Journal of the Nigerian Association of Mathematical Physics
Volume **16 (May, 2010), pp 441 - 450**

The Onchorcerciasis Disease: Population Dynamics of Its Host And The Vector.

G.C.E. Mbah and G. C. Okpala

Department Of Mathematics,University Of Nigeria, Nsukka
Enugu State, Nigeria
Department of Statistics,Institute of Management and Technology
Enugu.
Corresponding authors **e-mail: gcembah1@yahoo.com Tel 08034198454, 08057249727**

Abstract

Onchocerciasis disease is a debilitating disease that hampers the well-being and productive capacity of an affected person. In this work, we looked at the population dynamics of the Host (Man), the Vector (Simulium Damnosium – the Blackfly) and the possible Microfilaria output of the worm in the Host via Mathematical modelling. Equally modeled is the possible number of mature larva that can be inoculated back to the host by an infected vector.

These models go to show in greater detail the process of spread of the disease and the transmission pathways as well as the enhancing factors in the transmission process.

These models surely provide broader knowledge of the control processes on the disease spread and will serve a great purpose in the control mechanism of the disease spread.

Keywords: Man, Blackfly, Onchocerca Volvulus, Michrofilaria, Onchocerciasis.

1.0 Introduction

Onchocerca Volvulus is a filarial worm which infects over 40 million people in the world most of who lives in tropical Africa and localized areas of Yemen, Mexico, Guatemala, Venezuela, Colombia and Brazil [12]. This parasite causes the disease called Onchocerciasis, which is one of the most disabling and debilitating parasitic disease known to man, [13]. The characteristics presentation of this disease is dermatitis which not only leads to disfigurement and premature aging in appearance but constant prurities that normally result to incessant scratching. The parasite also causes another ailment referred to as "river blindness"". However, this disease does not necessarily kill on its own [6].

The host to this disease as earlier said is found mostly in tropical African countries and in the localized regions of some European Countries, [20]. Because of this, the victims are usually illiterate and poverty-stricken peasants in remote rural areas who hardly make a hue or cry about their plight and even when they do, not until recently, they may not even be heard. For any person to actually contract this disease, one has to be exposed to repeated bites of the vectors for a reasonable length of time. The size of the worm, which is about 19 to 42μm long and 130 to 210 μm wide for the male and 33 to 50 μm long to 270 to 400 μm

wide for the female, is relatively very small compared to that of the host such that it is only when the worm has accumulated that it can have a reasonable influence on the host as a disease, [19].

As for the vector, which is the black fly (Simulium Damnosium), two distinct strains, the forest and Savannah, have been identified which are different in epidemiology, clinical features and even vector infectivity [5], [6], [7].

The worms in the host are usually encased in the nodules that are mostly found above the waist in central America and below the waist in Africa. There are various species of the black fly. Their mouthparts are modified for feeding on tissue juice of animals including man. These juices are found under the epidemies such as in blood. On biting an infected man therefore, they ingest the microfilaria along with the tissue juice. The microfilaria are migratory and motile such that they can always be found in the skin, subcutaneous tissues, lymph, the eye and occasionally in the blood, urine and cerebrospinal fluid.

The reason for this study is to understand the behaviour of the adult worm and the young worm – Microfilariae. There are effects due to the adult worm and those due to the young microfilariae. In older infections, the worm induces tissue reactions, which results to dermatitis and formation of nodules. For the microfilariae, the effect is more serious which is due to large number of dead ones as they induce allergic reactions on the host ([3] and [17]). However, the most dreaded effect of the disease on the host is the river blindness, which may be impaired vision or total blindness.

2 The Host's Population Dynamics

We consider this rather mathematically believing that we are aware of certain facts about the worm and the Host . The Host population is divided into the susceptible, infected and the removed classes. The removed class refers to those Hosts that are dead not necessarily due to the disease, as the disease does not kill on its own but induce morbidity.

Suppose we denote these three classes or stratification of the Host population as S, I and R. Let the infection rate of the susceptible by the infected vectors be β. Let the infected vector be $I^{/}$. Then using mathematical model, the number of new infections occurring in the host population in time interval Δt is given as.

$$\beta S I^{/} \Delta t \tag{2.1}$$

Suppose the removal rate of the infected Host is σ. Then the removal occurring in the population in time Δt is given by

$$\delta I \Delta t \tag{2.2}$$

This σ may be natural.

We shall assume that all susceptible Hosts if allowed to remain in the affected area must be infected before they die. This argument is suggested by the fact that the microfilaria worm has been detected in a two months old child. We shall be interested on the fact that a Host was infected by the worm and not necessarily whether the worm is fully developed or have started producing microfilaria. Thus population stratification of the Host is therefore:

$$S \xrightarrow{\beta} I \xrightarrow{\delta} R \tag{2.3}$$

where β is the infection rate of the succeptible by the infected

δ is the removal rate of the infected

The Model Equations:

The change in the susceptible population over time is thus given as

$$\frac{dS}{dt} = -(\beta S I^{/} + d S) + bS \tag{2.4}$$

where dS represents number of deaths of the susceptible occurring naturally and

bS represents the number reproduced due to the presence of females in the population.

It has to be noted that Onchocerciasis infection is not hereditary so that at birth, the Host is susceptible.

For the infected population of the Host (I), we have the model for its change as:

$$\frac{dI}{dt} = \beta SI' - \delta I \tag{2.5}$$

Finally, we can get the model for the removed class. Since this disease does not kill, it then means that removal in this case (which means death) comes naturally. Thus

$$\frac{dR}{dt} = \delta I \tag{2.6}$$

Solution of the model equations

We solve this equation (2.4) by integration to get:

$$S(t) = S_o e^{(-\beta I' + \gamma)t} \qquad \text{where } \gamma = b - d \tag{2.7}$$

Equation (2.7) represents a case where there is no immigration into or emigration out of the population but only new ones are born. In case where this exists, then there will be appropriate adjustments in equation (2.4) to take care of this and thus a change in the present solution given in equation (2.7).

In a similar way, the solution to equation (2.5) is given as:

$$I(t) = \frac{\beta SI'}{\delta} + c e^{-\delta t} \tag{2.8}$$

At $t = 0, I(0) = 0$ so that $S = S_o$ and $I^{/} \neq 0$ but $I^1 = I_o^{/}$ and thus

$$0 = \frac{\beta S_o I_o^{/}}{\delta} + c$$

$\Rightarrow$ $c = -\frac{\beta S_o I_o^{/}}{\delta}$ so that equation (2.8) becomes

$$.. I(t) = \frac{\beta SI'}{\delta} - \frac{\beta S_o I_o^{/}}{\delta} e^{-dt}$$

$$= \frac{1}{\delta}\left(\beta SI' - \beta S_o I_o^{/} e^{-dt}\right)$$

$$= \lambda\left(SI' - S_o I_o^{/} e^{-dt}\right) \tag{2.9}$$

for $\lambda = \beta/\delta$

Equation (2.5) assumed that all the infected hosts are infective. This is not so since the infected host becomes infective after about 9 months from infection date. To therefore take care of this early case, a modification of equation (2.5) is necessary. Thus, for the fraction of the infected population that is infective, we shall have this as.

$\in = bI$, so that $I_1 = (1- \in)I$ where I_1 is the infected that is infective. Similar model as above can be used to describe the population change in this class.

Finally, equation (2.6) is a very simple first order differential equation which when solved yields

$$R(t) = \delta I t \tag{2.10}$$

We can see here that $R(0) = 0$ and over some time range,

$$R(t) = \sum_{t=0}^{\infty} \delta I t \tag{2.11}$$

since the system is not a continuous one.

ANALYSIS ON THE HOST'S INFECTIVITY

We have developed the model equations on the population stratification/changes in the host. In this part of the work, we have to establish the condition for the sustenance of the Onchocerca Volvulus in the host populations.

For the susceptible hosts to be infected, there must be interaction between the infected host with the susceptible vector and also the infected vector with the susceptible host.

Now let the total population of the host be n and let the infected in this population be I and mortality rate as σ. For the vector, let $n^{/}$ be its total population with the infected $I^{/}$ and mortality rates as $\sigma^{/}$ and $\overline{\sigma}^{/}$ where $\sigma^{/}$ is the natural death rate of the vector and $\overline{\sigma}^{/}$ is the death rate of the vector as a result of quest for blood meal.

In the population, the rate at which the susceptible contacts the disease must be greater than the rate at which the infected are removed from the entire population if the disease must spread or be maintained in the populations. Therefore;

$$\beta SI^{/} > \delta I \qquad \text{for the host} \qquad (2.12)$$

$$\beta^{/} S^{/} I > \frac{ai}{4}\left(\sigma^{/} + \overline{\sigma}^{/}\right) I^{/} \qquad \textit{for the vector}$$

where S is the susceptible class in the host populations with n = S + I + R

From equations (2.12), we have:

$$\beta S I' > \delta \frac{ai\,(\sigma' + \overline{\sigma}')\, I'}{4\beta' S'} \qquad (2.13)$$

that is

$$SS' > \delta \frac{ai\,(\sigma' + \overline{\sigma}')\, I'}{4\beta\beta' I'} = \delta \frac{ai\,(\sigma' + \overline{\sigma}')}{4\beta\beta'}$$

Equation (2.13) must be maintained in the two populations for the disease resulting from the spread of Onchocerca Volvulus by the host and vector to also be maintained or build up. Equation (2.13) is what is then called the threshold requirement in the initial density of the susceptible for an epidemic outbreak.

In this equation (2.13) we used the susceptible populations because we assumed that there are infected populations such that it is only the susceptible class that ensures the continuity of this disease. Let us again derive the requirement for the epidemic to build up. Since both population reproduces, this increases the number of susceptible such that from the equation (2.4), (2.9) and (2.25), (2.27) we have

$$\left.\begin{aligned} \ln\left(\frac{S}{S_o}\right) &= \{\gamma S - \beta I'\}t \qquad (2.14)\\ R &= \delta\, It \qquad (2.15)\end{aligned}\right\} \text{ for the host}$$

and

$$\left.\begin{aligned} \ln\left(\frac{S'}{S'_o}\right) &= \{\lambda S' - \beta' S' I\}t \qquad (2.16)\\ R' &= \frac{ai}{4}\{(\sigma' + \overline{\sigma}')I' + \xi S'\}t \qquad (2.17)\end{aligned}\right\} \text{ for the vector.}$$

If we divide equation (2.14) by equation (2.17) and equation (2.16) by equation (2.15), we then have

$$\ln\left(\frac{S}{S_o}\right) = \frac{4R'\{\gamma S - \beta I'\}t}{ai\{(\sigma' + \bar{\sigma}')I' + \xi S'\}t} = \frac{4R'\{\gamma S - \beta I'\}}{ai\{(\sigma' + \bar{\sigma}')I' + \xi S'\}} \tag{2.18}$$

and

$$\ln\left(\frac{S'}{S_o'}\right) = \frac{R\{\lambda - \beta' I\}t}{\sigma I t} = \frac{R\{\lambda - \beta' I\}}{\sigma I} \tag{2.19}$$

If we define the intensity of an epidemic as the population of the susceptible that can contract or did finally contract the disease, then since all such class must die off, then representing such intensities in both populations as I_o and I'_t, then

$$i_t = R_\infty \big/ n \qquad and \qquad I_t' = R_\infty' \big/ n \tag{2.20}$$

At $t = \infty$, we have the specification of the two populations as ($n - ni_t$, 0, ni_t) and ($n' - n' i'_t$, 0, $n' i'_t$) respectively.

Substituting equation (2.20) into equation (2.18) and (2.19), we have:

$$\ln(1 - i_t) = \frac{4i_t' n'\{\gamma n_1 - \beta I'\}}{ai\{(\sigma' + \bar{\sigma}')I' + \gamma S'\}} \qquad where \quad n_1 = S + I \tag{2.21}$$

and

$$\ln(1 - i_t') = \frac{i_{tn}\{\lambda - \beta' I\}}{\sigma} \tag{2.22}$$

Expanding the left hand side of equations (2.21) and (2.22) and taking only the first two terms and then multiplying them together, we have:

$$(i_t + \tfrac{1}{2} i_t)(i_t' + \tfrac{1}{2} i_t^i) = \frac{4i_t' n'\{\gamma n_1 - \beta I'\}}{ai\{(\sigma' + \bar{\sigma}')I' + \gamma S'\}} \times \frac{i_{tn}\{\lambda - \beta' I\}}{\sigma}$$

This equation describes the intensity of the disease in the host due to the intensity of the disease in the Vector.

Case 1: All the host population to be infected.

This will only occur if $\beta' I_1 + b' - \sigma' - \gamma$ will be very large. This implies that $\beta' I_1 + b' > \sigma' + \gamma$. In the Vector population, $\sigma' + \gamma$ is no far greater than $\beta' I_1 + b'$ since σ' which is the natural death rate of the vector is very high because of non-ready availability of blood for the Vector. As well, γ is large because of the measures taken by the susceptible host to protect himself from the biting by the Vector. Therefore, $\beta' I_1 + b'$ is not far larger than $\sigma' + \gamma$.

Let $1/(\beta' I_1 + b' - \sigma' - \gamma) = \alpha$. Then,

$$S(t) = \left[S_0^{\frac{1}{\alpha}} + S'^{(\beta I' - (b-d))} - S_0^{/(\beta I' - (b-d))} \right]^{\alpha}$$

For any given time t, $\left[S_0^{(\beta' I_1 + b' - \sigma' - \gamma)} - S_0^{/(\beta I' - (b-d))} \right]$ is constant so that we can write it as A. Thus,

$$S(t) = \left[A + S'^{(\beta I' - (b-d))} \right]^{\alpha}$$

In the same line of argument, $\beta I' + d - b > 0$ for the susceptible population to be maintained. This implies that $\beta I' + d > b$. We know naturally that $b > d$ for the population to be maintained so that this implies that $\beta I' > b - d$. Hence, new susceptible Vector must be recruited into the population so that the entire host population may be infected.

Case II: No further infection in the Susceptible population of the Host.

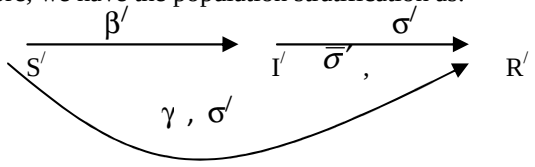
This implies that there should be no further recruitment of the susceptible vector in times to come so that after some time,

$$\beta I^{/} = b - d \qquad \text{so that } I^{/} = (b - d)/\beta.$$

Since b – d is almost constant, we require β to be very large so that $I^{/} \approx 0$. This means that there will also be no infected vector which will transfer the disease to the available susceptible host if they exist. The essence of this is to see how river blindness can be controlled. Report shows that there has been tremendous success in the fight against river blindness in African and beyond, [14] and [8]. Now by β being very large, we mean that the contact rate will be very large. This implies that all available susceptible population will contract the disease due to the high rate of contact between the susceptibles and the infectives if the infected vectors still exist. However, the only way that no further infection is noticed in the susceptible population is that b ≅ d and we know this is achievable with medical control.

2.1. The Vector

Like in the host population, the vector population is classified into the susceptible, infected and removed classes. Once the vector is infected, it never recovers or become immuned. In this case therefore, removal from the entire population means death. For survival of the vector (in particular the females), blood meal from the host is very necessary. When the vector takes a blood meal, it does not come back for another meal until at least 2 - 3 days during which it oviposites. It comes back for another blood meal after oviposition as this is necessary for activation
of another egg development for another oviposition . The blood meal taken by the vector induces mortality on it apart from the natural mortality. This induced mortality is as a result of the microfilariae ingested from the host in the process of taking a blood meal and this interferes with the body mechanisms.
Also, while trying to take blood meal, the vector is exposed to accidental death induced by the host in trying to prevent itself from being bitten by the vector. Generally, therefore, the blood meal and the process of obtaining blood meals induce mortality (death) on the vector apart from the natural mortality.
Since only the female vector sucks blood, our vector of interest is therefore the female Similium Damnosium. If those female vectors fail to obtain a blood meal after 2-3 days, they die off. With these facts therefore, we have the population stratification as:



where $\sigma^{/}$ = natural mortality rate, $\bar{\sigma}'$ = is the mortality induced by the process of obtaining blood meal and the blood meal itself; $\beta^{/}$ = is the biting rate of the vector and thus, the infection rate, γ = is the mortality induced by the process of obtaining a blood meal by the susceptible.
Since the vector can be infected only if it takes a blood meal form an infected and infective host and if we denote this host by I_1, then the number of new infection occurring in the vector's population is given as

$$\beta^{/} S^{/} I_1 \Delta t \tag{2.23}$$

Similarly, the number of removal occurring in the vector population over the time period is given as

$$[(\sigma' + \bar{\sigma}')I' + \gamma S']\Delta t \tag{2.24}$$

Since the vector, in the abundant supply of blood meal, oviposites up to six times
(Schulz-key & Karam, 1988) , (Schulz-key 1990) within a short interval of time (about 21 days), it means that a large number of susceptible are being added to the population.

The Model equations on Vector Population Change

The change in the susceptible Vector population over time is given as:

$$\begin{aligned} \frac{dS}{dt} &= -\beta' S' I_1 + (b - \sigma')S' - \gamma S' \\ &= (-\beta' I_1 + \rho)S' \qquad \text{where} \quad \rho = b - \sigma' - \gamma \end{aligned} \tag{2.25}$$

What equation (2.25) represents is the change in the susceptible vector population. But we know that for an infected vector to become infective, we need about six days for the microfilariae picked along with the blood meal to mature into a larva before it is given back to another or the same host. Thus, the infected susceptible that are infective will now be $(1-\epsilon)\,I_1$ where ϵ is some constant far less than 1.
The change in the infected vector population over a period of time is given as:

$$\frac{dI'}{dt} = \beta' S' I_1 - (\sigma' + \bar{\sigma}')I' \tag{2.26}$$

where I_1 = infected and infective vector.
Incorporating the four weather conditions (as stated below) into this, we have

$$\frac{dI'}{dt} = \beta' S' I_1 - \frac{ai(\sigma' + \bar{\sigma}')}{4} I' \tag{2.27}$$

Solutions to the Modelled Equations

Solving equation (2.25), we obtain

$$S'(t) = A e^{(-\beta' I_1 + \rho)t}$$

At t = 0, $S^{/}(0) = S^{/}_o = A$ and thus

$$S'(t) = S^{/}_o e^{(-\beta' I_1 + \lambda)t} \tag{2.28}$$

Weather plays important role in the population dynamics of the vector. Thus we graduate the year into 4 parts within which certain population increment level is noted. Therefore, to effectively reflect the appropriate population changes at these periods, the model and even the solution need to be modified. Thus, taking weather into consideration, equation (2.28). will be modified as

$$S'(t) = \frac{ai}{4} S^{/}_o e^{(-\beta' I_1 + \lambda)t} \tag{2.29}$$

where i = 1,2,3,4, reflecting the four weather conditions and **a** is some constant used for appropriate population reflection for the weather periods and thus differs for each period.

Also solving the equation (2.26), we obtain

$$\begin{aligned} I'(t) &= \frac{\beta'}{\frac{ai}{4}(\sigma' + \bar{\sigma}')}\left[S' I_1 - S^{/}_o I_{10} e^{-\frac{ai}{4}(\sigma' + \bar{\sigma}')t} \right] + I^{/}_o \\ &= \delta\left[S' I_1 - S^{/}_o I_{10} e^{-\frac{ai}{4}(\sigma' + \bar{\sigma}')t} \right] + I^{/}_o \qquad \textit{where} \quad \delta = \frac{\beta'}{\frac{ai}{4}(\sigma' + \bar{\sigma}')} \end{aligned} \tag{2.30}$$

2.2 Number Of Microfilaria Ingested Per Meal By A Vector.

Now, let us design a Mathematical model that can help us to determine the number of the microfilariae that are ingested along with the blood meal where we assume that the vector is fully grown and the variation in the quantity ingested is not dependent on the size of the vector.. To do this, we note that the number is dependent on the (a) age of the host (b) density of the microfilaria (Mf) in the part of the host that was bitten by the fly in order to get a blood meal (c) season or period of the year (d) the exposure level, ([2] and [4]).
We shall recall that if the host has been in the endemic area of the Onchocerciasis disease for a long time, he must have quite good concentration of the Mf in his body. Thus, the age of the host will be considered in terms of length of time of domicile and exposure in the area and this is a very important factor. The

number of the Mf picked at each meal will highly depend on the location of the body since according to [1] and [16], different parts of the body of the host have different concentration of the Mf. Even though different period of the year presents different inoculation levels, the Mf concentration in the body is appropriately adjusted to the required level both by the host's immunity and the embedding Onchocerca worms. Thus, for good reason, we may neglect this factor in designing the model. Finally, the exposure level greatly affects the level of Mf picked. In general therefore, apart from the above factors mentioned, the change in the level of Mf picked is a function of the exposure level of the host available to the vector.

The Model

Now, let A = age of the host (or length of residence of the host), D = density of the Mf in the part of the body of the host that is of interest, [16], P = exposure level of the host and N = number of Mf picked. Then change in the number picked due to change in the exposure level is given as

$$\frac{dN}{dp} = \gamma AD \Rightarrow N(t) = \gamma ADp = \xi p \tag{2.31}$$

$$where \quad \xi = \gamma AD \quad \text{is constant for a particular host at a particular site}$$

Note that change in the number of Mf picked is not necessarily a function of time but rather on exposure level. Now, let us consider the number of this picked Mf that matures into a larva and then inoculated back to the host by the vector in the subsequent taking of blood meal.

2.3 The Number Of Mature Larva Innoculated Back To A Host By A Vector.

Reports from researchers on Onchocerciasis ([2], [6], [11] and [15]) showed that the vector picks many of the Mf when they bite the host to suck blood and that only about 50% of this number actually develops into the larval stage of the vector. It was reported that the peritrophic membrane secreted by the vector on the blood meal reduces the mobility of the Mf and thus the number that migrates to the thoracic muscles of the vector where they molt. However, the number of Mf that can develop in the vector is put at about 20-30. [9] and [10] calculated that about 4.2 larva can be developed at any blood meal, which is the number of larva, that can be inoculated into the host. He however stated that this number may be on the lower side since some of the vectors have been taking blood meals for some time although there is no significant difference in the number due to number of times blood meals have been taken.

The Model

Based on these and other facts, we have the factors determining the number of larva inoculated back to the host on being bitten by the vector given that the vector had earlier taken a blood meal as:

(1) Vector's immune systems
(2) Number of Mf ingested
(3) Time
(4) Season of the year

Now let L = number of Larvae inoculated
v = vector's immunity, N = number of Mf ingested
and t = time, then

$$L = \varepsilon \nu N \tag{2.32}$$

where ε is a constant

Then
$$\frac{dL}{dt} = \varepsilon \nu \frac{dN}{dt} \tag{2.33}$$

where v is assumed constant

But
$$\frac{dN}{dt} = \frac{dN}{dp}\frac{dp}{dt} = \gamma AD\frac{dp}{dt}$$

$$\frac{dL}{dt} = \varepsilon\, v\gamma AD\,\frac{dp}{dt}$$

$$= \tau\, vAD\,\frac{dp}{dt} \qquad where \quad \tau = \varepsilon\,\gamma \tag{2.34}$$

:.

Equation (2.34) shows that change in the number of Larva inoculated back to the host with respect to time is a function of the change in the exposure level also over time.

For the removed class in the population stratification, the change in the population over time is given as

$$\frac{dR'}{dt} = (\sigma' + \bar{\sigma}')I' + \xi S' \qquad where \quad \xi' = \gamma + \sigma' \tag{2.35}$$

If the weather factor is introduced, we have

$$\frac{dR'}{dt} = \frac{ai}{4}\left[(\sigma' + \bar{\sigma}')I' + \xi S'\right] \tag{2.36}$$

The Solution

Solving equation (2.36) yields

The total population of the vector is n = S + I + R so that

$$R'(t) = \frac{ai}{4}\left[(\sigma' + \bar{\sigma}')I' + \xi S'\right]t \tag{2.37}$$

$$\frac{dn'}{dt} = \frac{dS'}{dt} + \frac{dI'}{dt} + \frac{dR'}{dt}$$

$$= -\beta' S' I_1 + \gamma S' + \beta' S' I_1 - \frac{ai}{4}(\sigma' + \bar{\sigma}')I' + \frac{ai}{4}\left[(\sigma' + \bar{\sigma}')I' + \xi S'\right]$$

$$= \gamma S' + \frac{ai}{4}\xi S'$$

$$dn' = \left(\gamma + \tfrac{ai}{4}\xi'\right)S'dt \qquad \Rightarrow \qquad n' = \left(\gamma + \tfrac{ai}{4}\xi'\right)S't + C$$

:.

At t = 0, $n' = n_o'$ so that $n'(t) = n_o' + \left(\gamma + \frac{ai}{4}\xi'\right)S't$

Changes in the Susceptible population of the Host

We consider here the special case of the variation in the susceptible population of the host as a function of the susceptible population of the Vector.

If we consider the variation of the susceptible population of the host due to a variation in the population of the susceptible Vectors , equation (2.1.3) , we have that

$$\frac{dS}{dS'} = \frac{(-\beta I' + m)S}{(-\beta' I_1 + \xi)S'} \qquad where \quad \xi = b' - \sigma' - \gamma \quad and \quad m = b - d$$

Solving this equation, we have that

$$S(t) = \left[S_0^{(\beta' I_1 + b' - \sigma'' - \gamma)} + S''^{(\beta I' - (b-d))} - S_0'^{(\beta I' - (b-d))}\right]^{\frac{1}{\beta' I_1 + b' - \sigma'' - \gamma}}$$

This expression gives the susceptible population of the host available at any time as a function of both the susceptible population of the Vector and the initial susceptible population of the host. A look at the solution shows that the more we have the vectors around, the more they infect the Host and thus the more we loose the susceptible Hosts.

In this paper therefore, we have been able to mathematically study the population dynamics of the Host and the vector. In particular we have derived formulae that can be used to estimate the number of microfilaria that are ingested by a particular blackfly after a blood meal and the subsequent quantity of the microfilaria that is now in the larva stage that will finally go back to another or the same host as the fly goes back for another blood meal after some 2 – 3 days.

These models will be very useful in the preventive mechanism of the disease spread. A check on the vector by reducing the immunity of the vector due to the effect of the microfilaria ingested during the blood meal can go a long way to exterminating the vectors and thus the disease.

REFERECES

[1] Akoh, J. I, Adeboke, D. A and Okolo, C.J (1993): Atomic site biting preferences by Similium Damnosum complex in the Northern Guinea Savanna Zone of Nigeria.

[2] Basanez, M.G; Remme, J.H.F., Alley, E.S. et al (1995): Density dependent processes in the transmission of human Onchocerciasis: Relationship between the number of microfilaria ingested and successful laval development in the Simuliid Vector. Parasitology, 110: 409 - 429

[3] Buck. A. A (1974): Onchocerciasis. Symptomatology, Pathology, Diagnosis. Geneva, World Health Organization.

[4] Dietz, K (1988): Density-dependence in parasite transmission dynamics. Parasit. Today; 4: 91 - 97

[5] Duke, B.O.L. (1962): Study on factors influencing the transmission of Oncho. II. The intake of Onchocerca Volvulus microfilaria by Similum Damnosum and the survival of the parasites in the fly under laboratory conditions. Ann trop. Med. Parasite. 56, 255

[6] Duke, B. O. L. and Lewis, D.J (1964): Studies on factors influencing the transmission of Oncho III. Observations on the effect of the peritrophic membrane in limiting the development of Onchocerca Volvolus microfilaria in Similum Damnosum. Ann. Trop. Med. Parasite. 58(1), 83-88.

[7] Duke, B.O.L (1967): Infective filarial larvae other than Onchocerca Volvolus in similium Damnosum. Ann. Trop. Med. Parasit. 65, 200.

[8] Duke, B.O.L. (1968) The effects of drugs on Onchocerca Volvolus., J. Methods of assessment, population dynamics of the parasite and effects of Diethylcarbamazine. Bull. Of Wld Hlth Org. 39, 137 - 146

[9] Duke, B.O.L (1968a) The biting cycles, infective biting density and transmission potential of fresh similium Damnosum. Ann. Trop. Med. Parasit. 62, 95

[10] Duke, B.O.L (1968b) The stages of Oncho vol. In wild forest. Similar Dam. The fate of the parasites in the fly and the age distribution of the biting population. Annal. Trop. Med. Parasit. 62. 107.

[11] Duke, B.O.L (1969c) The infective biting potential of similium Damnosum in different bioclimatic zones and its influence on the transmission potential. Ann. Trop. Med. Parasit. 62.

[12] http://usembassy.state.gov/Nigeria/wwwhp/20902a.html. The United State Diplomatic mission to Nigeria, Dc. 9th, 2002.

[13] http://www.alertNet.org/the facts/reliefresources/473576.htm Reuters AlertNet SSI: fighting the scourge of river blindness, 08 July, 2002.

[14] http://www.oneworld.org/ips2/feb99/1120-020.html Africa: Remarkable success in the fight against river blindness. World News Inter Press Service (IPS)

[15] Lewis, D.J (1950): A peritrophic membrane in similium. Nature (London). 165, 978.

[16] Mbah, G.C.E (2007): Estimation of the Microfilaria Load in a Host using the Microfilaria Count in a Skin- Snip Sample taken from any other part of the body of the Host below the Illiac Crest. (Accepted by Global Journal of Pure and Applied Sciences)

[17] Nelson, G.S (1970) Onchocerciasis. Adv. Parasit. 8, 173-224. Schulz-key and Karam (1988): Periodic reproduction of Onchocerca Volvolus Parasitology today Vol. 2 pp 284 – 286.

[18] Schulz-key and Karam (1988): Periodic reproduction of Onchocerca Volvolus Parasitology today. Vol. 2 pp 284 – 286.

[19] Schulz-key (1990) Observant on the reproduction biology of Onchocerca Volvolus Actor Leidensia 59 No 182, 27-43.

[20] WHO (2009): World Health report on Onchocerciasis disease in Africa