Journal of the Nigerian Association of Mathematical Physics
Volume **18 (May, 2011), pp 293 - 298**
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Mathematical Model for The Estimation Of Onchocerca Volvulus Worm Load/Burden in The Body of The Host.

G.C.E Mbah and C.C.Asogwa

Department of mathematics,
University of Nigeria Nsukka

Abstract

In this paper, we present a model for the estimation of onchocerca worm load/burden in the body of the host. Of vital importance to this work is the estimation of microfilaria load in a given host which has also been modeled, Mbah(2007). The age of the host, the transmission potential of the vector, the death rate of the larva and the inhibiting factors are all considered in the model and solved. Analysis of the model is subsequently given.

Keywords: , Hybrid points, Continuous LMM, stiffly stable.

1. Introduction:

Onchocerca volvulus is a filarial worm. The adult male female worms live under the skin of their host, encased in visible nodules in which one or more pairs of worm can be demonstrated [7]. The males measure 2to5cm in length and 0.02mm in diameter while the females are about 50cm in length and 0.04 to 0.06mm in diameter [3].

The adult worms are long thread-like filarial worms, which are white opalescent and transparent with distinct transverse striation of the cuticle. They are wire-like filiform and blunt at both ends.

When a female black fly (Simuluim Damnosum) bites a person infected with onchocerciasis, it sucks in with blood a few tiny worms known as microfilariae. These worms will undergo a certain development to reach the 3rd infective Laval stage in the body of the insect in about 6 or7 days. They are later transferred to a healthy person during the subsequent bites [7]. Once in a human body, the larvae develop to mature male and female worms in about 8-12 months [5]. The females on attaining this age starts to reproduce after the male worm might have fertilized it. The fertilized worm can produce 500,000 to 1million live embryos during its life time. The microfilarias measuring an average of 33microns are long and unsheathed with a characteristic head and pointed tail. They do not enter the blood system but are localized in the skin and eye tissue [2].

The body of a host maintains constant microfilaria density after which it can not accommodate the excess. If no further development of inoculated larva takes place in the skin, that is, when the host has attained a maximum microfilariae density, either they die off or they are ingested by a biting black fly for possible transmission to another human. It has been established that the microfilaria lives for about 1.5years while the lifespan of the adult worm is between 15 to 18 years [8]. For the maintenance of the microfilariae density in the host, there must be replacement of old and worn out worms.

Onchocerca is not a species. There are distinct strains biological variants of the parasite, each with its

Corresponding authors: ***G.C.E Mbah: E-mail: ---,*** Tel. +2348034198454

inherent properties that determine the pattern of biting. To be able to get the required model, thorough knowledge of the transmission mechanism of the microfilaria from one host to another. This we have tried to explain from where we shall then develop the model.

The Model.

Worm burden is defined here as the number of female worms in the body of the host that constantly reproduces the microfilaria. We define this so because, even in the absence of the male worms, the females produce the microfilaria, although very sterile ones. The factors determining the number of worms in a given host are numerous. Among these factors are:-

Age of the worms:-

When the worms are ageing, they release only very few microfilariae. But there is a required microfilariae balance in the body of an infected host. Hence, if the daily output is small against the required number, then there is the tendency for the daily inoculated larva to develop into adult worms to make up for the daily output.

The host's community:-

When certain number of worms exists in the host, the host tends to develop immunity against further development of the larva into worms.

The transmission potential of the fly:-

If we are considering a host that has just been introduced into an endemic area, then the transmitting potential of the flies will account for the rate of development of the worms in body.

Death rate of the worms:-

We know that the worms have a life span of up to 15 to 18years and that at all times, about 2/3 of the worms contain embryonic stages of development of the microfilariae or the microfilariae (mf) themselves. This means that 1/3 of the worms; most of the times are non-producing [5]. We know that it takes the mf about 3 to 4 weeks to develop from ooste to mature microfilariae. Also if the mf fails to leave the uterus of the worm in about 4 week's time, the microfilariae will be absorbed by it. Similarly, the mature worms have a reproducing cycle of about 2.5 to 3.5 months [6].

All these listed factors must therefore have great effect on the worm burden. For brief we list the major factors affecting the worm burden in a given host as follows:-

1. Age of the host A
2. Transmission potential T
3. Host inhibiting factor I
4. Death rate of larva (Natural) δ
5. Capacity for worm burden in the mf production (mf density in the body) D
6. Strain of the onchocerca.

The last factor shall always be remembered when the model on worm burden is being used, since there are different worm burden patterns in the forest and savanna strains of the onchocerca.

Let us briefly look at the variables to be considered. The age of the host affects the density of the microfilaria, and the death rate of the worms. What we mean here is that the density of the microfilaria in a host's body increases with age and added body surface area. Similarly, the immunity of the host against further worm development from the transmitted larvae increases with age when the host has attained microfilaria thresh hold level. The death rate of the worms increases as they age, since the worms have lifespan of between 15 and 18 years. This implies that for a newborn susceptible host, the death rate factor is nil.

In general, we are going to measure the age of the host in terms of the surface area of the human body available for microfilaria and worm inhibiting. This surface area is quite different from that of exposure. Recall that even when a certain part of the body is not exposed such as the breast buttocks, evidence shows that microfilaria inhibits such regions [1]. Hence, the more available surface areas, the more worms are required to produce microfilaria that will meet up the equilibrium microfilaria condition in the body.

Taking these into consideration, we denote the surface area as S, the worm burden as W, such that the variation of the worm burden with respect to time, t, is given as:-

$$\frac{dw}{dt} = \frac{ST}{D(\delta + I)} - \delta^1 w \,, \qquad D \neq 0 \qquad (1)$$

where δ^1 = death rate induced by the age of the host in an endemic area, beyond 10 years.
More generally, we have:-

$$\frac{dw}{dt} = \frac{ST}{D(\delta + I)^\alpha} - \delta^1 w \,, \qquad (2)$$

α = constant determining the state of the function.

Solving, we have:-

$$\frac{dw}{\delta^1 dt} = \frac{ST}{\delta^1 D(\delta + I)^\alpha} \text{-W}$$

$$= \frac{ST - \delta' D(\delta + I)^\alpha w}{D\delta'(\delta + I)^\alpha}$$

that is

$$\frac{D\delta'(\delta + I)^\alpha}{ST - D\delta'(\delta + I)^\alpha w} dw = \delta' dt$$

$$\rightarrow \qquad -\frac{1}{w - \frac{ST}{D\delta'(\delta + I)^\alpha}} dw = \delta' dt$$

Integrating gives

$$\log\left[w - \frac{ST}{D\delta'(\delta + I)^\alpha} \right] = -\delta' t + c \qquad (3)$$

To determine the value of the constant c, we have that at the initial time, t = 0, that is the time the susceptible host was introduced into the endemic area, w = 0 and δ = 0, δ' = 0 and D are relatively low.

$$\text{Thus } \log\left[-\frac{ST}{D\delta' I^\alpha} \right] = c$$

Substituting for c in equation (3) and simplifying, we have:

$$w - \frac{ST}{D\delta'(\delta + I)^\alpha} = \ell^{\left(-\delta' t + \log\left[-\frac{ST}{D\delta' I^\alpha}\right]\right)}$$

$$= -\frac{ST}{D\delta' I^\alpha} \ell^{-\delta' t}$$

Therefore,

$$w = \frac{ST}{D\delta'} \left[\frac{1}{(\delta + I)^\alpha} - \frac{\ell^{-\delta' t}}{I^\alpha} \right] \qquad (4)$$

ANALYSIS OF RESULT

From the above equation, it means that when the mf density (D) is low, then more worms will be produced from the number of larvae being inoculated by the vector. We can also see that when the surface area(S) is small, that is age say below 12years, the rate of change of worm burden is almost a function of $\frac{ST}{D(\delta + I)^\alpha}$ since by then, δ' = 0

After the attainment of this age, then S also increases with time (T) while the rate of inhibition of the development of further larvae into worm also increases. If the worm load in the body of the host is high, the δ will also be high so that the rate of maturation of larvae into adult onchocerca volvulus worm will be very small. Also, this increase in death rate might have been enhanced by the host inhibiting factor. Generally, by the age of about twenty years, we find that the worm load might have been stabilized. At this time, the surface area of the host body must have attained its maximum value although it changes if the individual increases in gait, that is, in size (but not in height) in the host. Hence, the host inhibiting factor will also stabilize and then permits the production of the required number of worms that are needed to beef up the microfilaria density in the body incase of the death of the worms or change in the surface area of the host's body.

Let us buttress this point by making the following assumptions. Suppose we have:
$S = 1.85$, $\delta = 0.8$, $T = 6$, $\alpha = 0.2$, $D = 0.9$, $l = 0.7$ $\delta' = 0.85$ and w_o at $t = 12$ is 20. We then have figure 1:

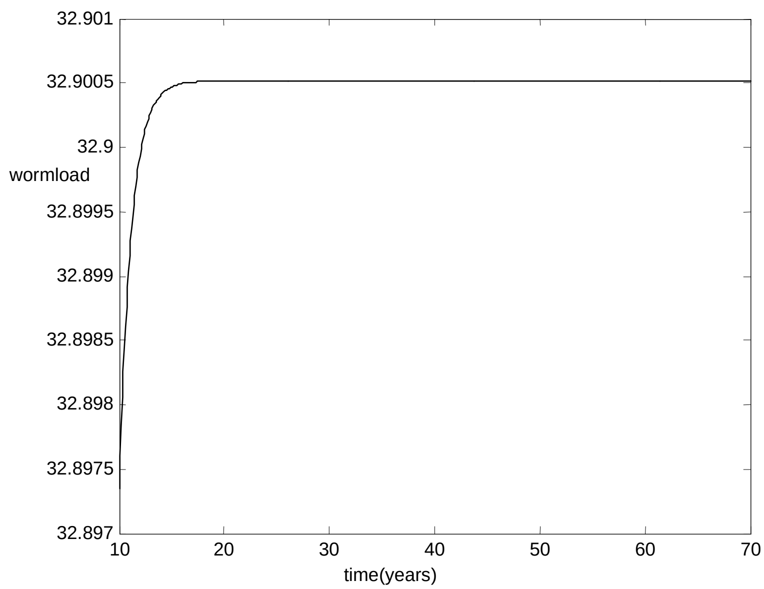

Fig. 1: Worm load stabilization in a host

From this graph, we can see that the worm stabilized at about the age of 20 years when the surface area must have almost attained its maximum size and also the host inhibitory factor has stabilized. We also observed from the model that if there is increase in the size (surface area), as earlier mentioned, the worm load increases to provide adequate microfilariae load necessary for the given size. For instance, when we let s increase from 1.85 to 2.0 with other parameters remaining the same as above, we obtain the Figure 2.

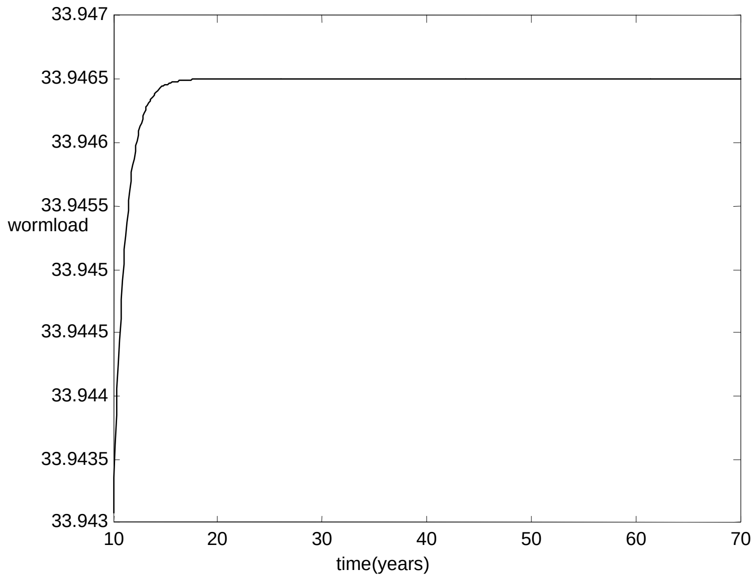

Fig. 2: Variation in Worm load as a result of variation in the size of the host

We can see that the shape of the graph in Fig. 2 remained the same but only a difference in the number of worms in the body.

We equally noticed that an increase in transmission potential increases the worm load. Thus for other parameters remaining the same as in fig. 1 but T increasing from 6.0 to 8.0, we obtain Fig. 3

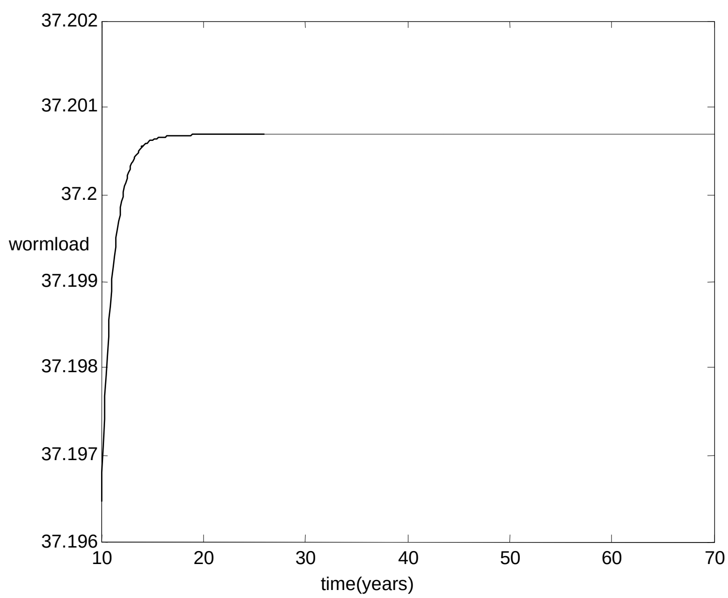

Fig. 3: Variation in worm load as a result of variation in transmission potential

Finally, we may conclude by saying that this model is very necessary and should be made available to the medical personnel and other field workers as this will help them in determining the worm load and subsequently the required medication for respective patients. We can see that differences in body size resulted to different worm load level in the patients. Equally, different transmission potentials present different worm load levels and as such, body size alone is not enough to determine the worm load level in a patient.

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