

Age-Structured Epidemic Model for Transmission Dynamics of Tuberculosis

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Abstract: We proposed an age structured transmission dynamics model for the transmission of Tuberculosis, along the line of the classical Mckendrick-Foerster, age-structured population models based on some assumptions contrary to the Castillo-Chavez assumptions of age depended mortality rate and age depended contact rate, vaccination and treatment rates of the infected. Rather, our model assumes constant contact rate, no vaccination and treatment, infectives are removed and quarantine till non- disease induced death. We then examined the existence of non trivial steady states of the model and discussed their stability via the basic reproductive number of the disease. Also the equations for the population compartments are obtained.

Key words: Transmission dynamics model, mortality rate, stability, population compartments, vaccination

INTRODUCTION

Research on Tuberculosis transmission dynamics is currently intensified, aimed at providing preventive, management and control strategies for victims and nonvictims of the diseases. Some of these research focuses on structured population along the line of the classical Mckendrick-Foerster structured population model as in the work of Castillo-Chavez (2004), Inaba (1990), Mats (2002) and non structured homogeneous population model, as in work of Castillo-Chavez (2004). However there have been several representations of the vital dynamics for the age structured model, which are extension of the classical Mckendrick-Foerster age structured population model as in the work of Gurtin and McCamy, (Cushing, 1994), Michel Langlais (1991), Mat (2002). However in this research, we examined the transmission dynamic of Tuberculosis, in a proportionate mixing population, constant aver rage per capital contact rate and that the disease affect the mortality and reproductive rates of the removed individuals.

THE MODEL EQUATION

The following parameters are defined as,

S(t, a): Population density of the susceptibles of age a . at time t.

I (t, a) : Population density of the infected individuals

of age a, at time t.

R (t, a) : Population density of the removed individuals of age a, at time t.

 β (a) : Fertility rate at age a.

μ (a) : Natural mortality rate at age a.

: Removal/progression rate to active

Tuberculosis.

: Average (per capital contact rate)

Consider an SIR epidemic model; where there is a constant recruitment into the population and natural mortality rate not age depended, the force of infection is of mass action, or proportionate mixing. Assume that the disease is not vertically transmitted to the new born by infected mothers and that, removed individuals are no longer sexually active since they are in their active stage of Tuberculosis and don't contribute to reproductive process. Assume that the disease transmission occur according to the proportionate mixing assumption,

$$S(t,a)\beta(a)c\int_0^\infty \frac{I(t,a)}{n(t,\overline{a})}k(t,\overline{a})d\overline{a}, \text{ where } k(a,\overline{a})=\ k(t,a,\overline{a})=k(t,\overline{a})$$

is the interaction coefficient, defined as the rate at which an average susceptible individual with age a, has contact with an individual of age at time t, defined in line with Inaba (1990) and Castillo-Chavez (2004), as

$$k(t, \overset{-}{a}) = \frac{n(t, \overset{-}{a})}{\int_0^\infty n(t, a) da}$$

 β (a), is the age specific (average) probability of becoming infected through contacts with infectious individual, c is the specific per capital contact/activity rate. The force of infection is.

$$\beta(a)c \! \int_0^\infty \! \frac{I(t, \overset{-}{a})}{n(t, \overset{-}{a})} k(t, \overset{-}{a}) \! d\overset{-}{a}$$

All newborn babies from both compartments, (susceptibles and infectives) per unit of time, are susceptibles and obtained by,

$$S(t,0) = N\mu$$

$$I(t, 0) = R(t, 0) = 0$$

Where μ is the natural mortality rate.

Using these assumptions and definitions we have the Mckendrick-Von Foerster type age-structured epidemics model for the population compartments of the heath states, as,

$$S_t(t,a) + S_a(t,a) = -\mu S(t,a) - \beta(a)c\eta(t)S(t,a)$$

$$I_{x}(t,a) + I_{x}(t,a) = -\mu I(t,a) + \beta(a)c\eta(t)S(t,a) - \psi I(t,a)$$
 (1)

$$R_{+}(t,a) + R_{a}(t,a) = -\mu R(t,a) + \psi I(t,a)$$

With the initial conditions, $S(0, a) = S_0(a)$, $I(0, a) = I_0(a)$ $R_0(a)$ and boundary condition, $S(t, 0) = N\mu$, I(t, 0) = R(t, 0) = 0 n(t, a) = n(t, a) = S(t, a) + I(t, a) + R(t, a),

Where
$$\eta(t) = \int_0^\infty \frac{k(t, \bar{a})}{n(t, \bar{a})} I(t, \bar{a}) d\bar{a}$$
 is the probability for

a susceptible to become infected by contact with an infected individual. Let us assume that the population is in an equilibrium state, so that its size and age-distribution are independent of time. This is only possible if $R_0=\int_0^\infty \beta(a)P(a)da^{=1}$. Where, $P(a)=e^{-\mu a}$ P (a) is the probability that an average individual survive up to age a, simply called the survival function. The equilibrium age density is obtained as, $n(a)=\frac{NP(a)}{L}=\mu*NP(a)$, (Inaba, 1990), where N is the stationary population size and $L=\int_0^\infty P(a)da=\frac{1}{\mu}$, is the life expectancy. The Crude death rate μ^* , satisfies $\mu*\int_0^\omega P(a)da=1$, (Horst and

Finaba, 1990).

Adding up the three equations gives the Mckendrick-, Foerster age-structured population model,

$$n_{+}(t,a) + n_{a}(t,a) = -\mu(a)n(t,a)$$

$$n(o,a) = n_0(a)$$

$$n(t,0) = N\mu$$

Thus, the steady demographic state of the Eq. in 2 is obtained as,

$$n(a) = N\mu e^{-\mu a} = S(a), I(a) = R(a) = 0$$

Under similar assumptions, but with treatment of the infectives and a constant average (per capital contact rate), Castillo-Chavez (2004) obtained the probability that

an individual of age $\delta + \tau$, who was infected with Tuberculosis τ units of time ago and still in class i, as

$$\begin{split} \text{where,} \quad & \frac{1}{\tau - \Psi} (e^{-(r_t + \psi)} e^{-(r + \mu)(\tau - u)} du = \alpha(\tau) e^{-\mu \tau} \quad , \end{split}$$

r is the treatment rate for the infected and Ψ is the progression rate to active Tuberculosis. Extend ding Castillo-Chavez (2004), definition to this problem, we have, $\gamma(\tau, \delta) = \int_{-\tau}^{\tau} e^{-(\mu^{t}+\psi)} e^{-\mu(\tau-\psi)} du = \alpha(\tau)e^{-\mu\tau}$,

where $\omega\left(\tau\right) = (1 - e^{-\nu\tau})$. The infectivity function $A\left(\tau, \epsilon, \eta\right)$ is obtained as, $A\left(\tau, \alpha, \delta\right)$, $= \beta(a)cp(\delta + \tau)\frac{\gamma(\tau, \delta)}{n(\delta + \tau)}$

(Castillo-Chavez, 2004) and reduced to the form, $A(\tau, \alpha, \delta) = f(a) g(\tau, \delta)$ using proportionate mixing assumption, (Castillo- Chavez, 2004), Where, $f(a) = \beta(a)c$, $g(\tau, \delta)$

$$= \ p(\delta + \tau) \frac{\gamma(\tau, \delta)}{n(\delta + \tau)} \ p\left(a\right) = \frac{n(a)}{\int_0^\infty n(a) da} \ .$$

The basic reproductive number is then defined in terms of this parameter as, $R_0 = \int_{\Sigma} \int_0^{\infty} A(\tau, \epsilon, \delta) S(\epsilon) d\tau d\delta$, (Castillo-Chavez, 2004; Diekmann *et, al.*, 1993).

EXISTENCE OF NON TRIVIAL STEADY STATES

Let $s^*(a)$, I^* (a) and η^* be the steady demographic states, (the state where infection is absent) then, the following equations holds,

$$\frac{ds^*(a)}{da} = -\mu(a)s^*(a) - \beta(a)c(a)\eta^*s^*(a), \quad a > 0$$

$$s*(0) = N\mu$$

$$\frac{d i^*(a)}{da} = -\mu(a)i^*(a) - \beta(a)c(a)\eta^*s^*(a) - \psi i^*(a) \qquad a > 0$$

$$i*(0) = 0$$

$$\eta^* = \int_0^\infty k(a)i * (a)da$$

Solving these equations leads to the steady states,

$$s * (a) = N \mu e^{-\mu a}$$

$$i * (a) = \eta * H(a) \int_0^a \beta(r) c(r) F(r) dr$$

$$F(a) = e^{*\int_0^\sigma \beta(r) c(r) dr}, H(a) = P(a)F(a) = e^{-\mu a} \, e^{-\int_0^a \Psi(r) dr}$$

If $\eta^*=0$, then the steady Demographic state or the disease –free steady state is obtained, while $n^*\neq 0$, gives the endemic states, $(s^*(\sigma), s^*(\sigma))$. Thus the non trivial steady states exist. The global and local stability of these states is examined via the basic reproductive number, as in (Murray, 1989; EL-Doma, 2004; Herffernan et al., 2005; Inaba, 1990). Using the representations in Castillo-Chavaz, (2004) for the infectivity function $A(\tau,\epsilon,\eta)$, we get the basic reproductive number of the infection as, $R_0=c\int_0^\infty \int_0^\infty B(\delta)g(\tau,\delta)S(\delta)d\tau d\delta$,

where
$$\begin{split} g(\tau,\delta) &= \frac{1}{N} \gamma(\tau,\delta) \text{ and } S(a) = \text{ N} \mu e^{-\mu a} \text{ , we have,} \\ \mu c \int_0^\infty \int_0^\infty B(\delta) e^{-\mu \tau} (1-e^{-\psi \tau}) e^{-\mu \delta} \mathrm{d}\tau \mathrm{d}\delta \quad \text{where } \ P(\delta) = e^{-\mu \delta} \;. \end{split}$$

Thus,
$$R_0 = \frac{\psi c}{\psi + \mu} \int_0^\infty \beta(\delta) e^{-\mu \delta} d\delta \quad \text{. For a stable disease}$$
 -free state, we must have
$$R_0 < 1 \text{. This is possible when,}$$

$$\int_0^\infty \beta(\delta) e^{-\mu \delta} d\delta < \frac{\psi + \mu}{\psi c} \quad P(\delta) = e^{-\mu A} \quad \text{or}$$

$$\int_0^\infty \beta(\delta) e^{-\mu \delta} d\delta < \frac{1}{c} + \frac{\mu}{\psi c} \quad \text{. Let us assume that } \mu < \Psi c \text{ and}$$

c >1, such that, $\frac{\mu}{\mu}$ is negligible to bring it line with the

definition of stability of the diseases-free steady state as in, Murray (1989), Diekmann *et al.* (1993). That is, the disease-free equilibrium state is stable if $R_0 = \int_0^\infty \beta(\delta) P(\delta) d\delta < 1$. We would have greater rate of progression to active Tuberculosis, than death from natural mortality and higher number of contacts per unit of time. Otherwise the disease-free steady state is unstable. Integrating (1) along characteristics lines, we get

$$\begin{split} S(t,a) &= S_0(a-t)e^{-c\mu\int_{x_t}^a\beta(a)\eta(t)da}, \text{ if } \quad a>t \\ &= S(t\text{-}a,0)e^{-c\mu\int_x^a\beta(a)\eta(t)da}, \text{ if } a$$

 $F(a) = e^{\mu a}, k(a) = S(t,a)$

the equation for the population density as,

$$\begin{split} I(t,a) &= p(a)G(a)(I_0(a) + c \int_{a-t}^a \beta(a)\eta(t)P(a)G(a)k(a)da, \\ if \ a > t \\ &= \ e^{-(\mu-\psi)a}I_0(a-t) + \\ c \int_{a-t}^a \beta(a)S_0(a-t)e^{-(\mu-\psi)a}e^{-c\mu \int_{a-t}^a \beta(a)\eta(t)da} \eta(t)da, \\ &= \ e^{-(\mu+\psi)a}I_0(t-a,0) + \\ c \int_{a-t}^a \beta(a)S(t-a,\ 0)e^{-(\mu+\psi)a}e^{-c\mu \int_0^a \beta(a)\eta(t)da} \\ \eta(t)da, & if \ a < t \end{split}$$
 Where $P(a) = e^{-\mu a}, \quad G(a) = e^{-\psi a},$

CONCLUSION

We have seen that stability of the disease-free equilibrium depends on the magnitude of the basic reproductive number of the disease and this holds if, $R_0 < \frac{\Psi + \mu}{\text{wc}} \quad \text{where } \Psi \text{, is the progression rate to active}$

Tuberculosis and μ is natural mortality rate assumed equal for all compartments. Since, we have assumed $\frac{\mu}{\psi c} < 1$, we

have $\mu < \psi c$ and $\psi > \frac{\mu}{c}$ Thus, the threshold conditions

for a stable disease-free steady state is, $c \geq 1$ and . $\frac{\mu}{c} < \psi$

Otherwise we will have an endemic disease state and the infection will persist in the population.

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