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AN SIRS EPIDEMIC MODEL OF JAPANESE ENCEPHALITIS

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ABSTRACT. An epidemiological model of the dynamics of Japanese Encephalitis (J.E.) spread coupling the SIRS (Susceptible/Infected/Removal/Susceptible) models of J.E. spread in the reservoir population and in the human population has been proposed. The basic reproductive rate $R(0)$ in the coupled system has been worked out. Using Aron's results (cf. [1] and [2]), it has been observed that the disease-free system is stable in this coupled system also, if $R(0)$ is less than unity, and if $R(0)$ is greater than unity, the disease-free system is unstable and there exists a unique stable endemic equilibrium.

The model also shows that in contrast to Aron's observations, loss of immunity is independent of the rate of exposure to the disease. This observation sheds light on the control measure of J.E. by vaccination. Passive immunization, i.e., administration of antibody at recurrent intervals is the correct method of vaccination to eradicate the disease.

KEY WORDS AND PHRASES. SIRS model, Japanese encephalitis, basic reproduction rate, stability analysis, control measure.
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1. INTRODUCTION.

Japanese Encephalitis (J.E.) is a mosquito borne disease where infection is transmitted from reservoir population (pig, cattle, equine, bird, etc.) to susceptible human population through a particular species of mosquito (Culex Vishnui). Man is the dead end of infection and as such harboring of infection from man to man is not possible. Immunity in both reservoir and human populations appear to be sustained by continual exposure. The present paper investigates into the epidemiological effect of boosting immunity in J.E. and the qualitative dynamics of the epidemiological model. Since transmission from man to man does not occur, man does not act as a carrier in J.E. On the other hand, the reservoir population (infective and immune carriers) not exhibiting the clinical symptoms, act as active hosts permitting transmission to both animal and

human susceptibles. Thus in J.E. some proportion of immune reservoir population also acts as infective (active carrier), and the constraint that the degree of infectivity reduces with the increase of immunity status in them has been incorporated in this model following Aron (cf. [1] and [2]).

2. MATHEMATICAL MODEL OF J.E.

Both human and reservoir populations are classified into three categories, namely susceptible, infected and removal classes. Let x_1, x_2 and x_3 be the proportion of susceptibles, infected and removed respectively in human population and y_1, y_2 and y_3 be those for reservoir populations. The removed class includes both recovered (immune) and dead by J.E. The dynamical system representing the epidemic spread in human and reservoir populations are then given by the following rate equations

$$\begin{aligned}
\frac{dx_1}{dt} &= \mu_1 - \mu_1 x_1 - h_1 x_1 + f_1 x_3 \\
\frac{dx_2}{dt} &= h_1 x_1 - \mu_1 x_2 - \gamma_1 x_2 \\
\frac{dx_3}{dt} &= \gamma_1 x_2 - \mu_1 x_3 - f_1 x_3 \\
\frac{dy_1}{dt} &= \mu_2 - h_2 y_1 - \mu_2 y_1 + f_2 y_3 \\
\frac{dy_2}{dt} &= h_2 y_1 - \mu_2 y_2 - \gamma_2 y_2 \\
\frac{dy_3}{dt} &= \gamma_2 y_2 - \mu_2 y_3 - f_2 y_3
\end{aligned} \tag{2.1}$$

where

$$\begin{aligned}
x_1(t) + x_2(t) + x_3(t) &= 1 \\
y_1(t) + y_2(t) + y_3(t) &= 1
\end{aligned} \tag{2.2}$$

and

$$h_1 = \beta_1 (k_1' y_1 + k_1'' y_3) x_1$$

is the effective exposure rate for man and

$$h_2 = \beta_2 (k_2' y_2 + k_2'' y_3) \tag{2.3}$$

is the exposure rate in reservoir population

$$0 \le k_1'' \le k_1' \le 1; \quad 0 \le k_2'' \le k_2' \le 1.$$

μ_1 is the birth and death rate in man, so that the population size remains the same. β_1 is the rate at which the human susceptibles become sick by mass action contact between susceptible man and infective reservoir populations. k_1' and k_1'' are the proportions of infected and immune reservoir respectively who are infective to man. Sick human individuals enter the removal class (recovered and dead) at the rate γ_1 and immune individuals (i.e., the recovered portion of the removal class x_3) become susceptibles at the rate f_1, f_1 being a function of h_1, and as derived by Aron [1] is

$$f_1(h_1) = \frac{(h_1 + \mu_1) e^{-(h_1 + \mu_1)\tau_1}}{1 - e^{-(h_1 + \mu_1)\tau_1}} \tag{2.4}$$

τ_1 being the unit of years in which immunity in man lasts unless reexposure occurs during that time interval. The function $f_1(h_1)$ is a monotonically decreasing function of h_1 (cf. [1]; see also

[2]). The rate constants μ_2, β_2 and γ_2 in reservoir population stand for the same connotations as for the corresponding rate constants in man. k_2' and k_2'' are the proportions of infected and immune reservoirs respectively who are infective to the reservoir population. We also have similar expression for $f_2(h_2)$ as in (2.4),

$$f_2(h_2) = \frac{(h_2 + \mu_2)e^{-(h_2+\mu_2)\tau_2}}{1 - e^{-(h_2+\mu_2)\tau_2}} \tag{2.5}$$

τ_2 is the unit of years in which immunity in the reservoir population lasts unless reexposure occurs during that time period.

It may be noted that h_1 involves second order term whereas h_2 involves first order term only. this is apparent from the mode of transmission of the disease which is unlike malaria. Transmission of J.E. in man takes place by the interaction of the susceptible human populations and infected or carrier reservoir population (mediated by vector population), whereas transmission of J.E. in reservoir population occurs by direct contact (through vector) amongst themselves.

Considering that the proportion of infective reservoir population effective for infecting the susceptible man is usually higher than or at most equal to that effective for infecting the reservoir population itself, we can assume $k_1' \geq k_2'$ and $k_1'' \geq k_2''$. Moreover, because of persistently boosted acquired immunity in the reservoir population, the transmission rates β_1 and β_2 satisfy the inequality relation, $\beta_1 \geq \beta_2$. Hence

$$\beta_1(k_1'y_2 + k_1''y_3) \geq \beta_2(k_2'y_2 + k_2''y_3). \tag{2.6}$$

Again, since h_1, the exposure rate in man can at most be equal to h_2, the exposure rate in the reservoir population, and also $x_1 \leq 1$, we can take as a particular case

$$\beta_1(k_1'y_2 + k_1''y_3)x_1 = \beta_2(k_2'y_2 + k_2''y_3)$$

$$i.e.,\ h_1 = h_2 = h. \tag{2.7}$$

When equality holds in (2.6) we have $x_1 = 1$, under which circumstances the disease process cannot start at all and hence, a requisite condition for the spread of the epidemic is that the inequality condition in (2.6) must be satisfied.

3. EQUILIBRIA OF MODEL.

For a particular h, the equilibrium values of y_2, y_3 and x_1, x_2, x_3 are

$$y_2 = \frac{h(\mu_2 + f_2(h))}{(\mu_2 + \gamma_2)(\mu_2 + f_2(h)) + h(\mu_2 + \gamma_2 + f_2(h))} \tag{3.1}$$

$$y_3 = \frac{h\gamma_2}{(\mu_2 + \gamma_2)(\mu_2 + f_2(h)) + h(\mu_2 + \gamma_2 + f_2(h))} \tag{3.2}$$

$$x_1 = \frac{(\mu_1 + f_1(h))(\mu_1 + \gamma_1)}{(\mu_1 + \gamma_1)(\mu_1 + f_1(h) + h) + hf_1(h)} \tag{3.3}$$

$$x_2 = \frac{h(\mu_1 + f_1(h))}{(\mu_1 + \gamma_1)(\mu_1 + f_1(h) + h) + hf_1(h)} \tag{3.4}$$

$$x_3 = \frac{\gamma_1 h}{(\mu_1 + \gamma_1)(\mu_1 + f_1(h) + h) + hf_1(h)} \tag{3.5}$$

and therefore

$$(k_1'y_2 + k_1''y_3)x_1 = \frac{h\{k_1'(\mu_2 + f_2(h)) + k_1''\gamma_2\}(\mu_1 + \gamma_1)(\mu_1 + f_1(h))}{\{(\mu_2 + \gamma_2)(\mu_2 + f_2 + h) + hf_2(h)\}\{(\mu_1 + \gamma_1)(\mu_1 + f_1(h) + h) + hf_1(h)\}} \tag{3.6}$$

= effective reservoir of infection for man.

Again from (2.2) and (2.6) we have

$$(k_1'y_2 + k_1''y_3)x_1 = \frac{h}{\beta_1}. \tag{3.7}$$

Now the equilibria exist when the two relations (3.6) and (3.7) are satisfied simultaneously and equilibria points are the points of intersection of the graphs of equations (3.6) and (3.7). Whenever $h = 0, y_2 = y_3 = 0$ and $x_2 = x_3 = 0$ and this is characterized by the disease free equilibrium. If $h \neq 0$, i.e., when disease is present, then following [2] we obtain the condition for an equilibrium as

$$R(h) = 1 \tag{3.8}$$

where

$$R(h) = \left[\frac{\beta_1\{k_1'(\mu_2 + f_2(h)) + k_1''\gamma_2\}}{(\mu_2 + f_2(h))(\mu_2 + \gamma_2) + h(\mu_2 + f_2(h) + \gamma_2)}\right] \times \left[\frac{(\mu_1 + \gamma_1)(\mu_1 + f_1(h))}{(\mu_1 + f_1(h))(\mu_1 + \gamma_1) + h(\mu_1 + f_1(h) + \gamma_1)}\right] \tag{3.9}$$

which contains the dynamics of both the systems, man and reservoir populations in contrast to Aron's model where only one system (only human population) was considered. Each of the bracketed terms in RHS of (3.9) is similar to equation (3.6) of Aron [2] and hence as shown by him is a decreasing function of h. Thus in our case also $R(h)$ is a decreasing function in h.

If $R(0) > 1$, a unique equilibrium exists with disease present. If $R(0) < 1$, the only equilibrium is the disease-free state. Thus $R(0)$ is the basic factor which determines the qualitative dynamics of the model. If $R(0) < 1$, the disease-free equilibrium (zero equilibrium) is locally stable (appendix A) and there is no other equilibrium. On the other hand if $R(0) > 1$, the zero equilibrium is unstable (appendix B). Thus $R(0)$ is the number of cases of infection in human susceptibles generated by a single infective individual in the reservoir populations through mosquito bite. In other words, $R(0)$ is the basic reproductive rate in the model. The disease will be present in the human population when $R(0) > 1$.

Now

$$R(0) = \frac{\beta_1\{k_1'(f_2(0) + \mu_2) + k_1''\gamma_2\}}{(\mu_2 + f_2(0))(\mu_2 + \gamma_2)} \tag{3.10}$$

where

$$f_2(0) = \lim_{h\to 0} f_2(h) = \lim_{h\to 0} \frac{(h + \mu_2)e^{-(h+\mu_2)\tau_2}}{1 - e^{-(h+\mu_2)\tau_2}}. \tag{3.11}$$

Again

$$R(0) = \frac{\beta_1 k_1'}{\mu_2 + \gamma_2} + \frac{\beta_1 k_1''}{(\mu_2 + f_2(0))(\mu_2 + \gamma_2)}\gamma_2 \tag{3.12}$$

where

$\frac{\beta_1 k_1'}{\mu_2 + \gamma_2}$ = transmission from the infected reservoir to the human population,

$\frac{\beta_1 k_1''}{\mu_2 + f_2(0)}$ = period of unboosted immunity in human,

and

$\frac{\gamma_2}{\mu_2 + \gamma_2}$ = probability of surviving period of infective state of the reservoirs to become immune.

Figure 1 clearly demonstrates the condition for existence of the non-zero equilibrium. If $R(0) > 1$, the slope of the curve (1) exceeds the slope of the line (2) at the beginning, and after a certain value of h, the latter exceeds the former, so that the two curves intersect two times, once at the origin (disease free state) and secondly at a point in the positive orthant.

4. CONTROL OF THE DISEASE.

Since J.E. is a communicable disease, it can be controlled by two ways – (i) by reducing transmission β_1 which can be achieved by controlling the vector populations (mosquitoes) and (ii) by immunizing the human susceptibles and gradually increasing the proportion of coverage of vaccination (v). The ultimate goal is to reduce $R(0)$ so that $R(0) < 1$ which will result in eradication of the disease. Let β_{1c} and v_c be the threshold values for transmission rate and vaccination coverages respectively where β_{1c} and v_c are determined by Aron [1] and Anderson and May [3].

$$\beta_{1c}(1 - v_c) = \frac{k_1'(\mu_2 + f_2(0)) + \gamma_2 k_1''}{(\mu_2 + \gamma_2)(\mu_2 + f_2(0))} = 1 \tag{4.1}$$

where for eradication the conditions required to be satisfied are $v > v_c$ and $\beta_1 > \beta_{1c}$. Table 1 shows the effect of reduction of transmission on the level of vaccination. It can be observed that increasing the level of vaccination in human means that less effort for reduction of transmission is required to eradicate the disease.

Now, if the infectivity k_1' is constant, then increasing the infectivity of the immune reservoir population (carrier) does not in contrast to Aron's findings, significantly increase the equilibrium levels of infection (Figure 2). The dynamics of J.E. spread is thus qualitatively different from that of the malaria epidemic and eventually poses less difficulty in eradication by vaccination.

Figure 3 shows the two curves for different combination of values for k_1' and k_1'' do not intersect at a non-zero point and, in fact, each curve is a multiple of another. This implies that, in J.E. loss of immunity (f_1) in man is independent of the exposure rate. This is also a characteristic property of J.E., in contrast to the model given by Aron. Thus from our results (Figure 3), it indicates that boostering of immunity against J.E. is feasible only by passive immunization, i.e., direct administration of J.E. antibody in man at recurrent intervals. Acquired immunity by the attack of the disease does not persist for a long time by continued exposure to the bites of infected mosquitoes.

5. CONCLUSION.

The J.E. model presented here is an extension of the SIRS model by coupling the dynamics of the disease in two populations, the reservoir and the human populations. The reservoir population does not itself show any pathological symptom of the disease but acts as an intermediate host medium to pass over the infection to man through a vector population (mosquito). Infection of J.E. cannot spread from man to man or to any other animals, that is to say, man is the dead end of infection.

We have assumed that the effective reservoir of infection (h_1) for man is proportional to the proportion of human susceptibles. The higher (lesser) is the proportion of susceptibles in a human population, the higher (lesser) is the effective reservoir of infection. In other words, in a human population where the number of susceptibles is zero, the effective reservoir of infection will be eventually nil.

It is also assumed, as in Aron ([1] and [2]), that immunes are no more infective than those who are infected both in the reservoir and human populations. The dynamic model of J.E.

spread in reservoir population is same as that of Aron. The reservoir system is also independent of the human system but not the reverse. Aron's system has a stable disease-free equilibrium ($h=0,\ y_2=0,\ y_3=0$) if $R(0)<1$ and a non-zero equilibrium (disease present) if $R(0)>1$. Substituting this result in the coupled system we have obtained similar results on stability properties of J.E. spread in disease by vaccination. In contrast to Aron's results we have observed that in J.E. the loss of immunity in man is independent of the rate of exposure to the disease. This implies that active immunization (direct administration of antigen in the form of live attenuated virus) does not give immunity or prolong acquired immunity in man. Vaccination by passive immunization (i.e., direct administration of serum containing antibody to man) at fixed intervals, on the other hand, will ensure control of the disease.

APPENDIX A. STABILITY OF ZERO EQUILIBRIUM.

Linearizing the system about the zero equilibrium ($x_2=0,\ x_3=0,\ y_2=0,\ y_3=0$), we get the biquadratic characteristic equation

$$(\lambda^2+\theta_1\lambda+\xi_1)(\lambda^2+\theta_2\lambda+\xi_2)=0 \tag{A.1}$$

where

$$\theta_1=2\mu_1+\gamma_1+h+f_1(h) \tag{A.2}$$

$$\xi_1=(\mu_1+\gamma_1+h)(\mu_1+f_1(h)) \tag{A.3}$$

$$\theta_2=2\mu_2+\gamma_2-\beta_2k_2'+h+f_2(h) \tag{A.4}$$

$$\xi_2=(\mu_2+f_2(h))(\mu_2+\gamma_2+h-\beta_2k_2')-\gamma_2(\beta_2k_2''-h) \tag{A.5}$$

The roots of (A.1) having negative real parts, the zero equilibrium is locally stable if and only if θ_1, ξ_1, θ_2 and ξ_2 are all positive.

Now θ_1 and ξ_1 are always positive. The condition that $\xi_2>0\Leftrightarrow R(0)<1$, where $R(0)$ is given in equation (3.10). Again $R(0)<1\Rightarrow\theta_2>0$. Hence if $R(0)>1$, the zero equilibrium is locally unstable.

APPENDIX B. STABILITY OF NONZERO EQUILIBRIUM.

Linearizing the system about the non-zero equilibrium (x_2, x_3, y_2 and y_3), we get the biquadratic characteristic equation

$$(\lambda_2+\alpha_1\lambda+\eta_1)(\lambda^2+\alpha_2\lambda+\eta_2)=0 \tag{B.1}$$

where

$$\alpha_1=2\mu_1+\gamma_1+h+f_1(h)+\beta_1(k_1'y_2+k_1''y_3)(1-x_2-x_3)-\beta_1f_1'(h)(k_1'y_2+k_1''y_3)x_3 \tag{B.2}$$

$$\begin{aligned}\eta_1=&\{\mu_1+\gamma_1+h+\beta_1(k_1'y_2+k_1'')(1-x_2-x_3)\}\{\mu_1+f_1(h)-\beta_1f_1'(h)(k_1'y_2+k_1''y_3)x_3\}\\&+\{\gamma_1+\beta_1f_1'(h)(k_1'y_2+k_1''y_3)x_3\}\{h+\beta_1(k_1'y_2+k_1''y_3)(1-x_2-x_3)\}\end{aligned} \tag{B.3}$$

$$\alpha_2=2\mu_2+\gamma_2+h+f_2(h)+\beta_2f_2'(h)k_2''y_3-\beta_2k_2'(1-y_2-y_3) \tag{B.4}$$

$$\begin{aligned}\eta_2=&\{\mu_2+\gamma_2+h-\beta_2k_2'(1-y_2-y_3)\}\{\mu_2+f_2(h)+\beta_2f_2'(h)k_2''y_3\}\\&+(\gamma_2-\beta_2f_2'(h)k_2'y_3)(h-\beta_2k_2''(1-y_2-y_3)).\end{aligned} \tag{B.5}$$

We see that the equilibrium rate of exposure h is the function of x_1, y_2 and y_3 as defined in equation (2.2) and (2.3). The roots of equation (B.1) having negative real parts, the non-zero equilibrium is locally stable if and only if all α_1, η_1, α_2 and η_2 are positive.

We note that, $\alpha_1>0$ always, since $-1<f_1'(h)<0$.

After canceling few common terms η_1 may be written as

$$\eta_1 = -(\mu_1+\gamma_1)\beta_1 f_1'(h)(k_1'y_2+k_1''y_3)x_3+(\mu_1+f_1(h))\beta_1(k_1'y_2+k_1''y_3)(1-x_2-x_3)$$
$$+\gamma_1\beta_1(k_1'y_2+k_1''y_3)(1-x_2-x_3)+(\mu_1+f_1(h))(\mu_1+\gamma_1+h)+\gamma_1 h.$$

Therefore

$$\eta_1 > 0 \text{ [as } -1 < f_1'(h) < 0]$$

From (2.3), (2.7) and $f_2'(h) > -1$ we have

$$h > \beta_2 k_2'' y_3 > \beta_2 k_2'' f_2'(h) y_3 \tag{B.6}$$

Again from (3.1), (3.2), (2.3) and (2.7) we have

$$\beta_2 k_2'(1-y_2-y_3) = \frac{\beta_2 k_2' y_2(\mu_2+\gamma_2)}{\beta_2(k_2'y_2+k_2''y_3)} < \mu_2+\gamma_2 \tag{B.7}$$

$\alpha_2 > 0$, always holds when a non-zero equilibrium exists.

η_2 may be written as

$$\eta_2 = (\mu_2+\gamma_2+h-\beta_2 k_2'(1-y_2-y_3))\beta_2 f_2'(h)k_2''y_3$$
$$+\beta_2 k_2'(y_2+y_3)(\mu_2+f_2(h))+\beta_2 f_2'(h)k_2'y_3\beta_2 k_2''(1-y_2-y_3)$$
$$-h\beta_2 f_2'(h)k_2'y_3+\{h\gamma_2-\gamma_2\beta_2 k_2''+(\mu_2+\gamma_2+h)(\mu_2+f_2(h))$$
$$-\beta_2 k_2'(\mu_2+f_2(h))\}+\gamma_2\beta_2 k_2''(y_2+y_3) \tag{B.8}$$

Since the equilibrium point must satisfy (3.8) which again implies

$$\frac{\beta_2\{k_2'(\mu_2+f_2(h))+k_2''\gamma_2\}}{(\mu_2+\gamma_2)(\mu_2+f_2(h))+h(\mu_2+\gamma_2+f_2(h))} = 1$$

the term within curly brackets is zero.

Again, from (3.1) and (3.2) we have

$$\gamma_2(y_2+y_3) = y_3(\mu_2+\gamma_2+f_2(h)).$$

The remaining terms in (B.8) may be written as

$$-h\beta_2 f_2'(h)y_3(k_2'-k_2'')+\beta_2 k_2''y_3(\gamma_2+\mu_2)\{(1+f_2'(h)+f_2(h)\}$$
$$+\beta_2 k_2'(y_2+y_3)(\mu_2+f_2(h))$$
$$+\beta_2 f_2'(h)k_2'y_3(1-y_2-y_3)\beta_2 k_2''-\beta_2 k_2'(1-y_2-y_3)\beta_2 f_2'(h)k_2''y_3 \tag{B.9}$$

The last two terms in (B.9) cancel each other and therefore $\eta_2 > 0$ since, $k_2' > k_2''$ and $-1 < f_2'(h) < 0$. Thus whenever the non-zero equilibrium exists, it is stable.

TABLE 1

Eradication criteria for J.E.

Level of vaccination $100\ v_c$	Reduction of Transmission $100\ (1-\frac{\beta_{1c}}{\beta_1})$
0%	93.9%
25%	89.9%
50%	81.9%
75%	57.9%
80%	45.9%
85%	25.9%

The eradication criteria are given in (4.1).

The parameters used are $\beta_1 = 1, \mu_2 = 0.02, \tau = 1, \gamma_2 = 0.1, k_2' = 1, k_2'' = 0.6, f_2(0) = 0.99$.

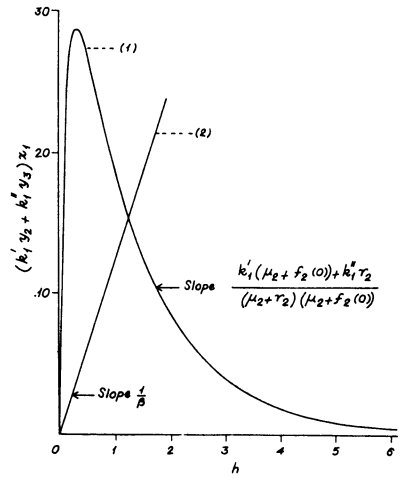

Fig.1 Effective reservoir of infection in man, $(k_1' y_2 + k_1'' y_3)\ x_1$ as a function of the rate of exposure h, according to (1) equation (3.6) and (2) equation (3.7). The parameters used are $\beta_1 = 8$, $\mu_1 = 0.02$, $\mu_2 = 0.2$, $\gamma_1 = 0.7$, $\gamma_2 = 0.1$, $\tau_1 = \tau_2 = 1$, $k_1' = 1$, $k_1'' = 0.5$

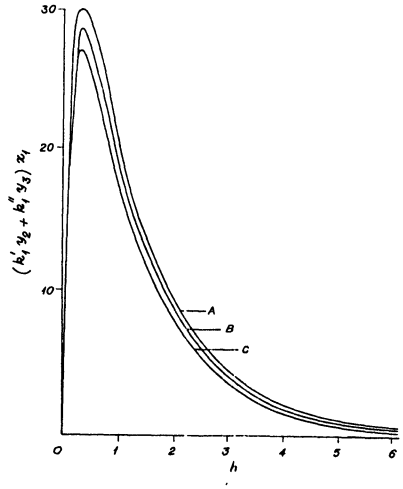

Fig.2 Effective reservoir of infection in man, $(k_1'y_2+k_1''y_3)\ x_1$ as a function of the rate of exposure h, according to equation (3.6): (A) $k_1'' = 1$, (B) $k_1'' = 0.5$, (C) $k_1'' = 0$. The rest of the parameters used are $\mu_1 = 0.02$, $\mu_2 = 0.2$, $\gamma_1 = 0.7$, $\gamma_2 = 0.1$, $\tau_1 = \tau_2 = 1$, $k_1' = 1$.

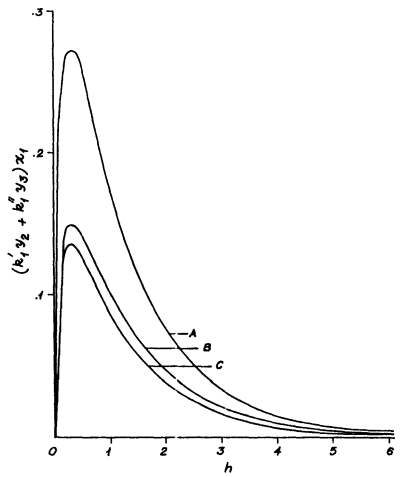

Fig.3 Effective reservoir of infection in man, $(k_1'y_2+ k_1''y_3)\ x_1$ as a function of the rate of exposure h according to equation (3.6): (A) $k_1' = 1$, $k_1'' = 0$, (B) $k_1' = 0.5$, $k_1'' = 0.5$, (C) $k_1 = 0.5$, $k_1'' = 0$. The rest of the parameters used are $\mu_1 = 0.02$, $\mu_2 = 0.2$, $\gamma_1 = 0.7$, $\gamma_2 = 0.1$, $\tau_1 = \tau_2 = 1$.

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