The Epidemic of Visceral Leishmaniasis in Western Upper Nile, Southern Sudan: Course and Impact from 1984 to 1994

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Background. Although endemic in parts of southern Sudan, visceral leishmaniasis (VL) had not been reported in Western Upper Nile (WUN) until an epidemic was confirmed in 1989. A combination of circumstances created conditions for transmission among a population of mainly Nuer and Dinka people who had no immunity. The civil war which restarted in 1983 has been a major contributing cause and continues to hinder provision of treatment, data collection and control measures. *Methods.* Since the first of three clinics to treat VL was established in WUN in 1989, data on the epidemic and mortality have been collected in seven retrospective surveys of villages and among patients. Adults were Interviewed about surviving family members and those who had died since the epidemic came. Survey death rates are used here to estimate mortality from VL and 'excess mortality' above expected levels.

Results. The surveys found high mortality at all ages and suggest an overall death rate of 38–57% since the epidemic started in 1984, and up to 70% in the most affected areas. Both methods of estimation suggest that around 100 000 deaths, among about 280 000 people in the epidemic area, might be attributable to VL.

Conclusions. This continuing epidemic has shown that VL can cause high mortality in an outbreak with astonishingly high infection rates. Population movement has been a major factor in transmission and poor nutritional status has probably contributed to the risk of clinical infection. Although over 17 000 people have been successfully treated for VL at the clinics in WUN, the disease is likely to become endemic there.

Keywords: visceral leishmaniasis, kala azar, Sudan, mortality, epidemic

It is late in the afternoon and two brothers are being registered at the visceral leishmaniasis clinic in Duar, southern Sudan. They are febrile and severely marasmic—just hot skin and bones with the huge spleen and dull eyes so common in kala azar patients. They are unable to say who their subchief is and in frustration the staff ask for their mother. Finally, a man tells them the boys were found that morning on a path leading to the clinic, sitting beside a mosquito net that covered their dead mother. Her other children and husband had died already that year, so in desperation she had set out for the new clinic in Duar to see if there was help there.

The epidemic of visceral leishmaniasis (VL) in Western Upper Nile (WUN), southern Sudan has devastated the family and tribal life of the Nuer and Dinka people who are the main inhabitants. Kala azar, the syndrome of fever, wasting, and enlarged spleen or lymph glands resulting from VL, is typically fatal unless treated. It has been reported in southern Sudan since 1904 and is endemic east of the White Nile above the Sobat River along the border with Ethiopia as far north as Kassala, and in a few smaller areas (Figure 1).¹ Until recently WUN was considered free of VL, but since 1984 there has been a severe and tragic epidemic. In this paper we outline the course of the epidemic and consider quantitative evidence of its impact in terms of the people who sought treatment or reportedly died from the disease. First, we give some background about the circumstances in which the epidemic occurred and the treatment centres that were established.

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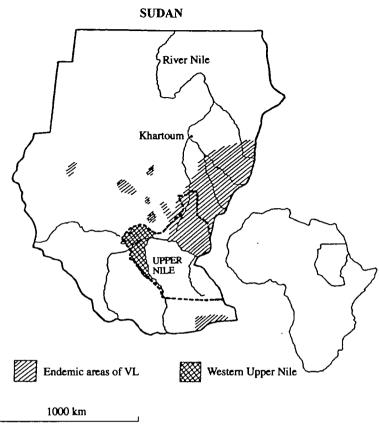


FIGURE 1 Endemic areas of visceral leishmaniasis in the Sudan

People, Environment, Civil War and Transmission

Western Upper Nile is situated in an open, flat, grassy plain which is a mixture of savannah and permanent swamp. There is a long hot dry season followed by a long wet season when much of the plain floods from about May-October. The people are Nilotic, mainly from the Nuer tribe to the south of the Bahr el Ghazal river and Dinka to the north in Panarou. Their livelihood is cattle-rearing, supplemented by cultivation of sorghum and maize and sometimes fishing. Traditionally people cultivate near their villages in the wet season and move with their cattle to find water in the dry season. They walk back and forth between cattle camps near the water and the relatively dry areas near their 'permanent' homes and also go in search of food in this 'hunger season'. Traditionally people walk in the cool of the night through the forests, exactly the time and place that sandflies which transmit VL are biting. The vector in WUN, as in the endemic area of Paloich-Malakal in eastern Upper Nile,¹ has been confirmed as Phlebotomus orientalis. In field studies in 1991, a high

proportion of specimens were found to be infected with Leishmania donavani indicating intense transmission.² P. orientalis was found from February to June, mainly in the Acacia/Balanites forests rather than in dwellings, and biting occurred all night.³ The main transmission season for VL lasts from March through May, which coincides with dry season movement of people through the forest. Many areas of WUN (particularly Jikany, Jegai, north Panarou and south Leek [Figure 2]) are wellwooded with various species of Acacia and some Balanites aegyptica, the known habitats of P. orientalis.⁴ The forests probably regenerated following destruction by unusual floods in 1960 to 1964 possibly caused by excessive overflow from the Owen Falls Dam at Lake Victoria, creating conditions suitable for P. orientalis and hence for transmission of VL.5

In 1983 the longstanding conflict between the culturally independent and mainly Nilotic people of the South and the government forces of the mixed Arab-African North restarted. It has been a civil war of low intensity, but has completely disrupted the little

WESTERN UPPER NILE

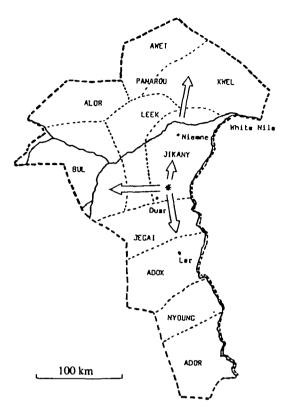


FIGURE 2 Spread of visceral leishmaniasis in Western Upper Nile

infrastructure that existed and put a stop to health care activities and commerce. Because of the war there has been additional movement of people, including soldiers, who could have been infected when passing through an endemic area and carried the parasite to Jikany in WUN. The inhabitants of that area, mainly people of the Nuer tribe, would mostly have had no immunity to VL, which accounts for the high incidence among all age groups.⁶ Lack of immunity was also a contributing factor in the major outbreak of VL in the southern Fung district of Blue Nile region in 1956 when there was an influx of people from an endemic area to the north following independence.⁷ Since the introduction of VL into Jikany, seasonal migration and movement to escape the fighting and to search for food or fertile land has probably increased the rate of transmission and facilitated the spread of the epidemic within WUN, and beyond.⁸ Agriculture and cattle rearing have been disrupted by the war, resulting in more persistent malnutrition which has probably

contributed to a higher conversion rate to clinical disease on contracting *L. donavani* and hence to high mortality in the absence of treatment.⁹

After the civil war restarted there was no treatment available for any medical condition in most of WUN until a clinic and feeding programme was established in Ler in 1988. Local people reported a 'killing disease' in an inaccessible area to the north, initially thought to be an outbreak of typhoid. However, later that year, VL was identified among displaced people from the Bentiu area of WUN who were living in Khartoum.¹⁰ Subsequently, in February 1989, serological tests on blood samples from patients in Ler indicated leishmaniasis and in May 1989 a cross-sectional survey in the village of Kuernyang, 40 km to the north of Ler, found 23.3% of the inhabitants were serologically positive.^{11,12} A special treatment centre for VL was immediately set up in Ler and a second clinic was planned for Duar, 80 km to the north, bud did not open until July 1990 because of poor security. In July 1993 a third VL treatment centre was set up in Niemne in north Jikany, to treat local people and those from Panarou who were able to cross the Bahr el Ghazal. Expatriate staff had to be evacuated in January 1994, but treatment by Nuer health auxiliaries has continued, with periodic supervision from expatriate staff still based in Duar and Ler. This report on the course of the epidemic and mortality in WUN is based on data collected in the period 1989-1994 by those working in these clinics.

METHODS

The circumstances of the war have precluded a comprehensive epidemiological survey of the whole area affected by VL in WUN. However, the data that have been collected compel us to report our impression of the impact of this epidemic. The main sources of data are interviews with local informants, and surveys of villages and among some of the more than 19 000 patients treated at the clinics. The clinical record cards of people treated for VL are updated daily and registration books and monthly reports also contain data on admissions, discharges and deaths during treatment. All the records have been kept under extremely difficult circumstances: expatriate doctors and nurses live in tents, there is no shelter for patients who usually sleep and receive treatment outside, and at times the patient census has been over 1500 (August-November 1991). Access to the clinics is by light aircraft only and the programme has frequently been disrupted by poor security and temporary evacuation of expatriate staff because of fighting. Since the VL clinics were first established many expatriate staff have been involved in recording data, but one of the authors (JS) has been working almost continuously on the programme since 1989, facilitating continuity in record keeping, interviews with local leaders and health auxiliaries, and the conduct of surveys.

There have been seven retrospective mortality surveys conducted in households or among patients in WUN between 1990 and 1994. In all but the last survey, a household consisted of a 'luak complex' (a barn [luak] and some smaller huts [tukuls]), the 'permanent' home of an extended family comprising the head of household and his associated relatives. An adult was asked what had happened to members of the household since the epidemic came and for the cause of any deaths. When death was attributed to VL people were able to distinguish signs and symptoms of kala azar quite well.

The first survey in 1990 used cluster sampling methods, blood tests and interviews to investigate seroprevalence and recent mortality in relation to VL in south Jikany and Leek. Four villages and a cattle camp in Jikany and two villages in the more densely populated Leek were surveyed. These villages probably each contained 400–1000 people before the epidemic and were chosen because of security and accessibility from Duar. Twenty-two clusters were selected which included at least 25 living people (for serological testing). The number of clusters in each village was approximately proportional to population size. The age and sex of living household members was recorded and also the number who were absent or had died since VL came.

The second survey in 1991 was intended as a cohort study of serology, immunology and mortality in relation to VL, but the follow-up in 1993–1994 was interrupted by fighting. For the initial survey, two villages were chosen, each with a population of about 200–300 people before the epidemic: Dejoul in Jegai near the epicentre of the epidemic and Kwangro in north Jikany where the epidemic had just arrived.¹³ Interviews to obtain age and sex of family members and reported deaths from VL, serological and skin tests, and examinations were conducted among all families in these two villages.

The third survey was conducted in January 1993 during an exploratory mission to two accessible villages in north Panarou, Weregnot and Mathieng. The survey team walked over a huge open grave, past recently burnt huts to the forest where people had fled from their homes. Poor security and limited time affected the conduct and quality of interviews, but the death and disease observed leant credibility to the tragic family stories. Interviews were conducted with most families from these villages where about 350 people reportedly lived before the epidemic. The fourth survey was conducted by a locally trained VL health worker who walked through the relatively densely populated south Bul district in 1993. In the seven villages he came to, he conducted interviews in every household and examined all those present. He recorded the number of family members who were alive and present, alive and absent, or had died since VL came. This provided data on over 2300 people who were alive when the epidemic arrived.

In the fifth survey, complete family histories were obtained in interviews with 100 out of 131 consecutive attenders at the VL clinics in Niemne and Duar in 1993. Interviewers questioned thoroughly to obtain the number of family members who had died or moved away since the epidemic came and the age and sex of those still living in the household. This provided data on over 1500 relatives, mainly from Panarou and north Jikany.

The sixth survey was conducted in April 1994 in the village of Gon in Jegai, with an estimated population of 500–600 people before the epidemic, chosen because it was only 10 km west of Duar. Interviews in all house-holds in one half of the village provided information on 262 people. Respondents were asked for the name, age and sex of all family members who were alive and present, absent, or had died since VL came. They were also asked about signs and symptoms of disease and when the person died.

In the seventh survey, the same questions were asked in interviews with half the Dinka patients from south Panarou at the treatment centre in Duar in April 1994. This provided data on a cohort of over 800 named family members alive when the epidemic came to Panarou. A different concept of extended family was used based on the culture of the Dinka people: a man's wives and children, the wives and children of his father's other sons, the wives and children of his father's brothers, and the wives and children of his father's brother's sons. This standardized definition helped avoid double counting which could have occurred in the previous surveys because 'luak household' composition may change, particularly when the head dies or a woman marries.

The results of the seven surveys, together with data from the 1983 census, were used to estimate mortality in the epidemic in WUN as a whole. The population of each district is not available from the census, but relative sizes were estimated after discussions with local leaders and health workers. As a check, the proportion of the population estimated to be in Panarou, Jikany, Leek and Bul districts (59.0%) was reasonably comparable with that for the equivalent districts, 412–414, available from the census (62.6%).¹⁴ The population in each district at the time that VL came was calculated assuming a growth rate of 2.9% per year estimated for the whole of Sudan for 1985–1990.¹⁵ The survey death rates may underestimate mortality as some families were completely killed, or had only a few survivors who fled and could not be interviewed. On the assumption that survey estimates were reasonably representative of the whole district, the range of death rates found was applied to the population before VL came, to estimate deaths from VL in each district. In view of uncertainty about the attribution of deaths to VL by relatives and high 'underlying mortality' in southern Sudan, the impact of the epidemic was also assessed in terms of 'excess deaths' since the epidemic started. The number of expected deaths was based on the crude death rate of 16 per 1000 per year, estimated for the whole of Sudan for 1985-1990.¹⁵ This was subtracted from the total number of deaths calculated by using the overall death rates from the surveys in each district, giving an estimate of 'excess deaths'. Because of the extrapolation of death rates from surveys to the whole population of a district, the estimates of mortality in WUN should be treated with caution, but they give an impression of the impact of the epidemic.

RESULTS

The Course of the Epidemic in WUN

Local informants reported that the first deaths in the VL epidemic in WUN occurred in 1984 in the area of Jikany just to the north-east of Duar. The course of the epidemic from 1984-1994 is outlined in Table 1. The epidemic peak in south Jikany and Jegai occurred in 1988-1989, coinciding with the famine which was widespread in south Sudan in these years. Incidence peaked in east Bul and south Leek in 1990, and in Adok district in 1990-1991 when the drought forced people to walk in search of food through the forests of Jegai. Incidence increased in north Leek, north Jikany and east Bul in 1991 and peaked in Panarou, the only Dinka area affected, in 1991-1992. In 1994, there was a resurgence in numbers of VL patients from three districts near Duar; Jikany, Jegai and Bul. This was probably caused by relocation of people into the acacia forest and when a market opened, to which people travelled through the forest near Duar when coming from the dry season cattle camps.

Treatment became available in Ler in mid-1989. This coincided with the peak in incidence in south Jikany and Jegai. Many people lived from 2–10 days walk away and were not able to reach treatment in time, did not know that it was available, or did not have any relatives left to assist them on the journey and during

TABLE 1 Timetable of events in the visceral leishmaniasis epidemic in WUN, southern Sudan

1984	First cases in south Jikany District (e.g. villages of Pak and Kuec, near Duar)			
1985	Spread: West in Leek District : South into Jegaı District			
1986	: To south Jegai District : To area north of Ler in Adok District			
1987	: To east Bul District : To Panarou north of Bahr el Ghazal			
1988-89	Peak mortality in south Jikany and Jegai Treatment available in Ler from mid-1989			
1990	Treatment available in Duar from mid-1990 Access to treatment difficult from Bul Patients arriving in Duar from Panarou			
1991	Peak mortality in north Jikany and north Leek People from Panarou no longer reaching Duar			
1991-92	Peak mortality in Panarou (Dinka people) Access still not possible from Panarou (fighting)			
1993	Epidemic mainly in Panarou and north Jikany Treatment available in Niemne from mid-1993			
1994	Resurgence of epidemic from July Many patients from Jikany, Leek and Jegai			

treatment. Tragically then, the majority of deaths there occurred before treatment became available. Treatment became available in Duar in mid-1990. In 1990–1991 more patients came from the Dinka area of Panarou than any other area, despite a hazardous walk of up to 15 days or more. Unfortunately, open warfare prevented the journey in the next year, 1991–1992, when incidence peaked in Panarou. Virtually no-one from that area was treated at that time, so the majority of deaths in Panarou occurred because treatment was not accessible.

The number of admissions for treatment in each year is summarized in Figure 3. The peak was in late 1991, with over 1500 patients admitted in each of the months September, October and November, mostly from Leek, Jikany, Jegai and Bul. A clear and consistent seasonal peak in admissions for VL has been observed in WUN despite the influence of other factors such as accessibility of treatment. In each year between 1989–1994, the number of admissions increased in the period July– November, 2–6 months after the end of the transmission season. In 1994, the seasonal increase in admissions in July also reflected a resurgence of the epidemic and at the end of the year there were again over 500 patients under treatment for VL in Duar, mainly from Jegai, Jikany, Leek and Panarou.

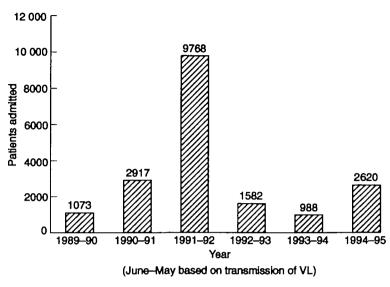


FIGURE 3 Patient admissions by year at VL clinics in WUN

Mortality from Visceral Leishmaniasis in the Population of WUN

The surveys have indicated very high mortality from VL in WUN. The death rates found in different districts are summarized in Table 2. Reported death rates were highest in the areas near the epicentre (south Jikany and Jegai) and in the area denied access to treatment (Panarou). Mortality is lower in areas where the peak in the epidemic arrived after accessible treatment was established (Leek and north Jikany). There was also a lower death rate in Bul, partly because the epidemic peaked after treatment was available, but also because less of the area is covered with acacia forest.

Mortality and serological data from the first survey in May 1990 tend to confirm the above account of the spread of the epidemic. The death rate found in south Jikany was 50.6% (n = 810 before VL), compared with 26.3% (n = 517 before VL) in Leek. Blood samples revealed seroreactivity for leishmania antibodies in about 30% (n = 236) of those normally resident in south Jikany, compared with only 5% (n = 400) of those from Leek. The higher prevalence of infection and higher mortality from VL in south Jikany reflect the earlier arrival of the epidemic there and the longer exposure compared with Leek where the epidemic had only recently started.

The survey begun in May-June 1991 again reflects the course of the epidemic, indicating its progression and the effect of treatment availability. Dejoul in Jegai was close to the early epicentre of the epidemic in an area severely affected by famine in 1988–1989. The death rate since VL arrived was reported at 64.7% (n = 258 before VL). In Kwangro in north Jikany, where the epidemic arrived later, the reported death rate was lower at 38.7% (n = 315 before VL). A follow-up survey of the cohort in 1993 found much the same death rate (39.1%), but the follow-up in 1994 found a further 2.2% (7) of the original cohort of 315 had died from VL. It was also found that 40 (20.7%) of the 193 alive in 1991 had since been treated for VL and were still alive. In total, 169 people were reportedly affected by VL (129 reported dead and 40 treated), which was 53.7% of the original cohort. This is more consistent with the death rates found in south Jikany and Jegai where the epidemic arrived earlier and peaked before treatment was available.

Table 3 summarizes the results of the surveys conducted in 1994 which primarily focused on mortality and provided additional information. The reported peak periods for mortality are consistent with the above account from local informants. In Gon, Jegai, most deaths reportedly occurred in the years 1985-1989, in the early stages of the epidemic before treatment became available in Duar. In Kwel, Panarou, most deaths were reportedly in 1991-1993, when access to treatment was precluded by fighting. The results are also consistent with the other surveys in showing relatively high death rates near the epicentre (Gon survey) and where treatment was not available (Kwel survey). In both surveys, high death rates were found among all age and sex groups, confirming an exceptional pattern of mortality since the VL epidemic started.

District		Number, type and location of survey	Year	Period of epidemic covered	Family members when VL came	Number who died from VL (% died)	Number who died from all causes (% died)
Jikany	(South)	[1] Cluster sample 4 villages/cattle camp near Duar	1990	1985–90	810	410 (50 6%)	na
	(North)	[2] All families Kwangro near Duar	1991	198591	315	122 (38.7%)	na
	(North)	[5] All patients from Jikany	1993	1985–93	110	43 (39.1%)	76 (69 .1%)
Jegai		[2] All families Dejoul near Duar	1991	198591	258	167 (64.7%)	na
		[6] Half of village Gon near Duar	1994	1985– 94	262	132 (50.4%)	134 (51.1%)
Panarou	(North)	[5] All patients from Panarou	1993	1987–93	1093	443 (40.5%)	753 (68.9%)
	(North)	[3] All families villages [.] Mathiang and Weregnot ^a	1993	1987–93	358	na	251 (70.1%)
	(South)	[7] Half patients in Duar from Kwel, Panarou	1994	1987–94	814	457 (56.1%)	458 (56.3%)
Leek		[1] Cluster sample two villages near Duar	1990	1985–90	517	136 (26.3%)	na
Bul		[4] All families in 7 villages	1993	1987–93	2353	711 (30.2%)	па

TABLE 2 Surveys of mortality in the visceral leishmaniasis epidemic in WUN

¹ Joint MSF-Holland/United Nations mission. In 1993 a World Food Programme/United Nations Childrens Fund mission to Nyarweng in Panarou interviewed 25 families who reported on 406 members of whom 45.3% had died in the last year from VL and other causes.

The survey results and information from local informants indicate that mortality was higher in Jikany, Jegai and Panarou, where most surveys were conducted. In Jikany, (pop: 25 000 before VL) where the epidemic started, the death rates from VL of 39-51% may be underestimates because many families were completely killed. For example, a Nuer health auxiliary was one of only four survivors from Duar in Jikany, which had about 1000 inhabitants before the epidemic arrived. Also, deserted villages were observed in 1991 when the survey team walked from Duar to Kwangro in Jikany, and there are reports of many other such villages. The surveys in Jegai (pop: 25 000) suggest 50-65% died from VL. Surveys of villages and patients from Panarou (pop: 60 000) suggest 40-56% died from the disease, despite the late arrival of the epidemic in 1987. One survey in Leek (pop: 48 000) and the large survey in Bul (pop: 60 000), suggest that about 30% had died from VL since 1985. According to local informants, the death rate may have been similar among people from Adok (pop: 60 000) after migration to the epidemic area of Leek.

The total population of WUN was found to be about 300 000 at the 1983 census,¹⁴ and possibly 280 000 people lived in the affected districts when VL arrived. On the basis of the population estimates and the range of survey death rates from VL in each district, 95-112 000 people may have died from VL in WUN since 1984 (Table 4). This high mortality attributed to one disease should be seen in the context of high 'underlying mortality'. About 15% of the people alive in WUN when the epidemic started in 1984 would have died anyway by 1994, assuming a death rate of 16 per 1000 per year. However, just in the periods covered by the surveys, between 38% and 57% of the population reportedly died, and up to 70% in the most affected areas. On the basis of the overall death rates found in the surveys in each district and population figures extrapolated from the 1983 census, 80-136 000 people who might have been expected to live, have died since 1984

Kwel survey (patients in Duar)				
Age in 1987	Females	Males	Total	
0-4	70.6% (24/34)	40.6% (26/64)	51.0% (50/98)	
5-14	73.3% (55/75)	62.8% (59/94)	67.5% (114/169) 47.8% (220/460)	
15-44	42.9% (93/217)	52.3% (127/243)		
45+	25.0% (1/4)	88.0% (73/83)	85.1% (74/87)	
All alive in 1987	52.4% (173/330)	58.9% (285/484)	56 3% (458/814)	
Born since 1987	46.7% (21/45)	27.8% (15/54)	36.4% (36/99)	
Gon survey (residents)		Death rates by age – Gon, Jegai % of family members who died		
Age in 1985	Females	Males	Total	
0-4	59.1% (13/22)	59.3% (16/27)	59.2% (29/49)	
5-14	52.6% (30/57)	50.0% (20/40)	51.5% (50/97)	
15-44	50.9% (29/57)	36.8% (14/38)	45.3% (43/95)	
45+	75.0% (6/8)	46.2% (6/13)	57.1% (12/21)	
All alive in 1985	54.2% (78/144)	47 5% (56/118)	51 1% (134/262)	
Born since 1985	17.5% (7/40)	33.3% (17/51)	26.4% (24/91)	
Year of death	Kwel survey	Gon survey		
(approximate)	Deaths among family	Deaths among family		
	of patients from Kwel	of residents in Gon		
	(% in each year)	(% in each year)		
1985-86	_	21 (13.4%)		
1987	_	11 (7.0%)		
1988	57 (11.5%)	23 (14.6%)		
1989	29 (5.9%)	55 (35.0%)		
1990	44 (8.9%)	10 (6.4%)		
1991	105 (21.3%)	7 (4.5%)		
1992	125 (25.3%)	9 (5.7%)		
1993	112 (22.7%)	11 (7.0%)		
1994	22 (4.5%)	10 (6.4%)		
Total	494 (100.0%)	157 ^a (100.0%)		

TABLE 3 Surveys of mortality by age and year of death in the visceral leishmaniasis epidemic in WUN

* Year of death was not recorded for one of the 158 deaths.

(Table 5). Allowing for extra deaths from VL among families with no survivors, and deaths since the surveys in areas with no access to treatment, around 100 000 people have probably died from VL in WUN.

DISCUSSION

In the period 1984–1994, VL has undoubtedly killed thousands of people in WUN who would not otherwise have died, as well as many who would have died from

other causes anyway. The first estimate of mortality made in 1989 put the death toll from VL at 20–30 000 Nuer people, but this was only based on local reports and there was no access to the area in Jikany where the epidemic had started. Thousands of Nuer have died since then from VL and at least half the Dinka people of Panarou. The problem of quantifying the impact of the epidemic on the people of WUN, on the basis of deaths reportedly due to VL, has to some extent been overcome by estimating 'excess mortality'. Many

District	Estimated distribution of population by District ^a	Estimated population at 1983 census	Year when VL came	Estimated population when VL came ^c	Survey estimates of death rate from VL	Estimated deaths from VL since epidemic started
Panarou	0.18	54 000	(1987)	60 542	40-56%	24 217-33 904
Jikany	0.08	24 000	(1985)	25 412	39-51%	9911-12 960
Leek	0.15	45 000	(1985)	47 648	26%	12 388
Bul	0.18	54 000	(1987)	60 542	30%	18 163
Jegai	0.08	24 000	(1985)	25 412	50-65%	12 706-16 518
Adok	0.18	54 000	(1987)	60 542	30%	18 163
Nyoung/Ador	0.15	45 000	Unaffected	-	_	_
Total WUN	1.00	300 000 ^b		280 098	_	95 548-112 096

TABLE 4 Estimate of deaths from visceral leishmaniasis by district in WUN

^a Based on discussions with local leaders, a judge, and 5 years' experience of working in the area.

^b Population of WUN at 1983 census: 297 761.

^c Assuming a growth rate of 2.9% per year: United Nations estimate for Sudan, 1985-1990.¹⁵

TABLE 5 Estimate of 'excess deaths' during visceral leishmaniasis epidemic in WUN

District	Year when VL came	Estimated population when VL came	Expected deaths among cohort in the period covered by surveys ^a	Estimated total death rate based on surveys ^b	Estimated deaths from all causes since VL came based on surveys	Estimated 'excess' deaths among cohort since VL came
Panarou	(1987)	60 542	5584-6464	56-70%	33 904-42 379	27 440-36 795
Jikany	(1985)	25 412	1969-3076	51 <i>-</i> 69%	12 960-17 534	9884-15 565
Leek	(1985)	47 648	3692	26-45%	12 388-21 442	8696-17 750
Bul	(1987)	60 542	5584	30-52%	18 163-31 482	12 579-25 898
Jegai	(1985)	25 412	2344-3434	5165%	12 960-16 518	9526-14 174
Adok	(1987)	60 542	5584	30-52%	18 163-31 482	12 579-25 898
Total		280 098	24 757-27 834	_	108 538-160 837	80 704–136 080

* Estimates assume cohort was reduced each year by 16 deaths per 1000 (crude death rate for Sudan as a whole estimated by United Nations)¹⁵ in the years covered by the surveys which gave the low and high estimates of total death rates (Table 2).

^b No overall death rate available for Leek, Bul and Adok. Low estimate assumes VL death rate was an overall death rate, high estimate assumes it was 58% of the overall death rate (as in survey [5]). Low estimate for Jikany assumes VL death rate in survey [1] was an overall death rate.

assumptions had to be made in the absence of recent data on the 'underlying' death rate in the area and because of poor data on the population affected in each district. Furthermore, the mortality estimates are subject to the limitations of retrospective surveys involving long recall periods and the extrapolation of death rates to whole districts. Even so, there is a consistency between the results from the surveys, which show higher death rates in the areas around the epicentre, where incidence peaked before treatment became available, and in areas with restricted access to treatment. The overall picture that emerges from the surveys and interviews with key informants is that this epidemic of visceral leishmaniasis has been a human tragedy on a vast scale and a social and demographic disaster for the agro-pastoralist people of WUN. Undoubtedly, the epidemic has inflicted great suffering and damage to family and community life. It is comparable in impact, if not in scale, with notorious epidemics in the past such as the 'Black Death' of 1346–1350, when whole villages died of plague and an estimated one-third of the population of Europe. Similar destruction was caused among the non-immune Amerindian population when smallpox was introduced from Spain in the early 16th century.¹⁶ The age-old cycle of war, famine and infectious disease is still part of the way of life among the threatened cultures of southern Sudan. Modern curative medicine has provided some relief for the people and probably over 17 000 lives have been saved at the VL treatment centres in WUN,¹⁷ but this provides no long-term solution to a disease that will become endemic if the civil war and associated conditions continue.

In the period 1984–1994, the epidemic in WUN has shown that visceral leishmaniasis can cause high mortality in outbreaks with astonishingly high infection rates. Population movement in response to war and food shortages has been a major factor in transmission and poor nutritional status, particularly in the dry season, probably increases the risk of clinical disease for those infected. Movement of people through the forest to trade in a new market may have contributed to the resurgence of the epidemic in 1994 in Jikany where it first started. The main focus of VL in southern Sudan continues to be around the original epicentre of the epidemic in WUN. However, a separate outbreak occurred in eastern Upper Nile in 1994, when many people moved through Acacia/Balanites forests to seek food and were infected with VL. Hundreds died before this epidemic was discovered, while the treatment centre established in Nasir District in November 1994 had to be abandoned in March 1995 because of fighting.¹⁸ The civil war continues to disrupt all aspects of life, hindering agriculture, relief and rehabilitation programmes and precluding widespread preventive measures against visceral leishmaniasis in southern Sudan.

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