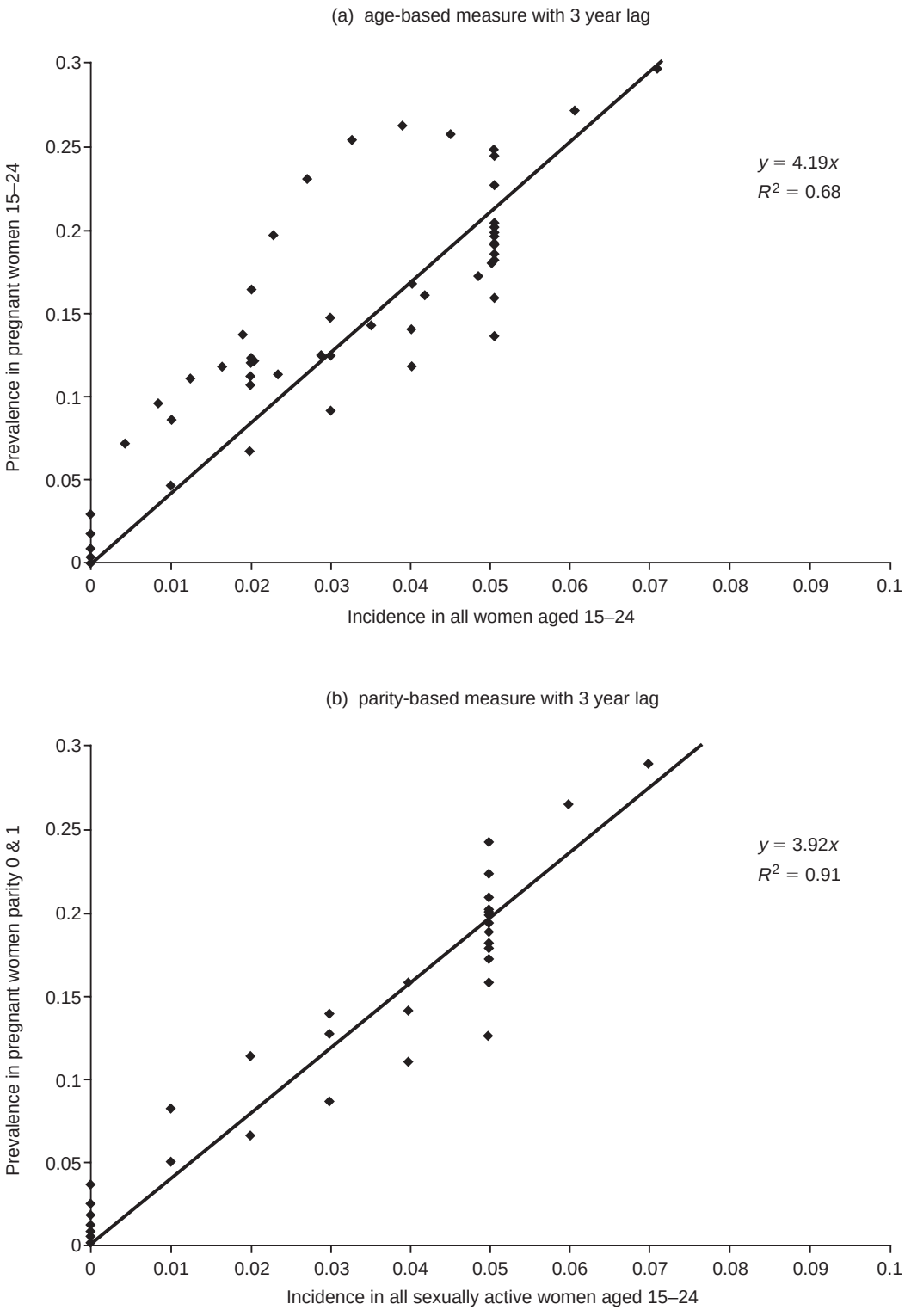

Fig. 3. Correlation of HIV incidence and prevalence in young pregnant women.

dence and prevalence in pregnant women. Other studies [32] showed that for stable epidemics, prevalence measures based on pregnant women aged 15–24 years or parity 0 and 1 continue to provide reasonable indicators of incidence levels in the population, even when fertility change is associated with risk-reducing behaviour. Parity-based measures have a slight theoretical advantage over age-based measures, as they are less affected by differing levels of HIV-associated fetal mortality.

In a few countries empirical data on HIV prevalence among women attending antenatal clinics suggest that prevalence declines first occurred in younger women. For example, in Uganda, such trends were observed from about 1993 [9,10]; in Chiang Rai, Thailand, HIV prevalence fell markedly among young childbearing women from 1994 [33]. If such changes occur, careful consideration needs to be given to a number of biases. These include changes in the quality of data collection and HIV testing, utilization of antenatal care, and biases related to age structure, selection for sexual activity and HIV-associated reduction of fertility [6]. At the national level the selectivity of antenatal HIV sentinel surveillance sites may also present a problem – in most countries rural sites are severely underrepresented.

HIV trend data from other population groups can provide important evidence to verify observed trends in antenatal women. In Uganda, HIV incidence and prevalence trends from population-based cohort studies provided support for the trends in young antenatal women [24,34]. In Thailand, a multitude of data sources documented a decline in HIV prevalence [35–37] and indicated a decline in the incidence of other sexually transmitted diseases [38].

From the programme evaluation perspective two important questions follow the observation of a decline in HIV prevalence among younger antenatal women: to what extent can such changes be attributed to changes in sexual behaviour, and to what extent can changes in sexual behaviour be attributed to interventions? In Uganda, survey data on sexual behaviour [9] and modelling [10] were used to assess whether behavioural changes had caused the decline in HIV prevalence. In Thailand, a large number of behavioural surveys showed rapid declines in visits to female sex workers and increases in condom use [35,38]. Programme output statistics in both Uganda and Thailand suggested that some of the behavioural changes were associated with interventions.

Our analysis and simulations show that HIV prevalence trends among young antenatal women can be used as indicators of HIV incidence trends in most circumstances, provided caution is exercised in interpreting episodes of rapid change. A wide age interval should be used (15–24 years) rather than 5-year age groups, and monitoring HIV trends by parity 0 and 1 further enhances the ability of antenatal data to describe trends in the sexually active population. Surveillance of the epidemic and monitoring and evaluation of interventions will greatly benefit from a renewed and concerted effort to obtain reliable data on HIV prevalence trends among young women in antenatal clinics at national and sub-national levels [13].

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Appendix A: the projection model

In general, the population aged $a+1$ at time $t+1$, $P(a+1,\ t+1)$ is derived from the population aged a at time t, by considering all decremental forces, $\theta_I(a,t)$, which remove women from the cohort. If all forces governing movements between population groups are assumed constant within a single year, we can write:

$$P(a+1\ ,t+1) = P(a\ ,t)\exp[-\Sigma_I\theta_I(a\ ,t)]$$

Since we consider a relatively narrow age range of women, we assume the force of mortality from causes other than HIV, μ^o, are the same for all ages and unchanging over time. The force of mortality due to HIV, μ^h, is assumed to depend only on duration since infection, d.

Subgroups of the population are subject to decremental forces other than mortality, in particular, the number of virgins, $P^v(a+1,t+1)$, is given by:

$$P^v(a+1\ ,t+1) = P^v(a\ ,t)\exp[-\mu^o - \sigma(a\ ,t)],$$

where $\sigma(a,t)$ is the force of sexual initiation at age a and time t.

The number of HIV-negative, sexually active women $P^S(a+1,t+1)$, is thus:

$$P^S(a+1\ ,t+1) = P^S(a\ ,t)\exp[-\mu^o - \eta(t)]$$

$$+P^v(a\ ,t)\exp[-\mu^o - \eta(t)/2](1-\exp[\sigma(a\ ,t)]),$$

where $\eta(t)$ is the force of infection among the sexually active at time t, assumed independent of age.

The number of newly infected women, $P^h(a+1,t+1,\ 0)$, is:

$$P^h(a+1\ ,t+1\ ,0) =$$

$$P^S(a\ ,t)\exp(-\mu^o)(1-\exp[-\eta(t)])+$$

$$P^v(a\ ,t)\exp(-\mu^o)(1-\exp[\sigma(a\ ,t)])(1-\exp[-\eta(t)/2]),$$

and the number of HIV-positive women infected $d+1$ years ago, $P^h(a+1,t+1,d+1)$, is:

$$P^h(a+1\ ,t+1\ ,d+1) =$$

$$P^h(a\ ,t\ ,d)\exp[-\mu^o - \mu^h(d)].$$

The total number of HIV-positive women, $P^h(a,t)$, is obtained by summing across duration groups:

$$P^h(a\ ,t) = \Sigma_d P^h(a\ ,t\ ,d).$$

Assuming the sexually active are subject to a constant fertility force ϕ, the proportion $f^b(a+1,t+1)$ of sexually active women remaining at parity b from one year to the next is:

$$f^b(a+1\ ,t+1) = f^b(a\ ,t)\ \exp(-\phi).$$

Since ϕ is independent of age, achieved parity will depend only on time since sexual debut. Assuming that women cannot experience more than one birth per year, the proportion of women γ years after sexual debut at parity b, $F^b(\gamma)$, is:

$$F^b(\gamma) = f^b(a\ ,\ t)\ \exp(-\phi\,\gamma)$$

We assume the initial population is quasi-stable, so that with a growth rate, r, the initial structure can be defined using:

$$P(a+1\ ,0) = P(a\ ,0)\ \exp(-\mu^o - r),$$

and the number of new 10-year old entrants each year, $P(10,t)$, is given by:

$$P(10\ ,t+1) = P(10\ ,\ t)\ \exp(r).$$