

Research Articles

Temporal and spatial epidemiology of sleeping sickness and use of geographical information system (GIS) in Kenya

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Abstract

Background & objectives: In Kenya, sleeping sickness (SS) caused by *Trypanosoma brucei rhodesiense* is confined to the Nyanza and Western Provinces tsetse belts. Over the last two decades, the disease has exhibited great spatial variability in its spread and distribution. The objectives of the study were to map the spatial and temporal distribution of SS and determine possible risk factors associated with the disease in western Kenya.

Methods: Geographical coordinates of villages were obtained using a Global Positioning System (GPS). SS data were analyzed retrospectively and the mapping of villages was done using MapInfo Software®. Epidemiological data of villages affected by SS were then correlated to human and cattle population.

Results: SS has spread northwards affecting the western parts of Busia, Teso, and of Bungoma districts in the late 1990s. Most of the SS cases were reported between March and June. The mainly affected age groups were from 20 to 49 years. SS was highest in areas with low human population density, ranging from 0–340/km² and high livestock population, ranging from 5000 to 10,000 cattle.

Interpretation & conclusion: There was a shift of SS occurrence from the old foci into new foci occurring at low transmission levels and causing occasional epidemic outbreaks. The study concludes that seasons influenced disease incidences with higher numbers of SS cases being recorded during the wet seasons. Gender and age determined the disease occurrence with most productive age groups being at higher risk. Areas with high livestock populations had low human population densities and had higher SS cases.

Key words Epidemiology – geo-reference – sleeping sickness – spatial – temporal – tsetse

Introduction

Human African Trypanosomiasis (HAT) or sleeping sickness (SS) continues to be a major cause of morbidity in sub-Saharan Africa. It is estimated that 55 million people in 36 countries are at risk of the disease¹. Although only 45,000 new cases were reported in 1999, the burden of SS was estimated at 66,000 deaths and two million disability-adjusted life years

(DALYs) lost¹. In Kenya, SS is caused by *Trypanosoma brucei rhodesiense* (*Tbr*). Kenya has experienced a series of SS epidemics in the last hundred years. The disease endemic areas are found in Nyanza and Western Provinces, which form a continuous belt with the south-eastern Uganda foci. Disease epidemics have occurred in Kenya during the years 1902–08, 1950–54, 1964–65, and 1980–84 in Nyanza Province and 1989–90 in Western Prov-

ince^{2,3}. The epidemics that occurred before 1940s were associated with *Trypanosoma brucei gambiense* (*Tbg*) based on clinical diagnosis but a review of historical medical records in Uganda where the disease is presumed to have originated from, confirms that the patients were suffering from *Tbr* and not *Tbg*. The first confirmed form of *Tbr* in Kenya was reported in 1942, having spread from south-east Uganda through the Sio River. The spread was attributed to *Glossina pallidipes* infestation in Samia location in Busia district.

The two sleeping sickness endemic foci (Nyanza and Western Provinces) in Kenya are separated by a vast terrain where cases of SS were reported in the 1940s and early 1960s although the vectors *Glossina fuscipes fuscipes* and *G. pallidipes* are present. Most sleeping sickness cases prior to 1990 were from Lambwe Valley in Nyanza Province. However, from 1990 to 2007, the majority of cases have come from new focus in Teso and Bungoma districts in Western Province. Therefore, the objectives of the study were to map the temporal and spatial distribution of SS from 1977–2007 and determine possible risk factors associated with the disease in western Kenya.

Material & Methods

The study was carried out in the humid and sub-humid zones of western Kenya where the foci of SS are confined. Secondary data on SS were reviewed and all the cases computed to obtain the annual cases of SS in Kenya from 1950 to 2007. The villages that had recorded SS through both active or passive surveillance systems at Kenya Agricultural Research Institute-Trypanosomiasis Research Centre (KARI-TRC) (formerly KETRI) and National Livestock Research Institute (formerly EATRO) hospitals' databases from 1977–2007 were identified. A review of SS databases from the hospitals was done to establish the nationality and determine their distributions per district. Only Kenyan cases were considered. The cases ($n = 333$) between 1977 and 2007 with adequate details were traced and geo-referenced. Out of 125 villages recorded in the SS database, 117 were

geo-referenced. The remaining eight villages could not be located for geo-referencing either due to wrong entry of the village name or the patient gave a non-existent village name. Each village was traced to its actual position on the earth's surface using either the village name or the patient's name. Geographical coordinates of the village positions were marked using Global Positioning System (GPS-Garmin 12 x L). The navigation setup was set as degree decimal units and WGS 84 as the map datum. The village coordinates were downloaded using Mapsource Program[®]. MapInfo version 6.0 Software was used to produce the final outputs and thematic maps of the villages. The most important landmarks having the village names such as schools, churches, market centres and dispensaries, which marked the centre of the village(s), were geo-referenced during the exercise. The geo-referenced villages in Western Province, which has been continually active since 1977–2006 were then linked to 1989 human population⁴ and 1993 cattle population (KARI-TRC, GIS Database).

Results

Annual sleeping sickness cases trends in Kenya (1950–2007): The results from hospital records (KARI-TRC and National Livestock Research Institute Hospitals) indicate that a total of 3539 sleeping sickness cases were reported from Kenya during the period 1950–2007. The annual sleeping sickness trends in Kenya are presented in Fig. 1 with an average of 62 per year. In the past 30 years, SS cases have been reported in 10 districts within Nyanza and Western Provinces. Table 1 shows details of distribution of SS cases per district in Kenya since 1977–2007. The distribution of SS in the affected districts from 1977 to 2007 was as follows: Suba (12%), Homa Bay (3.3%), Rachuonyo (0.6%), Migori (0.9%), Busia (24.6%), Teso (53.2%), Bungoma (4.5%), Mt. Elgon (0.3%), Mumias/Butere (0.3%) and Kakamega (0.3%). Nyanza Province had been SS free from 1990 to 2007 apart from a case reported from Migori in 2000. Over the last two decades, western Kenya contributed almost 80% of the cases.

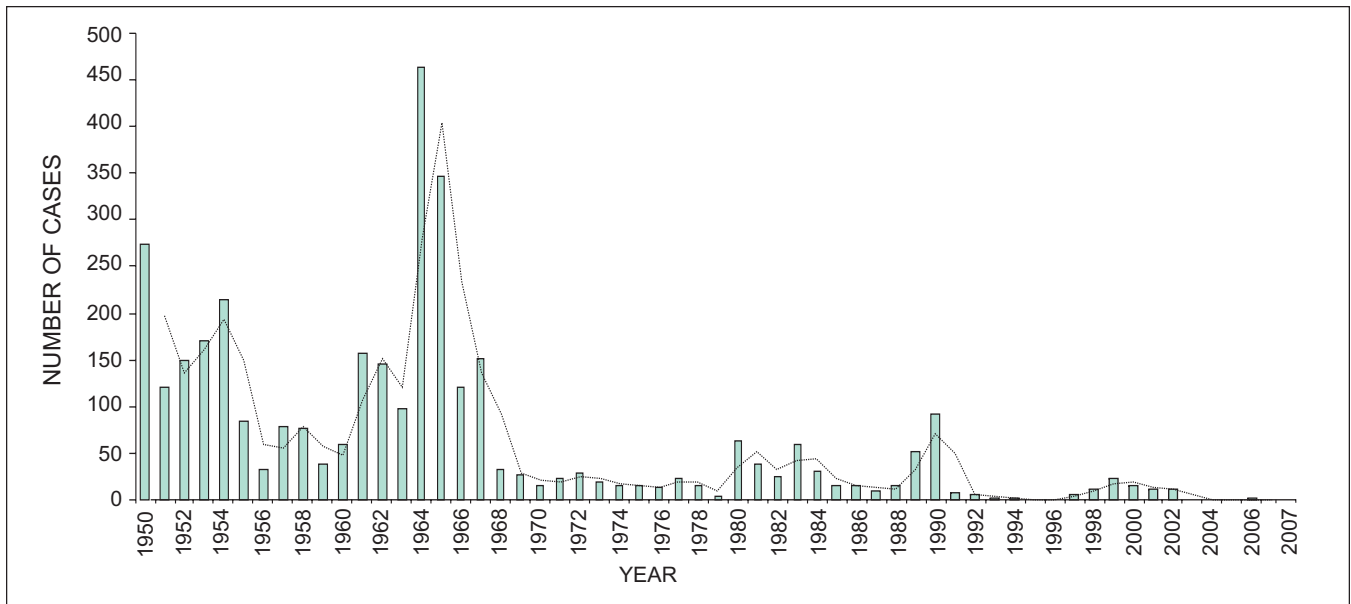


Fig. 1: Trends of sleeping sickness in Kenya from 1950–2007 (Source: KARI-TRC Reports)

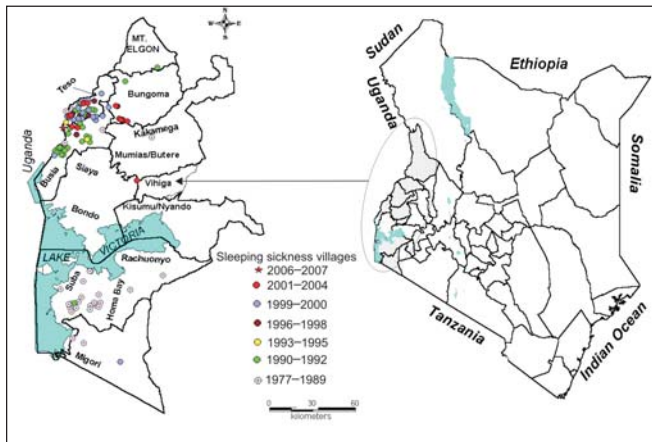


Fig. 2: Spatial and temporal distribution of sleeping sickness in Kenya

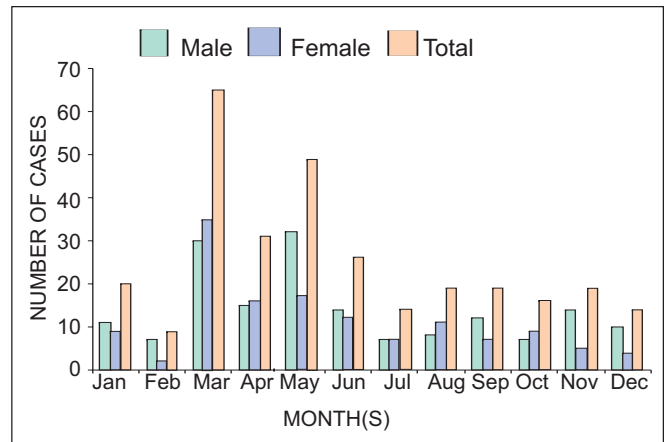


Fig. 3: Cumulative seasonal transmission of sleeping of sickness

The latest active SS focus has been restricted primarily to Western Province in Busia and Teso districts with Bungoma district emerging as a new focus in the late 1990s. However, from 2001–07, Busia, Bungoma and Teso districts have reported 18 cases of SS. Within the period (October 2002–05), the Western Province was SS free until January 2006 when one case was reported within Busia town, Busia district.

Geographical distribution of sleeping sickness in Kenya: The spatial and temporal distribution of SS was illustrated in Fig. 2. In Nyanza and Western Prov-

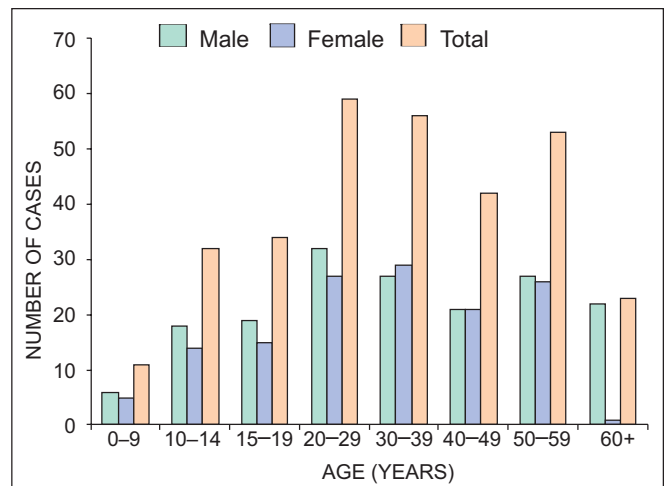


Fig. 4: Sleeping sickness distribution by age and sex

inces, 117/125 villages were identified and geo-referenced (Nyanza 28, Western 89). The geographical distribution of SS from 1977–2007 demonstrated changes in the temporal and spatial distribution over time. It is evident from Fig. 2 that the disease has spread northwards affecting the western parts of Busia, Teso, and of Bungoma districts in the late 1990s. In Nyanza Province, the districts initially affected by SS have not reported SS cases from 1991–2007.

The results were further analyzed and correlated with different variables such as age, sex, human and cattle population. The monthly distributions of SS from 1977–2007 are shown in Fig. 3. Most of the SS cases were reported between March and June of each year. The highest numbers of SS occurrence was in March, reporting 65 cases. The lowest occurrence was in February where only 10 cases were recorded. The male gender was the most affected group throughout

the months apart from March, April, August and October where females recorded the higher numbers. The prevalence of SS among the different age groups from 1977–2007 is illustrated in Fig. 4. Male gender also dominated in almost all age categories except 30–39 years age group. The prevalence of SS increased with age up to the age of 29 years before recording a slight decline. The age categories with high reported cases of SS were 20 to 39 and 50 to 59 years. The least affected age category was the young age group 0–9 years. The results indicate that villages that recorded high SS numbers were Apatit (42), Bukhwamba (14), Obuchun (11), Amaase (10), Katelenyang (9), Kokoki (9). A summary of some affected villages is presented in Table 2. The SS data were further correlated with human density and livestock population. The spatial data showed that SS was highest in areas with low human population density, ranging from 0–340/km² and high livestock population, ranging from 5000 to 10,000 heads of cattle (Fig. 5).

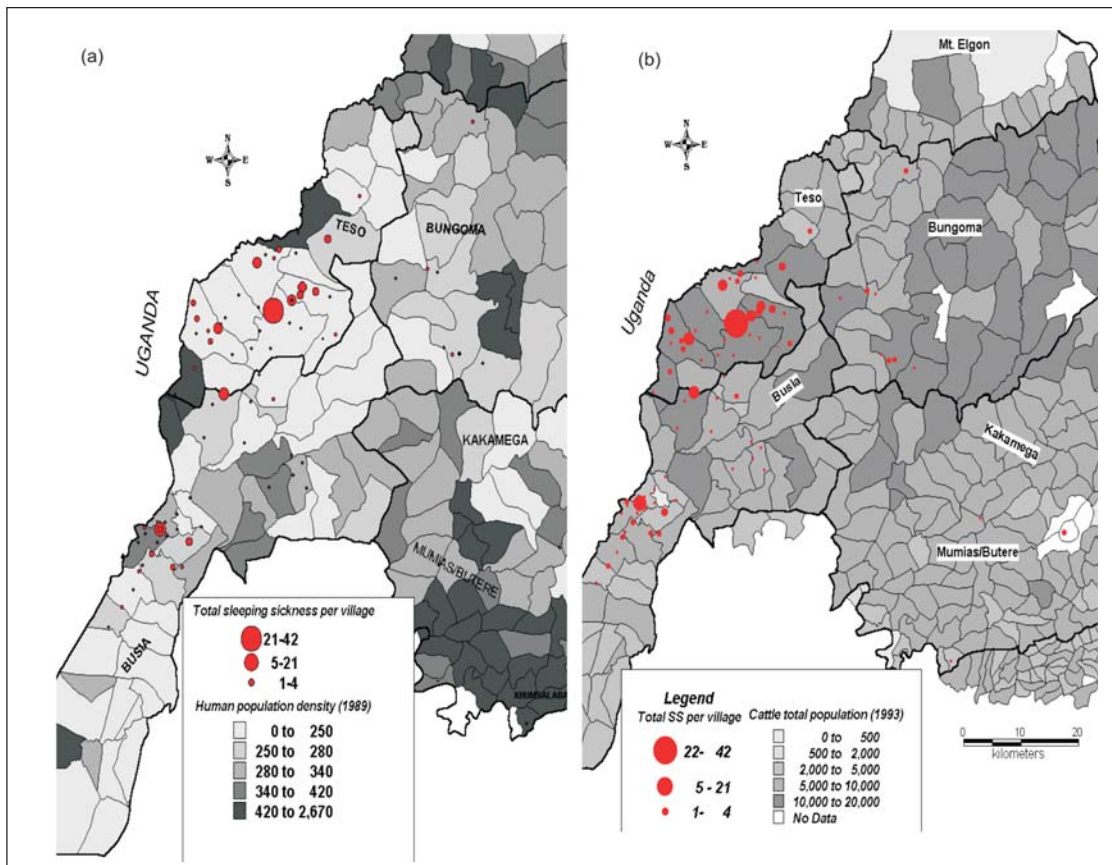


Fig. 5: Correlation of sleeping sickness (SS) incidence (1977–2007) with (a) human population density (1989), and (b) cattle population (1993) distribution

Table 1. Cases of sleeping sickness per province and district in Kenya from 1977 to 2007

Province	District	1977–80	1981–85	1986–90	1991–95	1996–2000	2001–05	2006–07	Total
Nyanza	Suba	0	20	20	0	0	0	0	40
	Homa Bay	0	7	4	0	0	0	0	11
	Rachuonyo	0	0	2	0	0	0	0	2
	Migori	0	2	0	0	1	0	0	3
Western	Busia	4	1	51	10	14	1	1	82
	Teso	1	2	100	4	62	8	0	177
	Bungoma	0	0	1	0	1	13	0	15
	Mt. Elgon	0	0	0	1	0	0	0	1
	Mumias/Butere	0	0	0	0	0	1	0	1
	Kakamega	0	0	1	0	0	0	0	1
Total cases		5	32	179	15	78	23	1	333

Table 2. Villages in Western Province with high SS cases (1977–2007)

Village	No. of cases
Akudiet	8
Amongura	4
Amaase	10
Amoni	4
Amukura	5
Apatit	42
Bukhwamba	14
Ikapolok	5
Katelenyang	9
Kodedema	5
Kokoki	9
Obekai	4
Obuchun	11

Discussion

In Kenya, SS was high during the colonial period but the highest peak recorded in 1964 immediately after Kenya attained its independence. The high SS numbers from 1950s to mid-1960s were attributed to little tsetse work undertaken because of the struggle for independence (Mau Mau Rebellion)⁵ and change of government from the British colonial rule to the independent Kenyan rule. This trend is not unique to Kenya as Médecins Sans Frontières (MSF)⁶ reported

that after gaining independence most SS endemic countries had inadequate budgets to continue with effective routine control activities and also lacked political goodwill to deal with key public health priorities. Moreover, control strategies used during the period included bush clearing, hand catching and insecticide application, mainly concentrated on areas affected by SS, hence, lacked efficacy in large-scale impact on vector.

Over the last two decades, the disease occurrence has been low and sporadic. While tsetse and trypanosomiasis control activities may have contributed to the observed decline in disease trend other important factors such as anthropogenic activities also played a significant role in reducing tsetse habitats and hence the disease incidence³. Cultivation and settlement for instance in affected SS foci such as Lambwe Valley in Nyanza Province increased from 11% in 1941 to 47% in 1993, while Busia district in western Kenya demonstrated change from 23% in 1967 to 47% in 1997³. The most recent data showed that farming activities contribute to 58% of the land cover in Busia district (Rutto, unpublished data). Tsetse and trypanosomiasis control in Western and Nyanza Provinces was mainly done using insecticides such as DDT, dieldrin, cypermethrin bush clearing and settlement to control the disease in human and livestock⁵. The sporadic cases that occurred in districts such as

Migori, Kakamega, Mt Elgon and Mumias/Butere are believed to be epidemiological 'accidents' without much impact on the spread of the disease. Such cases may have contracted the disease while visiting SS active foci. Elsewhere, Maudlin⁷ also attributed the spread of SS in Congo Free State to movement of infected people while another study attributed the disease spread into new foci to livestock restocking⁸.

The SS wave from 1981–90 period corresponds to the withdrawal of DDT and dieldrin use in ground spraying and the epidemics in south-eastern Uganda⁹. In Kenya, dieldrin was banned after 27 years of use in 1983^{5,10}. From 1987–90 cypermethrin application was initiated on trial basis but its efficacy was reduced by ultra-violet biodegradation before its improvement to the long-lasting cypermethrin available in the market currently. The cessation of cotton growing due to low world market prices after the introduction of synthetic fabrics perhaps, contributed indirectly to a surge in tsetse and trypanosomiasis prevalence as the insecticides used may have reduced the vectors in the affected regions. Furthermore, the dwindling government and donor funds and introduction of structural adjustment programmes (SAPs) in the 1990s contributed to a decline in tsetse and trypanosomiasis control activities since governments had limited funds for priority issues and also employees undertaking tsetse control activities were reduced through retrenchment, leading to an increase in fly population and consequently the observed SS numbers. Unfortunately, it is not possible to obtain data on vector population as Angus¹¹ noted that prior to 1990 there were no regular systematic fly-trapping and tsetse control was carried out on an adhoc basis in Kenya.

Most SS cases before 1990 were from Lambwe Valley in Nyanza Province while from 1990–2007 these were from Teso and Bungoma districts in Western Province. The absence of SS after 1990 in Nyanza Province could be due to concerted efforts of tsetse and trypanosomiasis control in the late 1980s^{2,12}. In 1988, Kenya Trypanosomiasis Research Institute initiated a tsetse suppression trial in Lambwe Valley

in Nyanza Province using baited insecticide-impregnated targets and achieved 95% reduction in fly numbers³. Recent tsetse survey in Lambwe Valley reported fly per trap per day (FTD) of 0.96% (Mwangangi *et al* 2007, unpublished report). Concerted tsetse control activities in Western Province in 1990s reduced tsetse fly FTD from 8 to 1.3 by 1994¹¹ which has remained at low levels ranging from 0 to 0.4 FTD. Currently in most areas with the exception of few isolated pockets in northern Busia district, where FTDs of over 200 (currently Teso district) as reported by Okoth¹³ and Rutto *et al* 2008 (unpublished data). The low numbers of tsetse flies experienced during the period and perhaps frequent use of trypanocidal drugs in cattle (to kill pathogenic trypanosomes and non-pathogenic *T. brucei* human infective species) could have contributed towards absence or reduction of SS in Nyanza and Western Provinces. A vast terrain separates the two SS endemic areas of Nyanza and Western Provinces and cases were reported in 1940s and early 1960s although the vector is still present.

The spread of HAT into new areas (Bungoma and Teso districts) could mean that the clean tsetse flies in the new foci might have been infected with the *T. brucei* human infective species. Harley¹⁴ reported tsetse infestation along Malakisi River which is adjacent to Teso district but the flies were not infected with human infective trypanosome species and hence the absence of SS cases reported from the area. Previous reports had also indicated that Busia and Bungoma districts¹⁵ had low tsetse fly numbers considered scientifically not capable of transmitting disease¹⁶, yet in the area a new SS focus has emerged.

The maximum rainfall in the region usually occurs between April and May with major rainy season starting from March to May^{17,18}. The short rain usually occurs from October to November while the dry season is experienced from June–September and December–February. SS incidences were highest in March each year. This coincides with the onset of the long rains and increased farming activities such as land preparation, and planting. The SS incidences

remained constantly high during the wet seasons (1977–2007). Furthermore, during the onset of the wet season, the tsetse fly's life expectancy is maximal, therefore, the flies survive for long periods, thus, enhancing their capability to transmit disease depending on their infection rates. Anthropogenic activities such as livestock keeping, crop farming and cultural activities increase the human/fly contact. Kokwaro *et al*¹⁹ demonstrated that socio-cultural and economic activities undertaken by different gender influenced human-vector interactions. Sociocultural and economic activities are gender designated which accounts for the gender differences in SS incidences.

The studies have shown that livestock could act as reservoirs of the parasite and as host for the tsetse fly^{11,20,21}. Enyaru *et al*²² found zymodemes from domestic animals such as cows and the pigs as being identical to those in man. The foregoing study supports the current findings that demonstrate a positive correlation of cattle population and SS incidences. The areas with high livestock populations had low human population densities. Low human population could lead to land under-utilization and consequently bush encroachment forming a suitable tsetse habitat.

The study showed a shift of SS occurrence from the classical or traditional foci into new foci occurring at low transmission levels and causing occasional epidemic outbreaks. While current SS incidence in Kenya is low, effective control strategies to control transmission or eliminate the disease are still needed. The study concludes that seasons influenced disease incidences with higher numbers of SS cases being recorded during the wet seasons. Gender and age determined the disease occurrence in the community with most productive age groups being the most affected. The study demonstrated a strong correlation between cattle numbers, human population and SS occurrence. The study also demonstrated that the use of GIS in research could assist in mapping disease, prevalence and spread over time and space. Therefore, GIS tool should be refined further to assist in forecasting SS, leading to a better management of the disease in future.

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