Periodicity in the Incidence of Filariasis and the Solar Cycle

K. Tennakone*

Institute of Fundamental Studies
Hantana, Kandy, Sri Lanka
and
Department of Physics
University of Ruhuna
Matara, Sri Lanka

and

D. Tantirigoda

Department of Physics
University of Sri Jayawardenepura
Nugegoda, Sri Lanka

Transmitted by John Casti

ABSTRACT

An analysis of data on the incidence of filariasis in Sri Lanka indicates a periodicity of 10–11 years. It is suggested that this epidemic possesses an intrinsic oscillatory character and the sunspot cycle via its influence on rainfall acts as a resonating force. A mathematical model is constructed to explain this phenomenon and the observed phase lag between the two cycles.

INTRODUCTION

Vector-borne parasitic diseases continue to be a serious health problem in the tropics. The mathematical modeling and statistical data analysis of these ecosystems have attracted much attention, and in some cases such studies

*Sumanasekera Professor of Natural Science.

655 Avenue of the Americas, New York, NY 10010 0096-3003/89/$03.50

have assisted in carrying out more successful eradication programs [1–5]. The models of malaria and schistosomiasis are the best-known, practical examples [6–7]. Another vector-borne parasitic infection whose control is becoming exceedingly difficult is filariasis [8–11]. Vast regions of Asia, Latin America, Africa, and the Pacific are permanent habitats for the disease, where it has been endemic for centuries. Although mosquito eradication programs and chemotherapy are reducing the incidence of the disease in some areas, complete control seems to be difficult, and the disease tends to spread into thickly populated and low elevation regions where stagnation is perennial [8–10].

We have carried out an analysis of the data on the incidence of filariasis in Sri Lanka for the past 17 years and found evidence for periodic behavior in the probability of its occurrence. The consecutive maxima and minima are found to appear at intervals of 10–11 years. As the solar (sunspot) cycle has the same duration, one would naturally suspect whether the observed periodicity is an influence of this external rhythm. An inconspicuous but detectable periodicity has also been noted in the rainfall pattern of the monsoon regions of South East Asia, including Sri Lanka [12–13]. A rainfall fluctuation could induce small but proportionate changes in the mosquito population. However, as the amplitude of the 10–11 year cycle in the rainfall is small [12–13], it is more likely that the rainfall via its influence on mosquito breeding acts as a periodic impetus on an intrinsic oscillation present in the epidemic, exciting a resonance.

A mathematical model is here constructed to illustrate the phenomenon. We have also noted that the peak position in the probability of incidence of the disease does not coincide with the solar maximum or minimum but found to have a phase lag of ~ 3 years. The effect can be understood as a consequence of the fact that prolonged exposure to mosquitoes is needed to acquire the disease. The implications of this study for the evolution and eradication of filariasis are discussed.

OBSERVATIONS AND RESULTS

The life cycle of the filarial parasite is well known [7–9]. The parasite reservoir and definitive host is man. Adult worms harbored in the lymphatic system discharge microfilariae into the blood stream. During the day, microfilariae hide in the lymphatic tissue, and they generally go into the blood stream at night. The disease is detected by screening blood samples taken at night (after 20:30) for microfilariae (night blood test).

The information we have used in this investigation are based on data obtained from the Antifilaria Campaign of Sri Lanka. In a number of urban and suburban regions a significant portion of population is kept screened for filariasis and the positive cases are given drug treatment. As a typical example, the time variation of the percentage incidence of the disease in an area of population 155,000 is presented in Figure 1. Clearly rising and declining portions followed by a maximum and minimum are noticeable. To locate the maximum and minimum points, the following procedure was used. The declining and rising portions are fitted with straight lines by linear correlation analysis; their intersections give the maximum and minimum positions. The analysis of data for the other regions of Sri Lanka (where the units of Antifilaria Campaign have conducted blood tests) revealed behavior similar to that of Fig. 1.

Table 1 gives the positions of maxima and minima for each region as determined by the method mentioned earlier. The population in these regions and the percentage incidence of the disease at the maximum positions varied in the ranges 150,000–10,000 and 1.5%–0.2% respectively. It is seen that the positions of maxima and minima in almost all regions occur at the same point on the time axis. Table 2 indicates the mean positions of maxima and minima

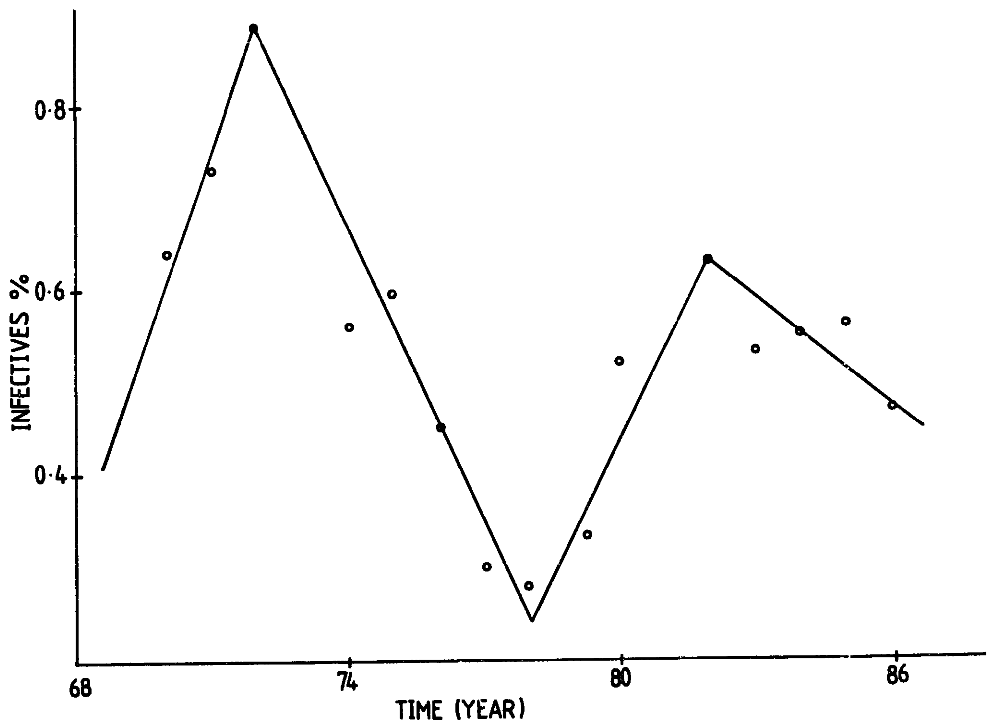

FIG. 1. Time variation of the percentage incidence of filariasis in a region as detected by the night blood test.

TABLE 1

THE POSITIONS OF MAXIMA AND MINIMA AND THE PERIOD FOR DIFFERENT REGIONS AS DEDUCED FROM THE ANALYSIS OF DATA ON NIGHT BLOOD TESTS

Region	Min	Max	Min	Max	Period (yr)
Dehiwela	1968	1972	1978	1984	10.4
Kotte	1968	1973	1979	1984	11.8
Maharagama	1968	1973	1980	1984	11.4
Godigamuwa	—	—	—	1984	—
Peliyagoda	—	—	—	1983	—
Udahamulla	1968	1973	1979	1982	—
Kelaniya	—	—	—	1984	—
Kalapaluwawe	1968	1973	1979	1984	8.5
Jambugasmulla	—	—	—	1984	—
Kolonnawa	1969	1973	1978	1983	7.0
Mulleriyawa	—	1974	1978	1983	8.4
Pannipitiya	—	—	—	1984	—
Gothatuwa	—	1974	1978	1984	8.4
Kotuwila	1968	1974	1977	1984	9.4
Homagama	—	—	—	1983	—
Dalugama	—	1973	1977	1983	13.8
Kadawatha	—	1971	1979	1983	11.8
Mahara	1968	1972	1978	1985	10.0
Hunupitiya	1968	1973	1979	1985	8.8
Biyagama	1968	1972	1979	1984	9.0
Ragama	—	—	—	1983	—
Makola	1968	1974	1980	1984	8.6
Wattala	—	1972	1978	1985	11.6
Hendala	1968	1974	1977	1983	7.8
Kadana	—	—	—	1982	—
Negambo	1968	1972	1979	1984	—
Seeduwa	—	—	—	1984	—
Gampaha	—	1972	1979	1984	11.4
Jaela	—	—	—	1984	—
Veyangoda	—	1973	1977	1983	10.4
Polgahawela	—	1973	1977	1983	10.4
Pothupitiya	1968	1974	1978	—	6.2
Maggona	—	1972	1979	—	10.4
Paiyagala	—	1972	1980	—	11.4
Walatara	—	1973	1979	—	7.0
Induruwa	—	1975	1978	—	8.2
Haburangala	1968	1972	1976	—	10.4
Kurunegala	—	—	—	1984	10.4
Wellaboda	—	1972	1978	—	9.5
Chilaw	—	—	—	1984	—
Batapola	1968	1974	1980	—	13.5
Battaramulla	—	—	—	1983	—
Talangama	—	—	—	1984	—
Hokandara	—	—	—	1983	—
Bathgamuwa	—	--	—	1985	—

TABLE 2

THE MEAN POSITIONS OF MAXIMA AND MINIMA (F. MAX, F. MIN) IN THE PERCENTAGE INCIDENCE OF FILARIASIS COMPARED WITH THE SOLAR MAXIMA AND MINIMA (S. MAX, S. MIN)[a]

S. Min 1965.0	S. Max 1969.3	S. Min 1976.1	S. Max 1980.3	S. Period 11
F. Min 1968.1 ± 0.1	F. Max 1972.8 ± 0.9	F. Min 1978.2 ± 1.3	F. Max 1983.7 ± 0.11	F. Period 9.8 ± 1.8
P. Lag 3.1 ± 1	P. Lag 3.5 ± 0.9	P. Lag 2.1 ± 1.3	P. Lag 3.4 ± 0.1	

[a]The phase lags (P. Lag) between the maxima and minima are given in the third row (S. Period = solar period; F. Period = mean of data in column 5 of Table 1).

for the solar cycle and the percentage of incidence of filariasis. A periodicity of 10–11 years and a phase lag of ~ 3 years from the solar cycle are evident.

THE MODEL

The sunspot cycle is known to cause changes in the ionosphere, which influence the rainfall. However, the observed effect, though detectable, is small [14–15]. As mosquito breeding depends on rainfall, one would expect that the mosquito population density n would also vary periodically. For simplicity we assume a sinusoidial oscillation of the form

$$n = A \sin wt + B \tag{1}$$

where A and B ($B > A$) are positive constants.

For the disease to progress a sufficient number of larvae must mature, overcoming the action of the immune system, which tends to eliminate them. Consequently a prolonged exposure to infected mosquitoes is necessary to propagate the disease. The rate of biting by mosquitoes depends on their population, and if we assume that on average a contact with mosquitoes for a time τ is necessary, the quantity that influences the development of the disease at time t is

$$\int_{t-\tau}^{t} (A \sin wt + B)\, dt = \frac{A}{w} \sin\left(wt - \frac{\tau}{2}\right) + B. \tag{2}$$

The above argument clearly demonstrates that if an external periodic factor has an influence on the degree of incidence of filariasis, a phase lag can naturally be expected. From the observed phase lag we conclude that $\tau \sim 6$ years, i.e. on average an exposure of a human population to mosquitoes for a period of ~ 6 years is necessary to develop the epidemic.

Since the 11 year cycle in the rainfall has only a small amplitude, it is more likely that the resulting perturbation merely acts as a driving mechanism for an intrinsic oscillation present in the ecosystem. The following model illustrates the possibility of such an intrinsic oscillation. If I, S, and M denote the relative population densities of the infectives, susceptibles, and the immunes (i.e. the population densities as fractions of the total population density, which is assumed to be constant), their rates of change can be expressed in the form

$$\frac{dI}{dt} = aIS - bI, \tag{3}$$

$$\frac{dS}{dt} = cM - dIS, \tag{4}$$

where a, b, c, and d are constants. Equation (3) states that infectives are produced at a rate proportional to IS and removed at a rate proportional to I; Equation (4) states that the interaction of susceptibles with the infectives removes susceptibles at a rate proportional to IS. The immunes can lose immunity and become susceptibles again. The relative population densities, I, S and M are also subjected to the constraint

$$I + M + S = 1. \tag{5}$$

At equilibrium (i.e. $dI/dt = ds/dt = 0$) Equations (3)–(5) have the solution

$$S = S_0 = b/a, \tag{6}$$

$$I = I_0 = \frac{c(a-b)}{ac+bd}, \tag{7}$$

$$M = M_0 = \frac{bd(a-b)}{a(ac+db)}. \tag{8}$$

In the linear approximation

$$S = S_0 + s,$$

$$I = I_0 + i, \tag{9}$$

where $s < S_0$, $i < I_0$, the equations (3)–(5) give a solution of the form

$$i = i_0 e^{-Ht} \sin(wt + p), \tag{10}$$

where

$$w^2 = 4c(a - b) \tag{11}$$

$$H = c + dI_0, \tag{12}$$

and $p =$ constant, provided the parameters a, b, c, and d satisfy the condition

$$4\frac{a-b}{c} > \left[\frac{a(c+d)}{ac+db}\right]^2. \tag{13}$$

Thus the system possesses intrinsic oscillatory behavior, i.e., the relative population density of the infectives undergoes damped oscillations of period $2\pi/w$ about the equilibrium position. In the presence of an external influence of the same periodicity, persistent oscillations could occur as a resonance effect. The oscillations appear only if the inequality (13) is satisfied; otherwise any perturbative deviations from the equilibrium decay exponentially, indicating that in this case, the equilibrium is asymptomatically stable.

CONCLUSION

According to the model, the epidemiology of the system possesses the ability to perform damped oscillations. As a result of damping, persistant periodic variations are possible only in the presence of an external influence of the same period. Here we suggest that the external factor is the periodic rainfall variation caused by the solar cycle. It is possible that the synchronism of the two periods has an evolutionary advantage in keeping the parasite reservoir at a higher level, thus preventing the breaking of the parasite cycle.

As a simplification, mosquito-host interaction was not taken into account explicitly in constructing the model. Models that include these details reduce to equations of the form (3)–(4) as an approximation. A preliminary examination of the data on the incidence of malaria in Sri Lanka did not reveal the existence of oscillations correlated with the solar cycle. Although equations of the form (3)–(4) could apply even in this case, a large deviation of the intrinsic period from the 11 year cycle is perhaps preventing any resonance phenomenon. Again, oscillations do not occur if the condition (11) is not satisfied. In most mathematical models of recurrent epidemics, the oscillations come as the result of terms that represent a steady influx of new susceptibles [16]. It is interesting to note that in the present model the oscillations occur even if the population density remains stationary.

This study suggests that exposure to mosquitoes (or equivalently the prevalence of conditions favorable for mosquito breeding) for a period of ~ 6 years is necessary for the development of the epidemic. Thus permanent measures taken to keep the mosquito level low would be more effective in controlling the disease than short term or intermittent eradication programs.

The authors wish to thank the Antifilaria Campaign of Sri Lanka for providing statistical data.

REFERENCES

1 N. T. Bailey, *Mathematical Theory of Infectious Diseases*, 2nd ed., Hafner, New York, 1975.

2 M. S. Bartlett, *Stochastic Population Models in Ecology and Epidemiology*, Methuen, London 1960.

3 D. G. Kendall, Deterministic and stochastic epidemics in closed populations, in *Proceedings of the 3rd Berkeley Symposium on Mathematical Statistics and Probability*, Vol. 4, p. 149.

4 J. C. Frauenthal, *Mathematical Modeling in Epidemiology*, Springer-Verlag, Berlin, 1980.

5 F. Hoppensteadt, *Mathematical Theories of Populations: Demographics, Genetics and Epidemics*, SIAM, Philadelphia, 1975.

6 G. Macdonald, *The Epidemiology and Control of Malaria*, Oxford U.P., London 1957.

7 A. W. Jones, *Introduction to Parasitology*, Addison-Wesley, Reading, Mass., 1967.

8 F. Hawking, A review of progress in chemotherapy and control of filariasis since 1955, *Bull. WHO* 27:551–568 (1962).

9 J. F. Kessel, Filariasis as a world problem, *Mosquito News* 26:490–499 (1966).

10 N. G. Gratz, Ecology and vector control, *Interdisciplinary Sci. Rev.* 3:214 (1978).

11 N. G. Gratz, Mosquito-borne disease problems in the urbanization of tropical countries, *Crit. Rev. Environ. Control* 3:455–495 (1973).
12 R. Suppiah, Trends and periodicities in the seasonal rainfall fluctuations of Sri Lanka, *Indian Geogr. J.* 61:1–14 (1986).
13 R. Suppiah, Atmospheric circulation variations and the rainfall of Sri Lanka, *Sci. Rep. Inst. Geosci. Univ. Tsukuba Sect. A* 9:1–141 (1988).
14 H. R. Olson, Sun-weather effects, *Nature* 275:368–369 (1978).
15 K. Frazier, *Our Turbulent Sun*, Prentice-Hall, Englewood Cliffs, N.J., 1981.
16 J. Gillis, The mathematical theory of epidemics, *Interdisciplinary Sci. Rev.* 4:306–314 (1979).