Epidemiol. Infect. (1991), **107**, 69–80
Printed in Great Britain

Civil war and the spread of AIDS in Central Africa

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(*Accepted 19 February*)

SUMMARY

Using ordinary least squares regression techniques this paper demonstrates, for the first time, that the classic association of war and disease substantially accounts for the presently observed geographical distribution of reported clinical AIDS cases in Uganda. Both the spread of HIV 1 infection in the 1980s, and the subsequent development of AIDS to its 1990 spatial pattern, are shown to be significantly and positively correlated with ethnic patterns of recruitment into the Ugandan National Liberation Army (UNLA) after the overthrow of Idi Amin some 10 years earlier in 1979. This correlation reflects the estimated mean incubation period of 8–10 years for HIV 1 and underlines the need for cognizance of *historical* factors which may have influenced *current* patterns of AIDS seen in Central Africa. The findings may have important implications for AIDS forecasting and control in African countries which have recently experienced war. The results are compared with parallel analyses of other HIV hypotheses advanced to account for the reported geographical distribution of AIDS in Uganda.

INTRODUCTION

Pioneering studies from Zaire and Rwanda in 1983 revealed the endemic nature of heterosexually transmitted acquired immunodeficiency syndrome (AIDS) in Central Africa [1, 2]. Subsequent surveillance programmes for both AIDS and its causative agent, the human immunodeficiency virus (HIV), have revealed the enormity of the AIDS problem in the region. For example, in Uganda alone, official estimates placed the number of HIV carriers at one million (6% of the Ugandan population) by the end of 1989, while the 12444 reported AIDS cases probably under-represents the true clinical AIDS burden by an order of magnitude [3, 4]. Two principal strains of HIV are currently recognized by Africa: HIV 1 and HIV 2. Virtually all AIDS cases reported from Central Africa, and for which serological information is available, have been associated with HIV 1 infection. Thus, in this paper, the term HIV will refer to HIV 1 unless otherwise specified.

Both the conveyors responsible for the evolving geographical patterns of AIDS in Central Africa and the corridors of virus spread remain ill-defined. In particular, scant attention has been paid to the historical association between soldiers,

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prostitute contact and the spread of sexually transmitted diseases [5, 6] for the diffusion of HIV in the countries of Central Africa which have recently experienced civil war. This is particularly surprising for a region where heterosexual intercourse is the predominant route of HIV exposure and where extraordinarily high HIV prevalences have been documented in female prostitutes [7–9].

In this paper, we present the results of statistical analyses which suggest that military-associated factors have been instrumental in the development of the apparent geographical pattern of clinical AIDS in one country of the region, Uganda. The results are compared with a parallel analysis of two other hypotheses, outlined below, which have been proposed to account for the distribution of AIDS in Uganda.

MATERIALS AND METHODS

Information relating to the place of residence of the 12444 Ugandan AIDS cases diagnosed by the end of 1989 and reported to the Ugandan Ministry of Health by 28 February 1990 was supplied by the Ugandan AIDS Control Programme [3]. These case totals are shown in Table 1. Cumulative AIDS case incidence rates per 100000 population for each of the 34 districts of the country were calculated by scaling these data by the 1989 mid-point population estimates of each district generated from the 1980 Ugandan Census [10]. The rates are illustrated in Fig. 1.

Figure 1 shows that a distinct bipolar pattern underlies the Ugandan AIDS epidemic. The southern districts of Rakai, Masaka and Kampala, situated along the shore of Lake Victoria, represent a primary focus of reported AIDS cases. In these districts, cumulative AIDS case incidence rates range between 167 and 517 per 100000 population and may be compared with the national rate of 74 per 100000. In the north of Uganda, a secondary focus of high AIDS incidence (255 per 100000) is centred on Gulu and neighbouring districts. In most other districts, incidence rates fall below 50 per 100000.

The pattern observed in Fig. 1 may partly reflect geographical variations in the completeness of AIDS case reporting in Uganda. While the possible impact of data quality is considered in more detail in our discussion of results, we preface our analysis with a cautionary note here. It is currently impossible to be certain about the completeness of the data because of the passive nature of the country's surveillance system [11]; geographical variations in both the provision and capabilities of health-care services may generate unknown spatial variations in the quality of AIDS data. In particular completeness of reporting is likely to be higher in major towns, such as Kampala, which have been foci of AIDS research. For example, Kampala district has an AIDS reporting rate (517 per 100000) which is twice that of any other district. In addition, the place of residence of an AIDS case is not necessarily the place of infection, especially given the long incubation period between primary HIV infection and the development of AIDS. However, despite these caveats, restricted analysis of HIV prevalence in healthy adult cohorts in different parts of the country broadly accords with the reported distribution of AIDS cases shown in Fig. 1 [12–17]. Two principal hypotheses have recently been proposed to explain how the apparently distinct distribution of AIDS in Uganda geography in Fig. 1 has evolved: (1) The *truck town hypothesis* [18]. This proposes that the geographical distribution of HIV and AIDS reflects a diffusion process in

Table 1. *Reported Ugandan AIDS cases to 28 February 1989 by district of residence* (*source*: ref. [3])

District	Cases	District	Cases
Apac	231	Lira	295
Arua	172	Luwero	178
Bundibugyo	9	Masaka	2433
Bushenyi	112	Masindi	36
Gulu	822	Mbale	136
Hoima	19	Mbarara	341
Iganga	117	Moroto	98
Jinja	267	Moyo	9
Kabale	170	Mpigi	599
Kabarole	435	Mubende	158
Kalangala	7	Mukono	357
Kampala	3335	Nebbi	251
Kamuli	29	Rakai	677
Kapchorwa	3	Rukungiri	55
Kasese	232	Soroti	84
Kitgum	216	Tororo	147
Kotido	39	Other	293
Kumi	82		

which major roads act as the principal corridors of virus spread between urban areas and other proximal settlements. (2) The *migrant labour hypothesis* [19, 20]. Here, HIV is hypothesized to have diffused from areas of labour demand in urban areas to areas of labour supply in rural districts through a process of return migration.

Truck town hypothesis

To test the truck town hypothesis as an explanation of the apparent disease pattern shown in Fig. 1, measures of the degree of urbanization of each district (absolute urban population levels, urban populations as a percentage of the total population, district population densities and growth rates) were taken from the 1980 census [10]. In addition four principal routeways were defined (see Fig. 2, with Fig. 1 for locations) [21].

Road A. The West–East truck route which links the countries of Zaire, Rwanda and Tanzania with the districts of southern Uganda and the Kenyan port of Mombasa. This road skirts the southern border of Uganda. It runs from Kabale to Masaka, follows the perimeter of Lake Victoria eastwards to Kampala, and then continues east into Kenya for Mombasa.

Road B. The truck route which links southeastern Uganda (Mbale) with the central and northern districts of Soroti, Lira and Gulu.

Road C. The road which links Kampala with Gulu via Masindi.

Road D. The road which links Zaire with southwest Uganda, running via the western border of Uganda from Arua in the northwest to Gulu, Masindi, Fort Portal and on to Kabale.

For the purposes of statistical analysis, the proximity of a district to each of these major roads was defined as the shortest distance from the geographical centroid of a district to the highway.

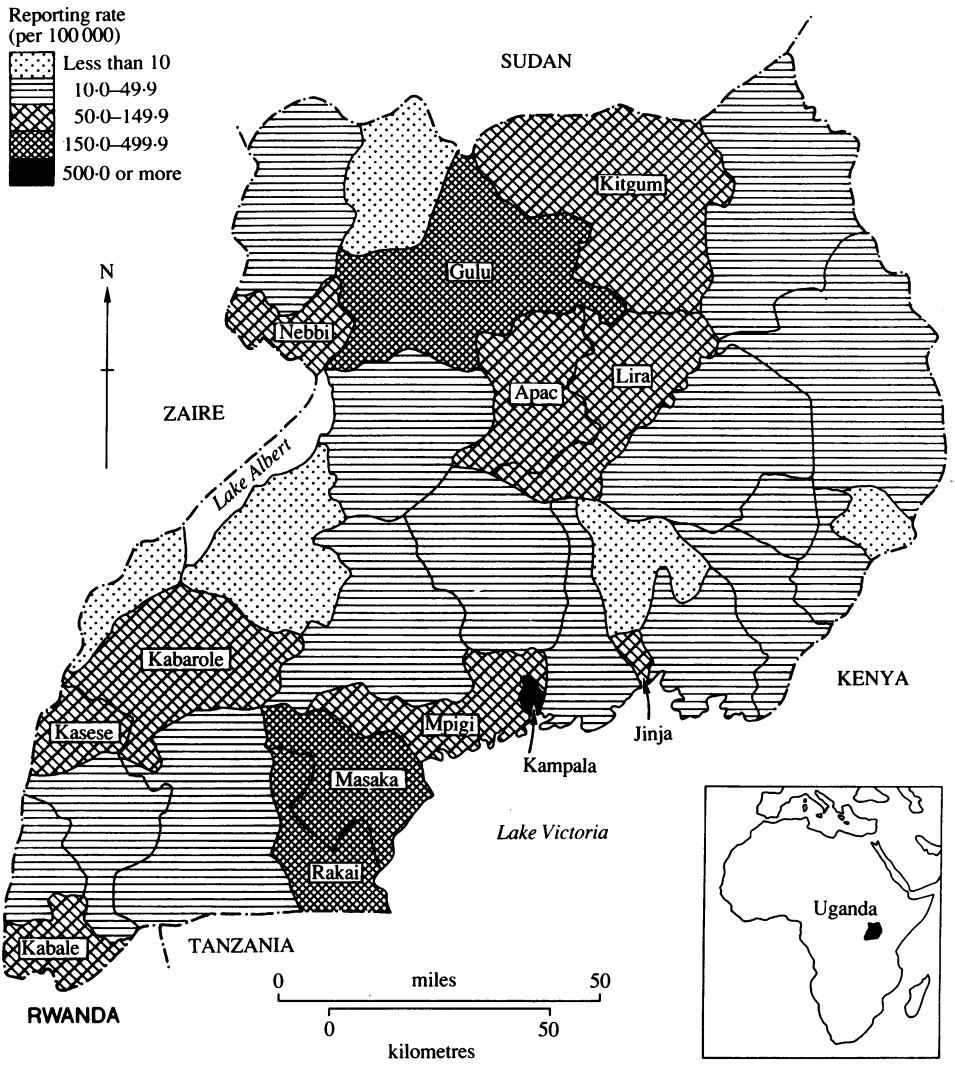

Figure 1. Reported cumulative AIDS incidence rates per 100 000 population to February 1990 for 34 districts of Uganda.

Migrant labour hypothesis

For the migrant labour hypothesis, principal labour source (labour reserve) and reception (labour concentration) regions were defined as outlined in the original formulation of the hypothesis [19] for the distribution of reported clinical AIDS in Uganda (see Fig. 3). Labour source regions include much of northern and southwestern Uganda, while principal labour reception regions encompass the southern districts situated along the shore of Lake Victoria. Employing the migration data sources used by Hunt [19], the vectors shown in Fig. 3 refer to migration patterns over the 1960s. The principal labour reserve and labour concentration regions are believed to have remained constant over time [19] and the dummy variable techniques applied in this paper (see below) avoid problems associated with recent changes in the relative size of these migration streams.

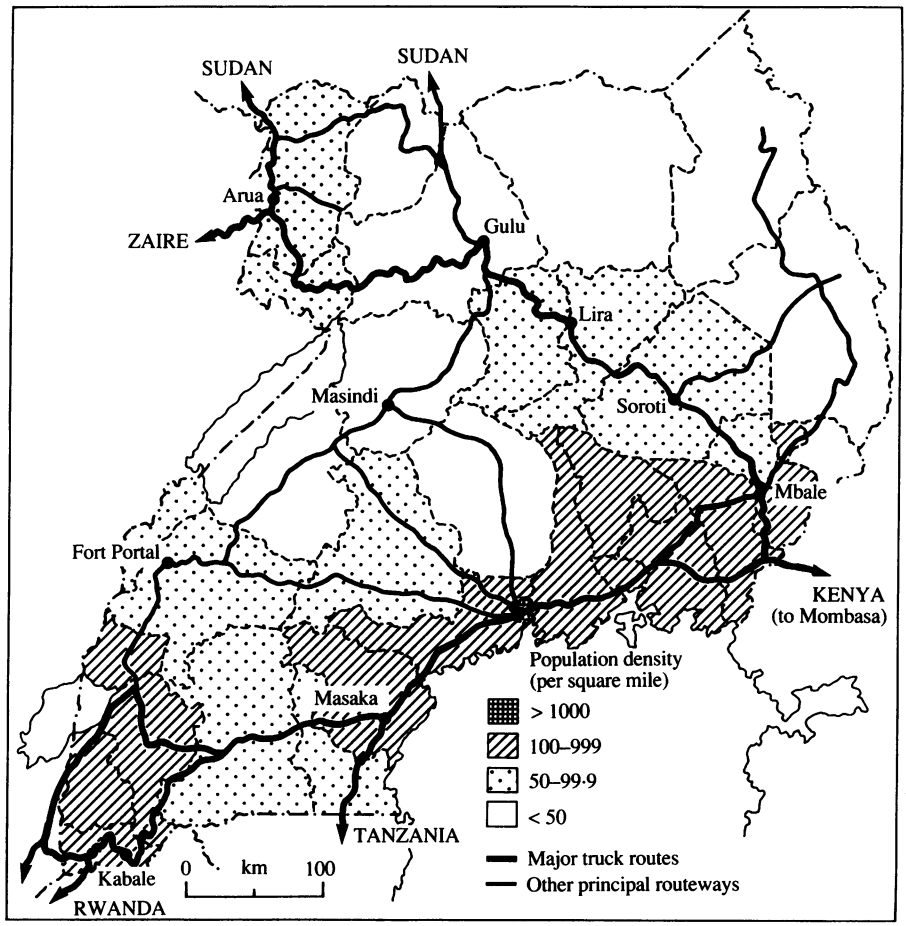

Figure 2. Location of principal roads in Uganda against a backcloth of district population densities per square mile.

Military involvement hypothesis

To assess a third hypothesis, that the Uganda military has been instrumental in the development of the apparent disease pattern shown in Fig. 1, ethnic recruitment levels into the Uganda National Liberation Army (UNLA) during November 1979, taken as a measure of the ethnic composition of the army shortly after the overthrow of Idi Amin in March 1979, were extracted from Omara-Otunnu [22]. Recruitment rates per 100000 population by major tribal grouping appear in Fig. 4. As this shows, UNLA recruitment rates varied greatly between tribes, ranging from 2.3 per 100000 among the Kakwa, Lugabara and Madi in the extreme northwest of the country to 250 per 100000 among the neighbouring Acholi. High recruitment rates are also noted among the Lango (77·7 per 100000) in Central Uganda and the Nkole (182·3) and Kiga (71·2) in the southwest. For all other tribes, recruitment rates fall below 30 per 100000

UNLA recruitment rates, by district, were estimated by projection of Fig. 4 onto a district map, with proportional redistribution of recruitment levels based upon district population sizes.

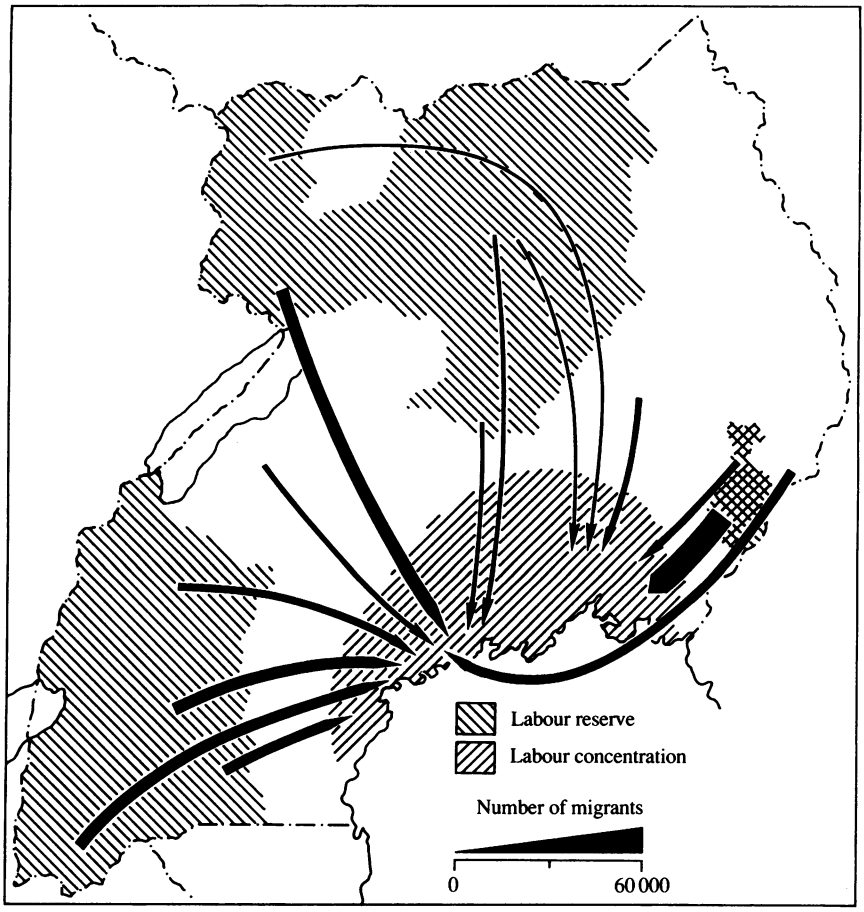

Figure 3. Principal labour supply and labour demand regions in Uganda. Vectors show inter-censal migration flows, 1959–69, upon which the original formulation of the hypothesis for migrant labour and the spread of AIDS in Uganda is based. Source: ref. [12].

Analysis

Each factor (measures of urbanization and proximity to roads, regions of in- and out-migration of labour and UNLA recruitment levels) was used as explanatory (x) variables in a series of simple and multiple regression models in which AIDS case incidence rates formed the dependent (y) variable. Model fitting was by ordinary least squares; n, the sample size, was 34, the number of districts. In preliminary analysis, Kampala district (incidence rate 517 per 100000) was found to act as an extreme outlier, heavily influencing the results of correlation and regression analysis. Accordingly, Kampala district was omitted from all procedures outlined below [23]. For the migrant labour hypothesis, dummy variable techniques were utilized; a source or reception district was allocated a value of 1 while all other districts were allocated a value of 0. Regression residuals were tested for evidence of spatial autocorrelation as described elsewhere in [24].

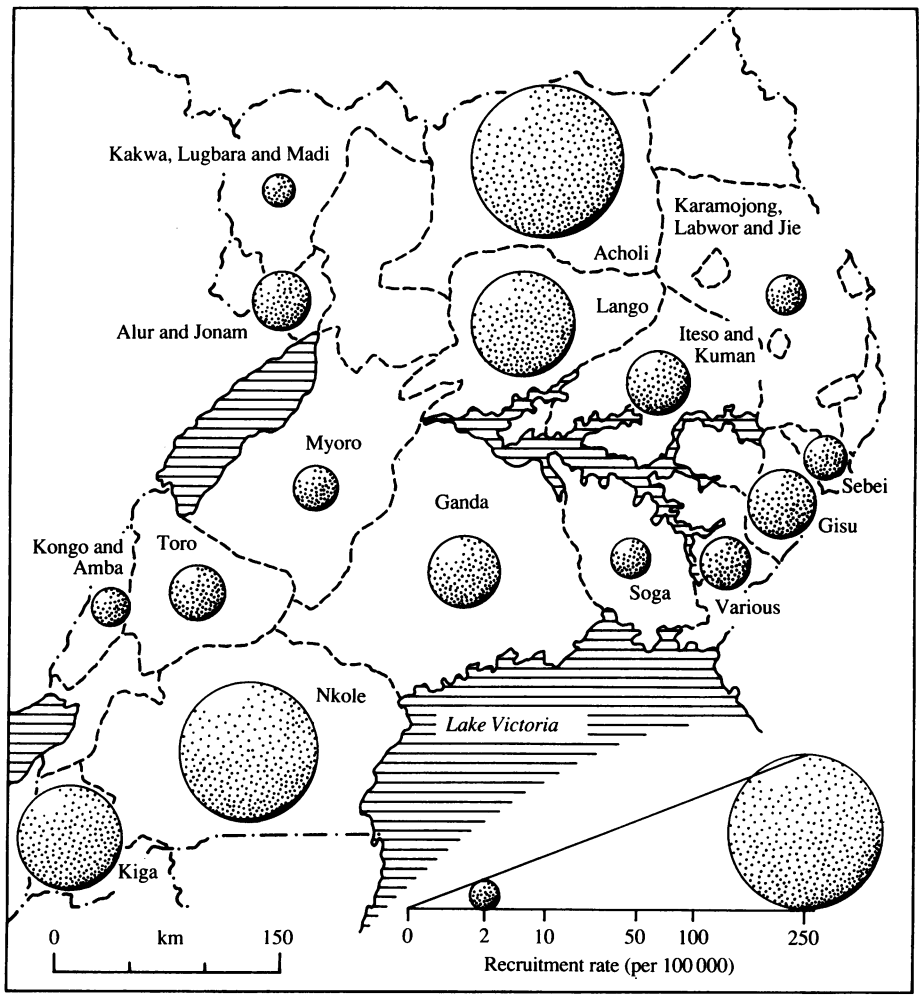

Figure 4. UNLA recruitment rates in Uganda by district. Levels are proportional to the volumes of the spheres.

RESULTS

The critical regression results are summarized in Table 2. The intercept and slope coefficients of each model are shown, along with their associated t-statistics in parentheses. The coefficients of determination, R^2, and their F ratios are also given. Statistically significant t and F values, using the $P = 0{\cdot}05$ level (one-tailed test), are indicated by an asterisk.

Regression models 1–6 in Table 2 relate components associated with the truck town hypothesis to the apparent disease pattern; models 1–4 examine the impact of urbanization while models 5–6 explore the association between AIDS reporting rates and district accessibility to the main highways. A statistically insignificant overall explanation (as judged by the F ratio) of the district to district variability in AIDS incidence rates was observed with all these models: accounted variability ranges from 0·2 to 17%. Table 2 shows a similar result for regression model 7, which relates AIDS incidence to the migrant labour component.

Table 2. *Regression results for tests of hypotheses to explain geographical patterns of AIDS in Uganda*

Model	Intercept (t-statistic in parentheses)	Slope coefficients for independent variables (t-statistic in parentheses)										R^2 (F ratio)
		x_1	x_2	x_3	x_4	x_5	x_6	x_7	x_8	x_9	x_{10}	x_{11}	
1	31·80	4·80											0·063
	(1·71)	(1·43)											(2·03)
2	48·60		0·04										0·002
	(19·94)*		(1·10)										(0·77)
3	30·60			0·001									0·075
	(1·62)			(1·56)									(2·44)
4	59·40				−2·50								0·002
	(1·79)*				(−0·23)								(0·05)
5	50·30					−0·015	0·02						0·004
	(1·72)*					(−0·12)	(0·25)						(0·06)
6	10·30					4·63	0·17	0·08	−0·33				0·174
	(0·15)					(0·28)	(0·77)	(0·51)	(−1·80)*				(1·09)
7	31·40									23·80	42·3		0·057
	(1·46)									(0·84)	(1·31)		(0·88)
8	21·90											0·60	0·430
	(1·96)*											(4·79)*	(22·95)*
9†	19·90											0·68	0·410
	(1·94)*											(4·39)*	(19·29)*

* Significant at $P = 0·05$ level (one-tailed test).
† Gulu and Kitgum omitted.

Independent variables
x_1 = percentage of district population living in urban centres.
x_2 = 1980 population density per square mile (see Fig. 2).
x_3 = absolute urban population size.
x_4 = 1969–80 annual percentage population growth rate.
x_5 = distance from road A (see text and Fig. 2).
x_6 = distance from road B (see text and Fig. 2).
x_7 = distance from road C (see text and Fig. 2).
x_8 = distance from road D (see text and Fig. 2).
x_9 = regions of labour out-migration (see Fig. 3).
x_{10} = regions of labour in-migration (see Fig. 3).
x_{11} = November 1979 UNLA recruitment rates per 100000 population (see Fig. 4).

In contrast, models 8 and 9 in Table 2, which relate UNLA ethnic recruitment rates to AIDS rates, display a markedly different picture. In model 8 there is a highly significant positive relationship between the two variables ($t = 4{\cdot}79$, $P < 0{\cdot}0005$), with 43% ($= 100R^2$) of the variability in AIDS incidence rates being explained by the recruitment patterns ($F = 22{\cdot}95$, $P < 0.01$). In model 9, the two northern districts of Gulu and Kitgum which, with high levels of Acholi recruitment, serve as outliers influencing the parameters of model 8, are omitted. As Table 2 shows, however, omission of these two districts has little impact; a highly significant overall explanation is maintained ($R^2 = 0{\cdot}41$; $F = 19{\cdot}29$, $P < 0{\cdot}01$).

Although the results are not tabulated, multivariate regressions using recruitment rates (x_{11}) with combinations of the variables x_1–x_{10} were also tried; in all cases, x_{11} made a significant contribution to R^2 even in the presence of the other variables.

Application of a test for spatial autocorrelation on the residuals of model 8 yields a standard normal score (z score) of $-0{\cdot}26$ with a probability of chance occurrence of 40 times in 100. The spatial randomness in the residuals implies that the recruitment rate variable accounts for all the systematic spatial pattern in district AIDS case incidence rates.

DISCUSSION

Currently available epidemiological data suggest that some of the most extreme HIV epidemics in the world occur in some countries of Central Africa [3, 8, 9]. As noted earlier, in Uganda alone, official 1990 estimates placed the number of HIV infections in the country at one million, or 6% of the population [3]. An understanding of the factors which have contributed to the evolution of the Ugandan AIDS epidemic is of critical importance if the repetition of such a phenomenon is to be prevented in other countries.

Two principal hypotheses, the truck town hypothesis and the migrant labour hypothesis, have gained currency as possible explanations for the current spatial pattern of reported clinical AIDS witnessed in Uganda [18–20]. However, the analysis presented in Table 2 reveals that both hypotheses fail fully to explain the district to district variability in AIDS incidence. This finding may be a function of the quality of Ugandan AIDS surveillance data. For example, recent serological analysis indicates that, at the present time at least, major roads are acting as principal corridors for the spread of HIV in Central Africa in general [25] and Uganda in particular [13, 17, 26]. However, given the observed long incubation period between primary HIV infection and the development of clinical disease, apparent geographical patterns of AIDS currently seen in Uganda may reflect factors contributing to an earlier historical spread of HIV.

Table 2 shows a highly significant positive relationship between indicators of the ethnic base of the Ugandan National Liberation Army and the currently reported spatial pattern of clinical AIDS in Uganda. The observed relationship is remarkable given the uncertain quality of AIDS surveillance data from the country [11]. The variability not accounted for by model 8 may be evidence of further, as yet unreported, factors which have influenced the evolution of AIDS

patterns in the country. However, since omission of a significant explanatory variable in regressions using geographically-located data usually gives rise to spatially autocorrelated regression residuals [24, p. 197], the lack of autocorrelation found in the residuals of model 8 implies that the residual variability in AIDS rates is probably associated with data quality factors rather than unknown causal mechanisms.

The historical link between servicemen, prostitutes and the spread of sexually transmitted diseases is well documented, both in the West [5] and in tropical Africa [6]. But, with the appearance of AIDS, most published studies regarding HIV infection in soldiers have concentrated upon the US military where infection has been associated with the classic Western risk factor of sex between men [27, 28]. In contrast, little attention has been paid to the link between soldiers, civil war and the spread of HIV in regions like Central Africa which display endemic heterosexually-acquired HIV infection.

The association reported here between UNLA and the spatial distribution of documented AIDS cases is supported by observations regarding soldiers, prostitute contact and war in the spread of HIV in Sudan, the country abutting the northern border of Uganda [29, 30]. The findings also accord with reports of high rates of HIV infection in soldiers in Angola [31] and Zimbabwe [32], and with the correlation between war-associated population displacement and HIV infection in Mozambique [33]. Also noteworthy is the impact of recent hostilities on the spread of HIV in Central America [34, 35], the evidence of HIV infection among former European soldiers deployed during the wars of Portuguese Africa [36–38] and HIV infection in Cuban soldiers returning from Angola [39]. Within Uganda itself, the findings are supported both by formal reports of high HIV prevalences among northern soldiers [40] and by anecdotal evidence [41].

We therefore tentatively suggest that the apparent geographical pattern of clinical AIDS in Uganda partially reflects the diffusion of HIV associated with civil war during the first 6 years of the post-Amin period. In particular, the association between 1979 UNLA recruitment patterns and the spatial distribution of documented AIDS cases in the mid-to-late 1980s is in accord with an estimated mean incubation period of 8–10 years for HIV [42] and suggests the need for cognizance of historical factors that may have influenced the evolution of spatial patterns of severe HIV disease. The bipolar pattern underlying reported clinical AIDS cases in Uganda may be explained by the deployment of soldiers, predominantly of northern origin, in southern districts of the country where HIV was spreading unnoticed in the late 1970s and early 1980s. Carriage of HIV from the south to the north by soldiers accords with evidence regarding the time of appearance of HIV in these regions [40, 43] and explains why other regions of Uganda remain, apparently, relatively unaffected.

However, despite the weight of evidence, a cautionary reminder must be sounded because of uncertainties over the quality of data. First, the completeness of Ugandan AIDS data remains unassessed due to the passive nature of the country's surveillance system [11]; geographical variations in both the provision and capabilities of healthcare services may generate unknown spatial variations in the completeness of AIDS data. Second, data availability compelled the use of November 1979 UNLA recruitment patterns as an indicator of the ethnic base of

the army. Throughout the early 1980s, the ethnic base of UNLA was highly fluid; composition was closely associated with the ethnic origin of leading politicians and military officials. In particular, in late 1979, the Acholi, Lango and Nkole were dominant (Fig. 4). Although information regarding the entire ethnic composition of UNLA is not available, geopolitical expedience and the defection of many Nkole into the National Resistance Army led to increased dominance of the Acholi from the early to mid-1980s [22]. Such time-related data uncertainties may account for some of the district-to-district variability in AIDS incidences unexplained by model 8 (Table 2).

Despite these caveats, as far as we are aware this is the first documented statistical evidence regarding the association between soldiers and the documented spatial pattern of clinical AIDS in any country. Similar patterns may ultimately be recognized in other African countries which have recently experienced internal unrest.

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