

MALARIA DURING THE 1991 - 1992 NORTH - EAST MONSOON SEASON IN A VILLAGE IN THE INTERMEDIATE RAINFALL ZONE OF SRI LANKA

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Abstract: A longitudinal epidemiological study of malaria was performed at Nikawehera, an established farming village in the intermediate rainfall zone of Sri Lanka. Observations were made over a period of eight months from October 1991 that included the north-east (NE) monsoon season and the early part of the south-west (SW) monsoon season. The incidence of malaria and clinical indices related to malaria at Nikawehera are reported here. Compared with observations made at Nikawehera in the previous year, the present results suggest that malaria transmission is variable and that suitable intervention e.g. through effective insecticide spraying or malaria education, can reduce malaria incidence in such locations.

Key words: Clinical indices, immunity, intermediate zone, malaria, monsoon, parasite densities.

INTRODUCTION

Malaria is a serious problem affecting the health and economy of the country. Environmental factors, particularly rainfall, determine to a large extent the occurrence of malaria in different parts of Sri Lanka.¹ Rainfall is unevenly distributed in the country. The wet zone receives rainfall (>2500 mm per year) throughout the year with two specific monsoonal periods of heavy rainfall viz. the north-east (NE) monsoon in October-January and the south-west (SW) monsoon in May-July. The dry zone gets maximum rainfall (<2500 mm per year) during the NE monsoon and little or no rainfall during the rest of the year. An intermediate zone with mixed properties may be demarcated between the dry and wet zones (Fig. 1). It is reported that the intermediate zone is more amenable throughout the year to pool formation that is suitable for the breeding of *Anopheles* mosquitoes.¹ The ratio of *Plasmodium falciparum* to *P. vivax* infection detected by blood filming in Sri Lanka has increased in recent times, from 1:33 in 1980 to 1:4 in 1991.² An understanding of malaria transmission in different regions of Sri Lanka is necessary for developing successful control and eradication measures including the possible use of a malaria vaccine.³ Recent information on malaria transmission in Sri Lanka shows that there

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is significant regional variation.⁴⁻⁷ A study was initiated in October 90 at Nikawehera, a farming village located in the intermediate rainfall zone, involving clinical and parasitological examinations as well as measurement of antibody levels at approximately three monthly intervals.⁷ The clinical and parasitological data obtained in the period October 91 to May 92 are reported here.

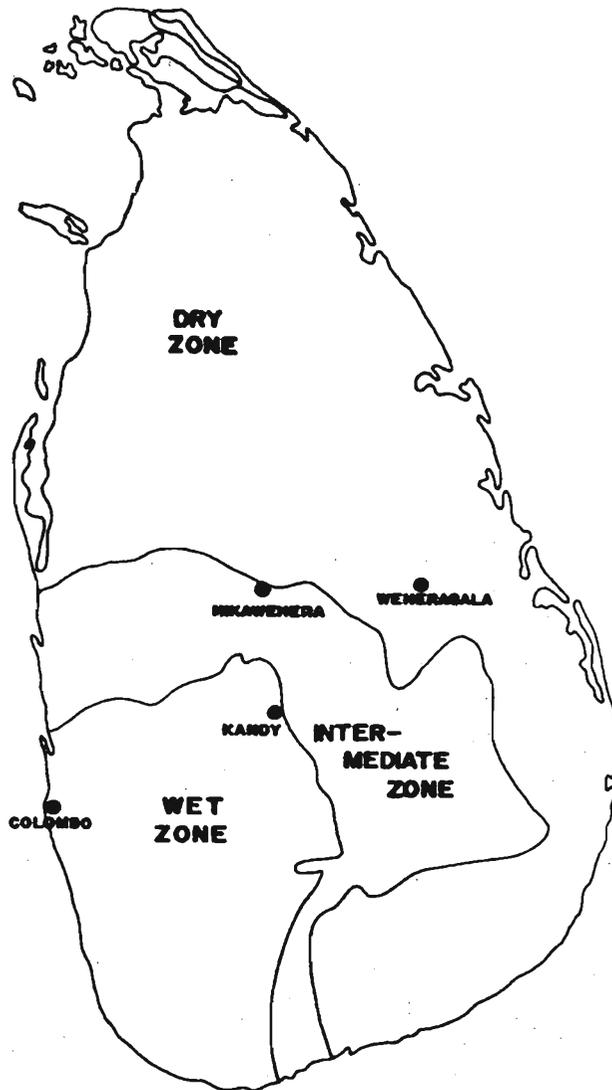


Figure 1: Map of Sri Lanka with the study site, Nikawehera, identified.

METHODS AND MATERIALS

Nikawehera, a village in the Matale district located in the intermediate zone but close to the dry zone (Fig.1), was chosen for the study. Nikawehera is predominantly composed of farming families that have been resident for many generations at the site. Records from Dambulla and Galewela district hospitals, located 10 and 5 km respectively from Nikawehera, show a high malaria incidence in the area (Fig.2). A sample of 147 persons composed of 34% children aged 7-15 years and 66% adults were selected for the study in Oct 1990.⁷ Of this initial population, 76 (children 36%), 82 (children 41%) and 82 (children 40%) were present in the October 91, January 92 and May 92 surveys respectively. During the period described in this investigation the age range of the same selected group of children is taken as 8-16 years. Informed consent was obtained from each person and from parents in the case of children, for the study. Houses in Nikawehera were sprayed with malathion by the Anti-Malaria Campaign, Ministry of Health during the period of study in October 91, January 92 and March 92, following standard anti-malaria spraying procedures.

Medical data collected at Nikawehera included characteristics of previous fever attacks and accompanying symptoms as well as the time elapsed since the last treatment with antimalarial drugs. Other parameters recorded were anaemia (clinically assessed by conjunctival pallor), hepatomegaly and splenomegaly (measured by conventional palpation with fingers). After the physical examination, blood samples were withdrawn under aseptic condition by venepuncture and thin and thick blood films were made and examined for malaria parasites after staining with Giemsa. The number of parasitised cells in fields containing 200 white blood cells (WBC) were determined and parasite densities calculated assuming an average of 8000 WBC per μ l of blood. Serum samples were used to detect antibodies against specific epitopes by immunoradiometric assays (submitted for publication).

Table 1: Prevalence of Parasitaemia at Nikawehera in 1991-1992.

	October 91		January 92		May 92	
	A	C	A	C	A	C
<i>P.vivax</i>	5/49 (10%)	2/27 (7%)	7/48 (15%)	2/34 (6%)	3/49 (6%)	8/33 (24%)
	n.s.		n.s.		p < 0.05	
<i>P.falciparum</i>	3/49 (6%)	3/27 (11%)	1/48 (2%)	0/34 (0%)	4/49 (8%)	1/33 (3%)
	n.s.		n.s.		n.s.	

A = adults; C = children;

n.s = not significant

Significance was determined by the chi square test.

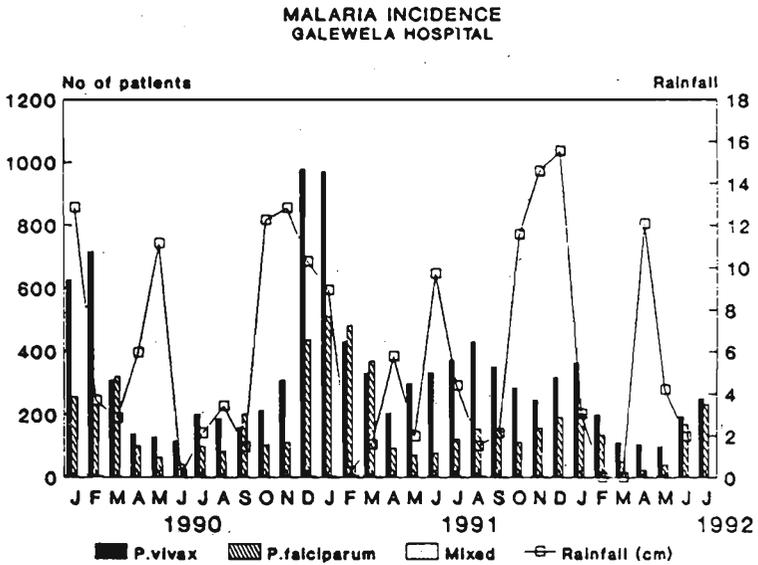
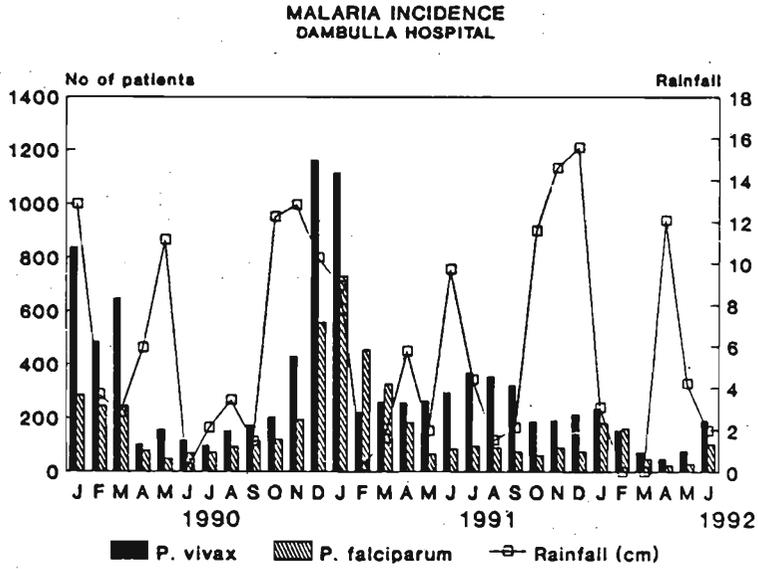


Figure 2: Numbers of malaria patients seeking treatment at government hospitals in Dambulla and Galewela related to rainfall during the period January 1990 - June 1992.

RESULTS

Questioning during each of the surveys revealed that about 50% of persons presenting for clinical examinations experienced one or more malaria attacks during the period October 91 - May 92. The prevalence of anaemia was 61%, 56% and 28% in October 91, January 92 and May 92 respectively. The incidence of splenomegaly and hepatomegaly in the study population are shown in Figures 3 and 4 respectively. The proportion of children among those with palpable spleens, were 9/16, 10/12 and 7/12 in October 91, January 92 and May 92 respectively. Children had higher spleen enlargement rates ($p < 0.05$ by chi square analysis) than adults in October 91 and January 92 (33% and 29% in children compared with 14% and 4% for the adults in October 91 and January 92 respectively). A similar comparison (21% children vs 10% adults) in May 92 showed that the difference was not statistically significant. 22%, 35% and 9% children had palpable liver in October 91, January 92 and May 92 respectively. Corresponding values for the incidence of anaemia in children were 52%, 50% and 18% respectively. Only the liver index showed a peak in January 92 in the study population. Table 1 gives the parasite prevalence rate and Table 2 the mean parasite densities in the study population. The total parasite prevalence rate was 17.1% in October 91, 12.2% in January 92 and 19.5% in May 92. There were no statistically significant differences between the parasite prevalence rates in adults and children except in May 92 when children had a higher prevalence of *P. vivax* ($p < 0.05$ by chi square analysis). The parasite densities in adults and children were not significantly different at any time.

Table 2: Parasite densities at Nikawehera.

	October 91		January 92		May 92	
	A	C	A	C	A	C
<i>P. vivax</i>	1424±484	1260±420	234±461	220±60	1340±774	740±313
	n.s.		n.s.		n.s.	
<i>P. falciparum</i>	1066±462	1093±162	1360	-	3140±2485	120
	n.s.		n.d.		n.s.	

A = adults; C = children;

n.s. = not significant; n.d. = not done

Significance was determined by the chi square test. The results are expressed as mean number of parasites per μl \pm standard error of the mean.

SPLEEN INDEX

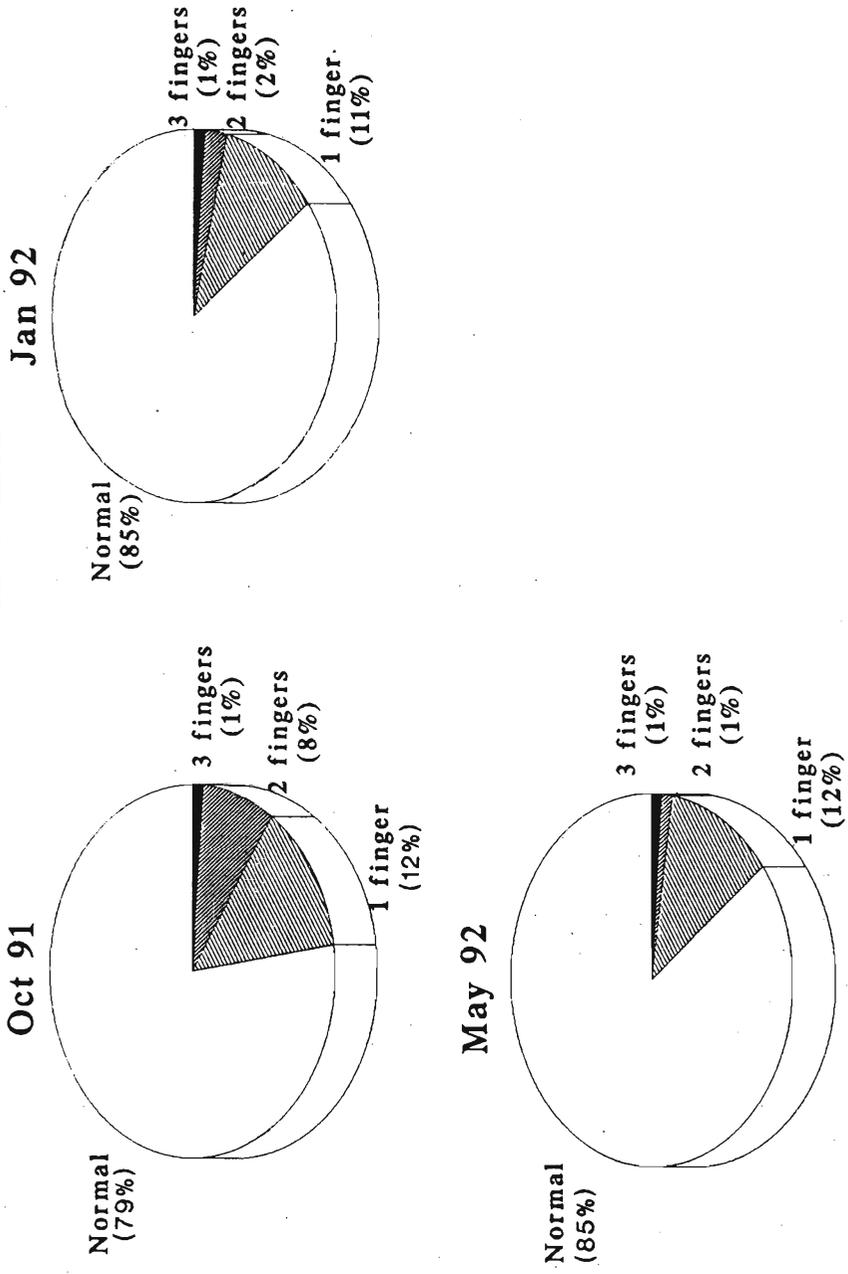


Figure 3: Spleen indices at Nikawehera.

LIVER INDEX

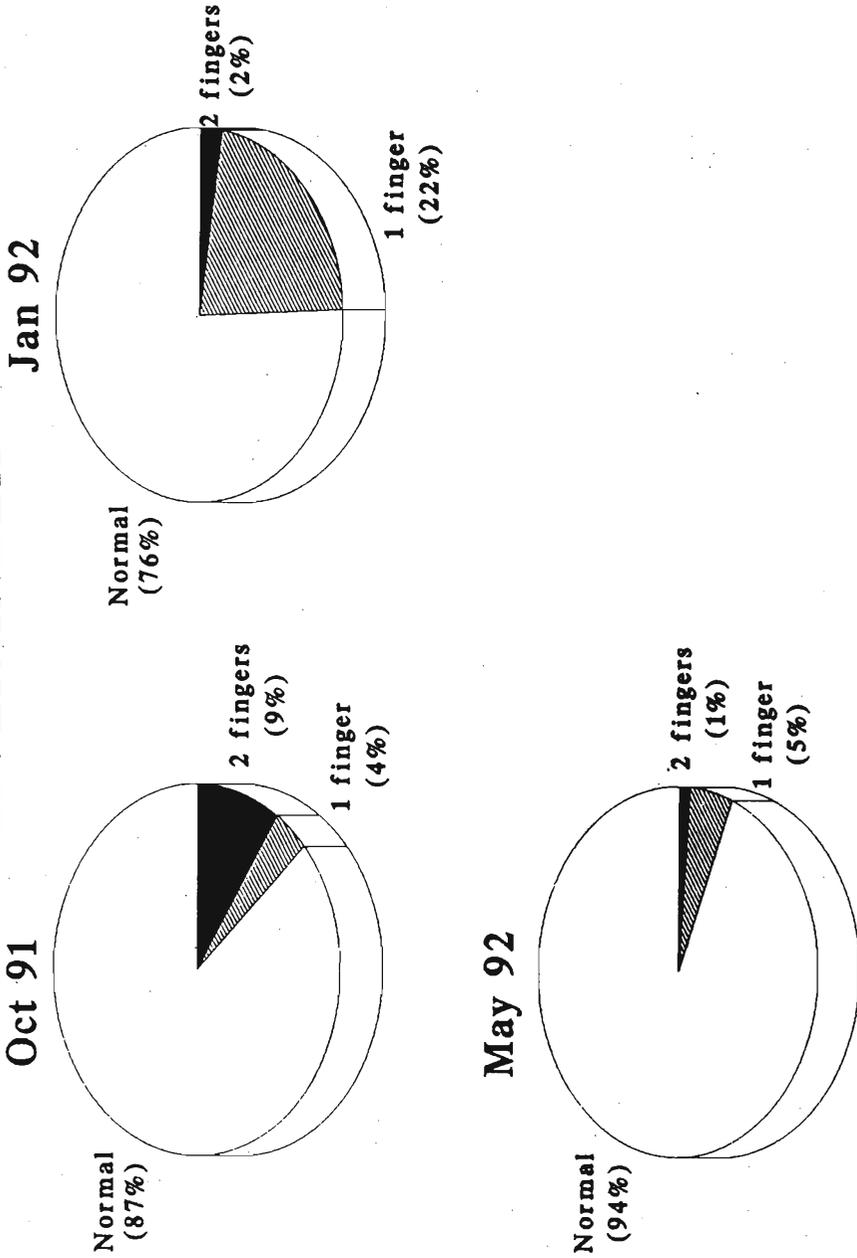


Figure 4: Liver indices at Nikawehera.

DISCUSSION

Results from the Nikawehera study obtained over the preceding twelve month period (October 90 - September 91), showed that malaria incidence and the clinical indices were related to entomological transmission rates of malaria.⁷ They indicated a tendency towards a peak of clinical symptoms and malaria incidence in January 91 at the end of NE monsoon transmission season. Antibody levels (submitted) and entomological inoculation rates⁷ were also maximal in January 91. In contrast, the present results show that there was a lower malaria incidence in January 92 than in May 92. It can be speculated that this difference may be related to more effective spraying by the Anti-Malaria Campaign during the 91-92 NE monsoon season (compared to the 90-91 season when spraying activities may have been partly disrupted as a result of civil unrest in 89 - 90) and the drought experienced in February - March 92 and followed by heavy rainfall in April - May 92. Malaria incidence among outpatients from Galewela and Dambulla hospitals in the vicinity of Nikawehera showed a smaller increase in December 91 - January 92 compared to December 90 - January 91. After a period of low malaria incidence in March - May 92, an increase was again seen in June 92 - July 92 among the outpatients. These results are consistent with the antibody responses observed against *Pvivax* and *Pfalciparum* circumsporozoite and merozoite surface proteins in the population in Nikawehera, during the same period (mean antibody levels and prevalences showing a peak in January 91 but not in January 92 accompanied by an increase in May 92 - submitted). Lower malaria transmission rates in the 91 - 92 NE monsoon season is also suggested by the failure to identify *Plasmodium* infections among the 513 *Anopheles* night biting mosquitoes collected at Nikawehera (submitted).

When compared to the preceding NE monsoon when the proportions of palpable spleens were 24% in October 90, 40% in January 91 and 27% in May 91 respectively and the proportions of anaemic individuals were 76%, 71% and 14% in October 90, January 91 and May 91 respectively,⁷ the present observations show lower spleen rates and overall fewer anaemic persons. This may partly be due to lower malaria transmission rates in the 91 - 92 NE monsoon season at Nikawehera and partly to our observations that persons in the study group having been educated on malaria for several months, took more rapid treatment after the onset of symptoms of malaria. Apart from significantly higher spleen rates in children in October 91 and January 92 and the higher *Pvivax* incidence among children in May 92, our results did not show other evidence for differences in clinical immunity to malaria between 8-16 y olds and adults. This may be partly due to the small sample size in our study, since studies in highly malaria endemic areas of Africa show that there is significantly greater immunity in adults.⁸ However, we have observed that the population in Sri Lanka, in contrast to Africa, have more rapid access to medical treatment.⁹ We have suggested that this difference may result in a slower rate of acquisition of clinical immunity with age in Sri Lanka, which may in turn reduce immunity differences between 8-16 y old children and adults⁹. Younger children (<8y), who may have been significantly more susceptible

than adults to malaria, were not included in the study at Nikawehera for logistical reasons.

During the 91 - 92 NE monsoon season, Nikawehera had characteristics of a location mesoendemic for malaria (spleen and parasite rates typically between 11 - 50%). In contrast, our studies showed that in Weheragala in the dry zone⁶ and in Nikawehera during the 90 - 91 NE monsoon season,⁷ malaria transmission had characteristics of hyperendemicity (spleen rates in children > 50%). The results therefore suggest that malaria transmission in Nikawehera is variable from year to year. This may be due to factors such as rainfall, a better understanding of malaria among the population and the efficacy of spraying insecticides at Nikawehera.

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