

Trend in HIV prevalence among tuberculosis patients in Tanzania, 1991-1998

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SUMMARY

OBJECTIVE: To determine the trend in human immunodeficiency virus (HIV) prevalence among tuberculosis patients in Tanzania and estimate what proportion of the increase in notification rates between the surveys was directly attributable to HIV infection.

METHODS: Consecutive tuberculosis patients were enrolled over 6-month periods in most regions. Demographic and clinical data were collected on standard forms and a single HIV ELISA test performed. Trends in tuberculosis incidence were estimated from regional notification data.

RESULTS: Of 10 612 eligible tuberculosis patients, 44% had HIV infection, compared with 32% in the previous survey. The largest increase was observed in the young-

est birth cohorts, suggesting active HIV transmission. Approximately 60% of the increase in notification rates of smear-positive tuberculosis between surveys was directly attributable to HIV infection.

CONCLUSION: The HIV epidemic has had a strong influence on tuberculosis incidence. However, since 1995, tuberculosis notification data have increased less steeply, AIDS notifications have gone down, and HIV prevalence in blood donors has not increased a great deal. Another survey among tuberculosis patients in 5 years' time may show whether the HIV epidemic in Tanzania has reached a maximum or steady state.

KEY WORDS: tuberculosis; HIV; prevalence; epidemiology

HUMAN immunodeficiency virus (HIV) infection is the strongest known risk factor for progression from tuberculosis infection to disease.¹ In Sub-Saharan Africa, the HIV epidemic has had a strong influence on tuberculosis epidemiology.²⁻⁴ Tuberculosis incidence rates in Africa are among the highest in the world.^{5,6} These rates have increased strongly over the past two decades as a result of the HIV epidemic.⁷

Tanzania is among the 22 countries with the highest tuberculosis burden in the world.⁸ Of these 22 high burden countries, only three came close to meeting the World Health Organization (WHO) targets for tuberculosis control, Tanzania being one of these three.⁸ The notification rate of smear-positive tuberculosis in Tanzania increased from 38 per 100 000 population in the period 1983-1987 to 69/100 000 in the period 1993-1997. Most of this increase is attributed to the HIV epidemic.⁹⁻¹² By the end of 1998, the Tanzania National AIDS Control Programme had reported 109 863 AIDS cases since the beginning of the AIDS epidemic and estimated that 1.63 million people (approximately 10%) in the age group 15 years and above had HIV infection.¹³

In 1991-1993, HIV prevalence among new smear-positive tuberculosis patients in Tanzania was found to be 28%.¹² We undertook a second survey in the period 1994-1998, in order to determine the trend in HIV prevalence among tuberculosis patients and estimate what proportion of the increase in notification rates between the surveys was directly attributable to HIV infection.

METHODS

The data collection methods for the second survey were the same as those used in the first.¹² Consecutive tuberculosis patients aged 15 years or more were enrolled in each region in specified study periods, usually lasting 6 months. A pre-coded form with a study identification number was used to record demographic and clinical information. Blood samples were labelled with the same identification number and sent for anonymous HIV testing. For HIV testing, a single ELISA test (Vironostika HIV Uni-Form II, Organon Teknika, Boxtel, The Netherlands) was used, as the aim of the survey was to obtain prevalence estimates,

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and results were not used for individual case management. The study protocol was reviewed and approved by the Tanzania Ministry of Health and the National AIDS Control Programme.

Data were double entered and discrepancies resolved after comparison against the study forms. In the case definition of tuberculosis patients the following categories were distinguished: 1) new pulmonary smear-positive tuberculosis; 2) new pulmonary smear-negative tuberculosis with the diagnosis supported by an abnormal chest X-ray; 3) new extra-pulmonary tuberculosis; and 4) relapsed cases with pulmonary smear-positive tuberculosis. Patients who did not fit any of these categories, for instance due to missing information on any of the required variables, were excluded from the analyses concerned.

Notification rates of smear-positive tuberculosis were calculated as a moving average of three consecutive years. In order to present trends in notification rates of smear-positive tuberculosis in regions, we categorised regions into three groups, depending on the notification rate in 1998 relative to the period 1981–1985. The following cut-off points were selected for this division: relative notification rate <1.50 (group 1), 1.50–1.99 (group 2), and 2.00 or more (group 3).

In the first HIV survey among tuberculosis patients in Tanzania it was estimated that 86% of new smear-positive tuberculosis in patients with HIV infection was directly attributable to HIV, using the following formula:¹²

$$AF = 1 - (1/RR)$$

where AF is the attributable fraction in those with HIV infection and RR the relative risk of tuberculosis in HIV-infected people compared with the non-HIV-infected. RR was estimated from the odds ratio in a case-control study. From this, the population attributable fraction (PAF) can be estimated as 86% times the HIV prevalence among tuberculosis patients.

We estimated the population attributable fractions using results in the first and second survey, to estimate which fraction (F) of the increase in notification rates of new smear-positive tuberculosis between the surveys was directly attributable to HIV infection, using the following formula:

$$F = (\text{Notif2.PAF2} - \text{Notif1.PAF1}) / (\text{Notif2} - \text{Notif1}),$$

where PAF 2 and PAF1 refer to the population attributable fractions during the second and first survey, respectively, and Notif2 and Notif1 to the crude tuberculosis notification rates during the second and first survey, respectively. The numerator represents the increase in notification rates directly attributable to HIV infection and the denominator the total increase in notification rates. F was considered to have a maximum of 100%.

RESULTS

Over the study period, 12 938 tuberculosis patients were enrolled in the study, 83% of whom had complete information and were included in the analysis (Table 1). Kigoma Region did not participate in the second survey due to organisational problems. Zanzibar joined the second survey but had not participated in the first. Thus, the analysis of change over time is restricted to mainland Tanzania, excluding Kigoma Region.

In most regions of Tanzania mainland an increase in HIV prevalence among tuberculosis patients was observed (Table 1). However, the increase varied strongly between regions, with the strongest increases (odds ratios [OR] >3) in the Mtwara, Rukwa, Tabora and Tanga Regions and little or no increase (OR 1.0–1.2) in the Kagera, Kilimanjaro, Morogoro and Ruvuma Regions.

The greatest increase in HIV prevalence was seen in those aged 35–54 years (Table 2, Figure 1). However, a different perspective is provided by an analysis of HIV prevalence in birth cohorts: the strongest increase in HIV prevalence was observed in the youngest birth cohorts (Table 2, Figure 2). Although HIV prevalence in the first survey was associated with female sex, urban residence, smear-negative and extra-pulmonary tuberculosis, and the presence of a BCG scar,¹² the increase in HIV prevalence was not associated with these variables (Table 2).

Trends in notification rates of smear-positive tuberculosis varied strongly between regions (Figure 3), with high relative increases in Dar es Salaam, Pwani, Iringa, Mbeya, Kilimanjaro and Kigoma (Table 3). In all regions of Tanzania mainland the rate of increase appears to have slowed down since 1995 (Figure 3). In no region did the year of steepest increase occur after 1995 (Table 3). HIV prevalence in tuberculosis patients during the first round was strongly associated with the relative increase in notification rates ($r = 0.66$, 95% confidence interval [CI] 0.30–0.85).

The increase in HIV prevalence tended to be largest in regions of group 1, which had shown the least increase in tuberculosis notification rates, and had the lowest HIV prevalence during the first round (Table 2). However, the association between the relative tuberculosis notification rates of the regions and their increases in HIV prevalence was not significant ($r = -0.04$, 95% CI -0.47 – 0.41). In two regions the results were particularly unexpected. In Tabora Region the notification rate was stable, at 33–34/100 000, while HIV prevalence increased from 13% to 44%. In Morogoro tuberculosis notification rates increased from 54 to 71/100 000, although HIV prevalence among tuberculosis patients decreased from 31% to 25%. How-

Table 1 HIV prevalence in tuberculosis patients in two surveys in Tanzania: 1991–1993 and 1994–1998

Region	1991–1993				1994–1998				Odds ratio (increase)
	Year	Examined n	Eligible* n (%)	HIV positive (%)	Year	Examined n	Eligible* n (%)	HIV positive (%)	
Arusha	1991	337	258 (77)	21	1995	876	579 (66)	25	1.3
DSM	1991	456	381 (84)	41	1994–1995	1 258	1 053 (84)	49	1.4
	1993	458	423 (92)	46	1996	1 140	976 (86)	56	1.8
Dodoma	1992	242	227 (94)	21	1995–1996	609	547 (90)	48	1.3
	1993	714	613 (86)	59	1997–1998	441	372 (84)	40	2.5
Kagera	1991	357	303 (85)	37	1995	904	643 (71)	67	1.4
Kigoma	1993	193	169 (88)	19	1995	472	395 (84)	39	1.1
	1991	172	147 (85)	42	—	—	—	—	na
Kilimanjaro	1992	213	198 (93)	14	1995	555	513 (92)	45	1.1
	1992–1993	258	246 (95)	17	1996	389	358 (92)	32	2.9
Mara	1991	247	187 (76)	55	1994–1995	626	514 (82)	29	1.9
Mbeya	1992	504	474 (94)	33	1995–1996	569	409 (72)	68	1.7
Morogoro	1992	403	388 (96)	10	1996	557	467 (84)	33	1.0
Mtwara	1991	594	553 (93)	26	1994–1995	806	723 (90)	26	3.3
Mwanza	1992	274	229 (84)	31	1995–1996	349	307 (88)	51	1.4
Pwani	1993	150	142 (95)	44	1998	249	237 (95)	77	2.4
Rukwa	1993	137	125 (91)	40	1998	260	220 (85)	40	4.3
Ruvuma	1992–1993	273	262 (96)	25	1996	562	500 (89)	30	1.0
Shinyanga	1993	229	198 (86)	21	1996–1997	384	297 (77)	28	1.3
Singida	1993	185	156 (84)	16	1996–1997	267	233 (87)	47	1.5
Tabora	1991	532	405 (76)	19	1995	1 119	889 (79)	44	4.6
Tanga	na	—	—	—	1994–1995	117	104 (89)	21	3.3
Zanzibar	na	—	—	—	1994–1995	117	104 (89)	21	na
Subtotal†		6 735	5 915 (88)	32		12 821	10 612 (83)	44	1.7
Total		6 928	6 084 (88)	32		12 938	10 716 (83)	44	1.7

* Eligible: no missing values in sex, type of tuberculosis and HIV result and age at least 15 years.

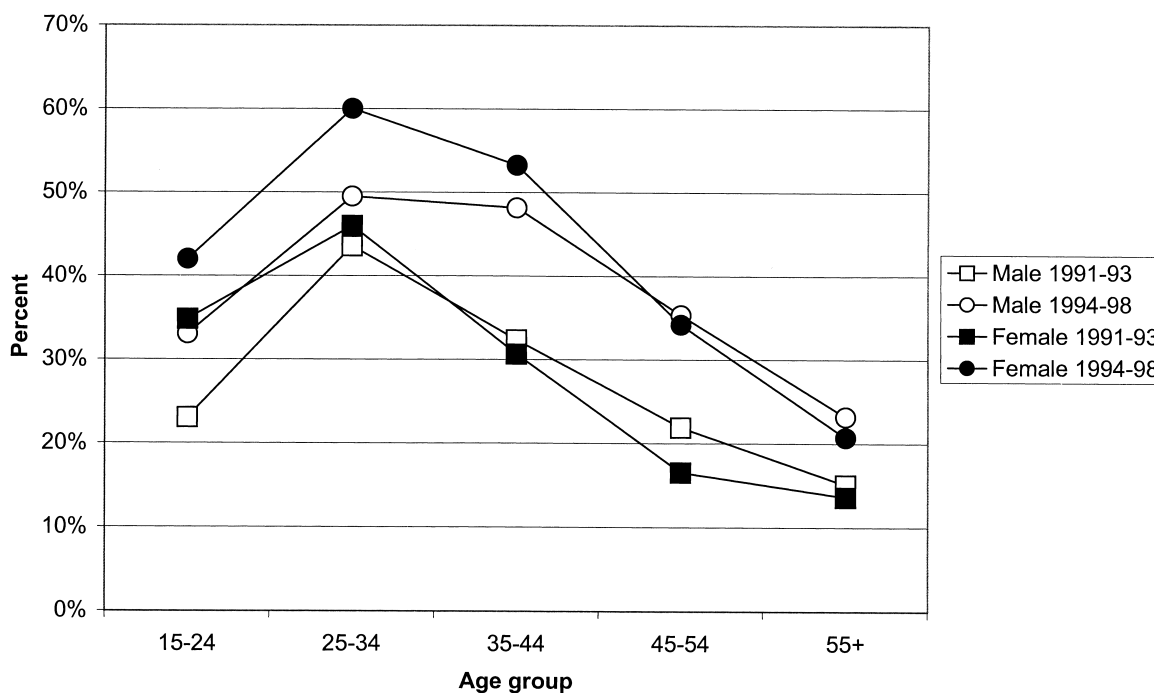
† Excluding Kigoma and Zanzibar.

DSM = Dar es Salaam; na = not applicable.

Table 2 Increases in HIV prevalence in tuberculosis patients in Tanzania by sex, age, birth cohort, urban-rural residence, type of tuberculosis and presence of BCG scar

	1991–1993 HIV+/total (%)	1994–1998 HIV+/total (%)	Odds ratio (95%CI)
Sex			
Male	1138/3813 (30)	2667/6507 (41)	1.6 (1.5–1.8)
Female	782/2271 (34)	1986/4105 (48)	1.8 (1.6–2.0)
Age group (years)			
15–24	334/1176 (28)	771/2043 (38)	1.5 (1.3–1.8)
25–34	904/2027 (45)	1963/3628 (54)	1.5 (1.3–1.6)
35–44	398/1253 (32)	1148/2292 (50)	2.2 (1.9–2.5)
45–54	162/798 (20)	483/1377 (35)	2.1 (1.7–2.6)
55+	122/830 (15)	288/1272 (23)	1.7 (1.3–2.2)
Birth cohort			
1968–1977	344/1135 (30)	1444/3116 (46)	2.0 (1.7–2.3)
1958–1967	858/1932 (44)	1807/3322 (54)	1.5 (1.3–1.7)
1948–1957	381/1191 (32)	792/1800 (44)	1.7 (1.4–2.0)
1938–1947	148/752 (20)	306/1048 (29)	1.7 (1.3–2.1)
Before 1938	114/788 (14)	200/954 (21)	1.6 (1.2–2.0)
Residence			
Urban	951/2557 (37)	2520/5201 (48)	1.6 (1.4–1.8)
Rural	969/3527 (27)	2133/5411 (39)	1.7 (1.6–1.9)
Type of tuberculosis			
New smear-positive	1136/4061 (28)	2699/6745 (40)	1.7 (1.6–1.9)
New smear-negative	445/1093 (41)	1156/2299 (50)	1.5 (1.3–1.7)
New extra-pulmonary	255/646 (39)	620/1146 (54)	1.8 (1.5–2.2)
Relapse (smear-positive)	84/284 (30)	178/422 (42)	1.7 (1.3–2.4)
BCG scar			
Present	937/2384 (39)	2508/5133 (49)	1.5 (1.3–1.6)
Absent	742/2888 (26)	1906/4925 (39)	1.8 (1.7–2.0)
Not recorded	241/812 (30)	239/554 (43)	1.8 (1.4–2.3)
Group*			
1	248/1323 (19)	629/2093 (30)	1.9 (1.6–2.2)
2	647/2470 (26)	1323/3668 (36)	1.6 (1.4–1.8)
3	1007/2122 (47)	2672/4790 (56)	1.4 (1.3–1.6)

* Groups of regions were defined according to the relative tuberculosis notification rate in 1998 compared with 1981–1985 (see Table 3). Group 1: RR <1.50, Group 2: RR 1.50–1.99, Group 3: RR 2.00 or more.
HIV = human immunodeficiency virus; CI = confidence interval; BCG = bacille Calmette Guérin.

**Figure 1** HIV prevalence by age and sex in tuberculosis patients in Tanzania: 1991–1993 and 1994–1998. HIV = human immunodeficiency virus.

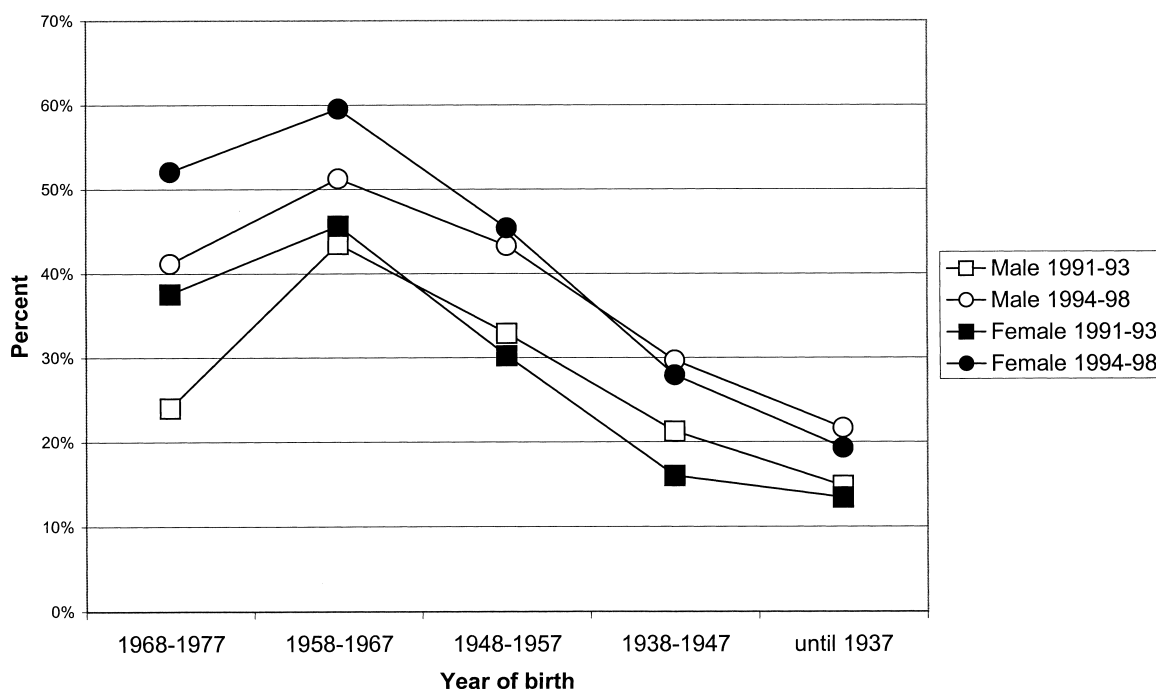


Figure 2 HIV prevalence by birth cohort and sex in tuberculosis patients in Tanzania: 1991–1993 and 1994–1998. HIV = human immunodeficiency virus.

ever, this decrease in HIV prevalence was not significant ($\chi^2 1.5, P > 0.2$).

In the first HIV survey among tuberculosis patients in Tanzania it was estimated that 86% of new smear-positive cases with HIV infection were

directly attributable to HIV.¹² If this proportion has not changed, it can be estimated that approximately 60% of the increase in tuberculosis notification rates between the surveys was directly attributable to HIV (Table 4).

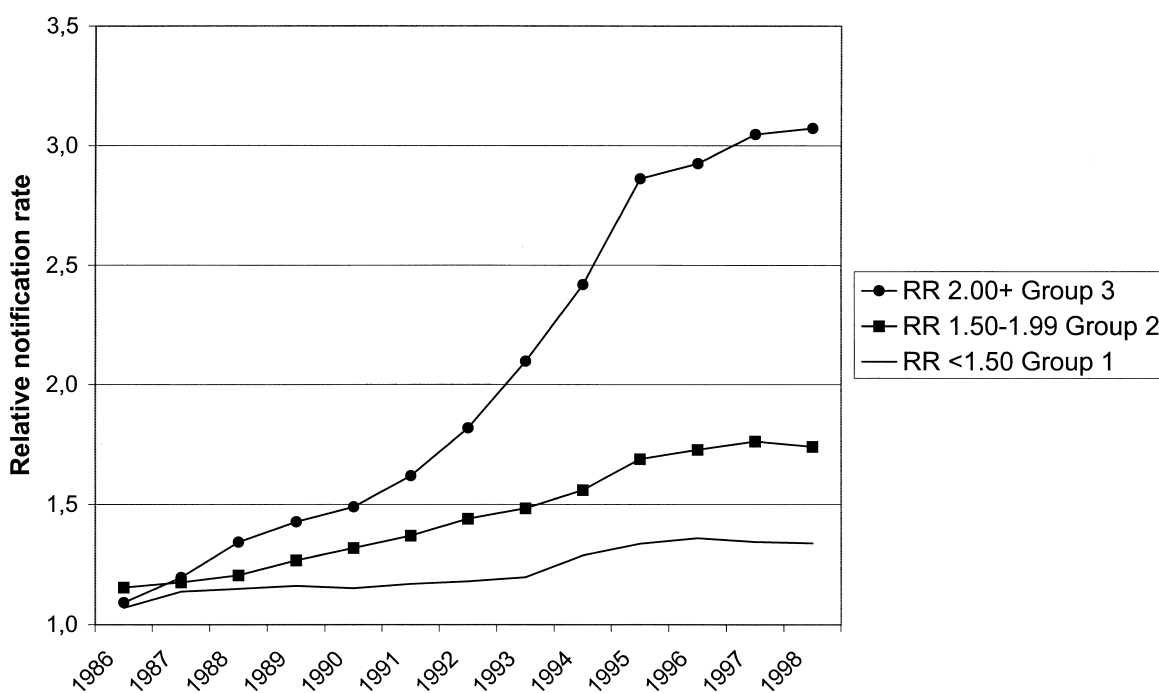


Figure 3 Notification rates of smear-positive tuberculosis in four groups of regions in mainland Tanzania, relative to the period 1981–1985.

Table 3 Trend in tuberculosis notification in Tanzania mainland, in comparison with the period 1981–1985

Region	Relative notification rate 1998	Year in which increase first exceeded baseline (1981–1985) by			Peak increase		Group
		30%	50%	100%	Size (%)	Year	
Arusha	1.4	1994	—	—	16	1986	1
Dar es Salaam	4.8	1987	1988	1991	26	1993	3
Dodoma	1.6	1994	1995	—	19	1995	2
Iringa	2.7	1988	1989	1993	34	1986	3
Kagera	1.7	1989	1992	—	14	1994	2
Kigoma	2.2	1989	1993	1995	26	1987	3
Kilimanjaro	2.6	1992	1993	1994	21	1993, 1995	3
Lindi	1.3	1996	—	—	8	1994	1
Mara	1.7	1994	1995	—	15	1995	2
Mbeya	2.8	1990	1991	1994	12	1990	3
Morogoro	1.9	1991	1994	—	15	1995	2
Mtwara	1.4	1995	—	—	12	1994	1
Mwanza	1.5	1990	1992	—	6	1991	2
Pwani	2.6	1988	1991	1995	19	1986	3
Rukwa	2.9	1986	1991	1994	20	1995	3
Ruvuma	1.5	1995	—	—	13	1991	1
Shinyanga	1.9	1988	1990	—	9	1992	2
Singida	1.2	—	—	—	11	1987, 1989, 1994	1
Tabora	1.2	1989	—	—	15	1987	1
Tanga	2.0	1990	1994	—	18	1995	1
Total	2.2	1989	1992	1995	12	1995	

DISCUSSION

Between 1991–1993 and 1994–1998, HIV prevalence among tuberculosis patients in mainland Tanzania (excluding Kigoma) increased sharply from 28% to 40% in new, smear-positive tuberculosis patients. The steepest increase in HIV prevalence was observed

in the youngest birth cohorts, suggesting that there has been much HIV transmission among young adults in recent years. Between the surveys, notification rates of new smear-positive tuberculosis increased from 54 to 74/100 000 population, and we estimate that approximately 60% of this increase was directly attributable to HIV.

Table 4 Regional increases in notification rates of smear-positive tuberculosis and proportion of increase directly attributable to HIV infection

Region	Round 1: 1991–1993				Round 2: 1994–1998				
	Year	Notification rate	HIV positive (%)	Pop. attrib fraction (%)	Year	Notification rate	HIV positive (%)	Pop. attrib fraction (%)	Increase explained by HIV (%)
Arusha	1991	47.2	17	15	1995	56.1	23	20	47
DSM	1991	136.9	36	31	1994–1995	227.2	48	41	56
	1993	208.3	43	37	1996	287.9	48	41	50
					1997	301.2	41	36	39
Dodoma	1992	33.3	15	13	1995–1996	46.3	38	32	82
Iringa	1993	62.7	57	49	1997–1998	83.5	67	57	83
Kagera	1991	34.0	34	29	1995	39.4	37	31	48
Kilimanjaro	1991	30.5	33	28	1995	61.0	41	35	42
Lindi	1992	62.4	8	7	1996	81.5	30	25	86
Mara	1992–1993	37.1	15	13	1996	49.9	28	24	56
Mbeya	1991	29.3	43	37	1994–1995	44.2	65	56	92
Morogoro	1992	54.4	31	27	1995–1996	70.6	25	22	6
Mtwara	1992	97.5	9	8	1996	125.4	26	22	73
Mwanza	1991	63.1	27	23	1994–1995	64.7	29	25	85
Pwani	1992	69.9	28	24	1995–1996	90.3	45	39	90
Rukwa	1993	24.2	47	41	1998	36.1	75	65	100
Ruvuma	1993	36.9	39	33	1998	42.1	41	36	52
Shinyanga	1992–1993	34.8	23	20	1996	37.1	27	23	68
Singida	1993	45.6	22	19	1996–1997	50.3	26	22	51
Tabora	1993	32.6	13	11	1996–1997	33.8	44	38	100
Tanga	1991	56.8	20	18	1995	74.9	42	36	96
Total	1991–1993	54.0	28	24	1994–1998	74.4	40	34	61

DSM = Dar es Salaam; HIV = human immunodeficiency virus.

Since 1995, tuberculosis notification rates in Tanzania have still been increasing, but not as steeply as before. There may be various reasons for this more gradual increase, one of which might be that the HIV epidemic is reaching a maximum or steady state in many regions. However, this explanation would require supporting evidence, as alternative explanations need to be considered as well, such as deterioration of the reporting system or a reduction in the tuberculosis case detection rate.

Some supporting evidence is provided by HIV/AIDS surveillance. The number of AIDS notifications was approximately 35 500 in 1992–1994 and 23 500 in 1995–1997.¹³ In 1995–1997, HIV prevalence among male blood donors was stable, at around 7.5%, and among female blood donors it increased from 9.4% to 11.6%.¹³ Sentinel surveillance among pregnant women at eight sites in four regions suggested no increase since 1995 at seven sites in three regions.¹³ Unfortunately, these data do not allow a clear conclusion to be drawn as to the trend of the HIV/AIDS epidemic: AIDS notifications are incomplete, and groups included in HIV sero-surveillance may suffer from variation in selection bias over time. For instance, as noted in the previous report,¹² in high prevalence areas increasing emphasis may be given to the recruitment of low-risk blood donors, making trends in HIV prevalence over time difficult to interpret. Sentinel surveillance among pregnant women covers four regions only, and thus does not provide information on trends in the rest of the country.

It is possible that the HIV epidemic has reached a maximum in some regions, but not in others. An example of the former may be Dar es Salaam, where HIV prevalence in tuberculosis patients appears to have been stable since 1995. As selection bias among tuberculosis patients is probably less important than among blood donors, it may be useful to repeat this study after 5 years to provide more conclusive evidence on whether or not the levelling off of tuberculosis notification rates at regional and national level can be explained by the HIV epidemic having reached a maximum or steady state.

The effect of the HIV epidemic on tuberculosis incidence is complicated. HIV increases susceptibility to primary tuberculosis and to reactivation of latent tuberculous infection, and thus increases tuberculosis incidence directly. This increased susceptibility to tuberculosis disease increases with advancing immunodeficiency due to HIV infection, and thus may vary over time. As a substantial proportion of the additional tuberculosis cases directly attributable to HIV infection is smear-positive, the HIV epidemic may lead to increased transmission of *Mycobacterium tuberculosis* and thereby increase tuberculosis incidence indirectly.¹⁴ Therefore, even if increasing tuberculosis incidence were completely due to the HIV epidemic, this would not be measured completely by the direct

estimates from the present study. However, these direct estimates suggest that at least a substantial proportion of the increase was attributable to HIV infection.

In two regions the trend in tuberculosis notifications was different from the trend in HIV prevalence in tuberculosis patients. In Tabora notifications were stable, while HIV prevalence increased strongly; in Morogoro, the opposite was observed. It is difficult to determine the causes of these inconsistencies. In Tabora, HIV prevalence in blood donors had increased much less (from 4% in 1993 to 6% in 1997) than is reported here among tuberculosis patients. This suggests that either the increase in HIV prevalence in tuberculosis patients was overestimated, or that blood donor selection changed strongly over time. In Morogoro, the stable HIV prevalence in tuberculosis patients between 1992 and 1997 is consistent with stable HIV prevalence among blood donors.¹³ If no change has occurred in the recruitment of blood donors, the increase in tuberculosis notifications may be due to factors other than HIV infection, such as improved tuberculosis notification or case detection, or increased transmission due to failing control.

We conclude that HIV prevalence in tuberculosis patients and tuberculosis notification rates has increased considerably in recent years. The increase in the youngest birth cohorts suggest ongoing HIV transmission. However, there are some indications that these increases may slow down over the next few years. Continued surveillance of tuberculosis and HIV/AIDS is important to assess these trends.

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R É S U M É

OBJECTIF : Déterminer en Tanzanie la tendance de prévalence du virus de l'immunodéficience humaine (VIH) parmi les patients tuberculeux et estimer la proportion de l'augmentation des taux de déclaration entre deux enquêtes qui est strictement attribuable à l'infection par le VIH.

MÉTHODES : On a enrôlé pendant des périodes de 6 mois, dans la plupart des régions, des patients consécutifs atteints de tuberculose. On a recueilli les données démographiques et cliniques sur des formulaires standard et pratiqué un seul test ELISA pour le VIH. Les tendances quant à l'incidence de la tuberculose ont été estimées à partir des données régionales de déclaration.

RÉSULTATS : Parmi 10.612 patients tuberculeux éligibles, l'infection VIH a été décelée chez 44% par comparaison avec 32% lors de l'enquête précédente. L'aug-

mentation la plus importante a été observée dans les cohortes de naissances les plus récentes, suggérant une transmission active du VIH. Environ 60% de l'augmentation des taux de déclaration de tuberculose à bacilloscopie positive survenue entre les deux enquêtes est directement attribuable à l'infection par le VIH.

CONCLUSION : L'épidémie VIH a une forte influence sur l'incidence de la tuberculose. Toutefois depuis 1995, les données de déclaration de tuberculose augmentent de façon moins importante, les déclarations de SIDA diminuent et la prévalence du VIH parmi les donneurs de sang n'augmente pas beaucoup. Une étude supplémentaire pratiquée dans 5 ans parmi les patients tuberculeux pourrait démontrer si l'épidémie de VIH en Tanzanie a atteint son maximum ou un niveau de stabilité.

R E S U M E N

OBJETIVO : Determinar la tendencia de la prevalencia del virus de la inmunodeficiencia humana (VIH) entre los pacientes tuberculosos en Tanzania y estimar qué proporción del aumento en las tasas de notificación entre las encuestas era atribuible directamente a la infección por VIH.

MÉTODOS : Los pacientes tuberculosos consecutivos fueron agrupados en períodos de 6 meses en la mayoría de las regiones. Se recogieron datos demográficos y clínicos en forma estándar y se efectuó un único test VIH-ELISA. Las tendencias en la incidencia de tuberculosis se estimaron con los datos de notificación regional.

RESULTADOS : Sobre 10.612 tuberculosos elegibles, el 44% tenían infección por VIH, comparados con el 32% en la encuesta previa. Los mayores aumentos se obser-

varon en las cohortes más jóvenes, lo que sugiere una transmisión activa del VIH. Aproximadamente el 60% del aumento en las tasas de notificación de los tuberculosos con esputo positivo entre las encuestas fue atribuido directamente a la infección por VIH.

CONCLUSIÓN : La epidemia de VIH ha tenido una gran influencia en la incidencia de la tuberculosis. Sin embargo, desde 1995, los datos de notificación de la tuberculosis aumentaron menos rápidamente, la notificación de VIH descendió y la prevalencia entre los dadores de sangre no aumentó mucho. Otra encuesta entre los enfermos tuberculosos dentro de 5 años mostrará si la epidemia de VIH en Tanzania ha alcanzado un máximo o está estabilizada.
