日本脳炎流行の理論的考察

和田 義人

熱帯医学

1975年

http://hdl.handle.net/10069/4172
Theoretical Considerations on the Epidemic of Japanese Encephalitis

Yoshito WADA

Department of Medical Zoology, Nagasaki University School of Medicine

ABSTRACT: Based on the view that Japanese encephalitis is essentially a disease of pigs in Japan, a mathematical model for the epizootic in pigs was developed under some assumptions and conditions, and the relation to the epidemic in man was considered. As given in the below, simulation studies with the model revealed several features in pig epizootic and human epidemic, many of which are otherwise not easy to understand. It can be said that the density of vector mosquitoes and the scale of human epidemic are positively related each other, but the increased number of mosquitoes much less sensitively influences the scale of pig epizootic. The time, in relation to mosquito prevalence, of the initiation of pig infection is important to affect the number of infected pigs, and also the number of transmissible mosquitoes which is positively related to the number of human cases. The existence of threshold densities of mosquitoes and susceptible pigs below which the pig epizootic tends to extinguish is clearly indicated. The increase of the daily survival rate of mosquitoes greatly influences the pig epizootic and the human epidemic, as in the increase of the mosquito density. By the artificial immunization of pigs, the number of infected pigs decreases and the rate of pigs having antibody becomes high. Also the number of transmissible mosquitoes, and therefore the number of human cases can be greatly reduced by the pig immunization, but the reduced number is still fairly large when the mosquito density is very high.

Japanese encephalitis (JE) is a very important disease in Japan and some other countries, but it is not easy to understand exactly by what the number of human cases is controlled, because many complicated factors are involved in it. In such a case it was thought that a mathematical model may be effectively used to understand the epidemiology, as in the case of malaria (see MacDonald, 1957), and two separate papers on the subject, both written in Japanese, were published (Wada, 1972a, b). The present paper is principally based on the previous two papers, but the model is more collectively considered and some new topics by simulation studies are added.
MODEL FOR JE EPIZOOTIC IN PIGS

Although there is an opinion that JE has evolved as a disease of birds inhabiting marshes (Mattingly, 1960), it may be regarded as a disease of pigs at least at the present time in Japan. Infected humans can not be origins of the next infection cycle (Fukumi, 1964). Such wild birds as black-crowned night herons may be involved in the infection cycle of JE (Scherer et al., 1959), but their importance as amplifying animals is far less than pigs, in view of the scarcity of individual pigs and the long period for generation turnover. In other words, the infection cycle of JE in Japan is maintained in the epidemic season mainly between the most important amplifying animal, the pig, and the most important vector mosquito, Culex tritaeniorhynchus (referred to simply as mosquito hereinafter), and accordingly the human epidemic is directly influenced by the pig epizootic. For this reason, the theoretical model for JE epizootic in pigs was developed.

To develop the model, the following assumptions are set up:
1) There are a certain number of pigs (n) within a range of normal flight activity of mosquitoes (cf. Wada et al., 1969).
2) There is no emigration nor immigration in the populations of both pigs and mosquitoes.
3) Age distribution of pigs is stable, births and slaughtereds being balanced.
4) Pigs are slaughtered at a definite age of a months.
5) Pigs have maternal antibody for a constant duration of b months from the birth.
6) Probability of being bitten by a mosquito is the same in any pigs.
7) Mosquitoes feed only on pigs.
8) Probability of mosquito survival through one day is constant (p) irrespective of age.
9) Mosquitoes which fed on a viremic pig are all infected, and all infected mosquitoes which survived a certain duration of k days become transmissible.
10) Immediately after infected mosquitoes became transmissible, they feed on pigs.
11) All susceptible pigs which were fed on by the transmissible mosquito become viremic for m days after a certain duration of l days.
12) All infected pigs become immune after a viremic state.

Under the above assumptions, the number of pigs without maternal antibody (N) in a population of n pigs is given by

\[ N = n \times \frac{a - b}{a} \] ................................. (1)

where a is the slaughtered age and b the duration with maternal antibody of pigs. The duration of one infection cycle (c months) is obtained from \( k + l + \frac{m}{2} \), where k is the duration from the infection of the mosquito to the time when it becomes transmissible, l the duration from the infection of the pig by the bite of the transmissible mosquito to the time when it becomes viremic, m the duration of viremia in the pig. Since births and slaughtereds of pigs are balanced, their number produced during every c months (d) is

\[ d = n \times \frac{c}{a} \] ................................. (2)
Letting the survival rate of the mosquito through one day be $p$, then the survival rate from the infection to the time of becoming transmissible ($p'$) is

$$p' = p^k.$$  \hspace{1cm} (3)

At time $t$ in terms of infection cycle, letting the number of mosquitoes biting one pig in one night be $M(t)$, the number of infected pigs $C(t)$, the number of infected and immune pigs just after infection $A_1(t)$, and the number of susceptible pigs just after infection $S_1(t)$, then

$$A_1(t) = C(t) \hspace{1cm} \text{..........................................................................................} \hspace{1cm} (4)$$

$$S_1(t) = N - A_1(t) \hspace{1cm} \text{..........................................................................................} \hspace{1cm} (5)$$

And at time $t+1$, the number of transmissible mosquitoes, $T(t+1)$, is shown as

$$T(t+1) = M(t) \times C(t) \times p'. \hspace{1cm} \text{..........................................................................................} \hspace{1cm} (6)$$

The probability for a given mosquito to be transmissible, $R(t+1)$, is

$$R(t+1) = \frac{T(t+1)}{M(t+1) \times n}. \hspace{1cm} \text{..........................................................................................} \hspace{1cm} (7)$$

Therefore, the probability that a given pig is infected by the bites of $M(t+1)$ mosquitoes, $I(t+1)$, is expressed by

$$I(t+1) = 1 - Q(t+1) = (t+1), \hspace{1cm} \text{..........................................................................................} \hspace{1cm} (8)$$

where $Q(t+1)$ is the probability for one mosquito not to be transmissible, being equal to $1 - R(t+1)$. Just before infection at time $t+1$, the number of immune pigs, $A_2(t+1)$, is

$$A_2(t+1) = A_1(t) \times \left(1 - \frac{d}{N}\right) \hspace{1cm} \text{..........................................................................................} \hspace{1cm} (9)$$

and the number of susceptible pigs, $S_2(t+1)$, is

$$S_2(t+1) = N - A_2(t+1). \hspace{1cm} \text{..........................................................................................} \hspace{1cm} (10)$$

Therefore, the number of infected pigs, $C(t+1)$, at time $t+1$ is given by

$$C(t+1) = S_2(t+1) \times I(t+1). \hspace{1cm} \text{..........................................................................................} \hspace{1cm} (11)$$

The number of infected and immune pigs just after infection at $t+1$ is

$$A_1(t+1) = A_2(t+1) + C(t+1). \hspace{1cm} \text{..........................................................................................} \hspace{1cm} (12)$$

The number of infected pigs at $t+2$, $C(t+2)$, can be obtained similarly by using Expression (12) and, after substituting $t+2$ for $t+1$, Expressions (6) – (11). In this way, we can get the theoretical epizootic process, if the values are given to the constants which were described in the assumptions, and to the initial number of infected pigs at time $t$, $C(t)$.

When the number of mosquitoes is not changed but constant $M$ in relation to time, Expression (7) becomes

$$R(t+1) = \frac{T(t+1)}{M \times n}$$

$$= \frac{M \times C(t) \times p'}{M \times n}$$

$$= \frac{C(t) \times p'}{n}$$

and therefore Expression (11) can be written as

$$C(t+1) = S_2(t+1) \times \left(1 - (1 - \frac{C(t) \times p'}{n})^n\right). \hspace{1cm} \text{..........................................................................................} \hspace{1cm} (13)$$
As mentioned above, $A_1(t)$ or $A_2(t)$ is the number of immune pigs (infected pigs inclusive) just after or before infection, therefore $\frac{A_1(t)}{N}$ or $\frac{A_2(t)}{N}$ is the rate of immune pigs among the all pigs excepting those with maternal antibody. However, this rate is usually lower than the rate among slaughtered pigs which are older in age. Because the rate of slaughtered pigs with HI antibody can be used as a good measure to indicate the evidence of pig epizootic, and for this reason it has been examined routinely in every prefecture in Japan, the model was modified to demonstrate the immune rate of slaughtered pigs as in the following.

Having let the life span of pigs be $a$ and the duration with maternal antibody be $b$, the age of pigs involved in the infection of JE ranges from $b$ to $a$. Since the duration of one infection cycle is $c$, it seems appropriate to divide the pigs involved in the infection cycle into $\frac{a-b}{c}$ age groups. Here, $C(t)$, $A_1(t)$, $S_1(t)$, $A_2(t)$, and $S_2(t)$ in age group $i$ are designated as $C(t,i)$, $A_1(t,i)$, $S_1(t,i)$, $A_2(t,i)$, and $S_2(t,i)$, where $i=1,2,\ldots, j \left(=\frac{a-b}{c}\right)$, age group 1 being the youngest, then the following Expressions hold.

\[
C(t) = \sum_{i=1}^{j} C(t,i) \tag{14}
\]

\[
A_1(t) = \sum_{i=1}^{j} A_1(t,i) \tag{15}
\]

\[
S_1(t) = \sum_{i=1}^{j} S_1(t,i) \tag{16}
\]

\[
A_2(t) = \sum_{i=1}^{j} A_2(t,i) \tag{17}
\]

\[
S_2(t) = \sum_{i=1}^{j} S_2(t,i) \tag{18}
\]

When $C(t)$ pigs are firstly infected at time $t$, there is no reason to assume different infection rates in different age groups. Thus,

\[
C(t,1)=C(t,2)=\cdots=C(t,j)=\frac{C(t)}{j} \tag{19}
\]

and accordingly

\[
A_1(t,i)=C(t,i) \tag{20}
\]

From Expressions (15), (20), and (19)

\[
A_1(t) = \sum_{i=1}^{j} A_1(t,i)
\]

\[
= \sum_{i=1}^{j} C(t,i)
\]

\[
= C(t).
\]

This is the same as Expression (4). Also, it is apparent that

\[
S_1(t,i)=d-A_1(t,i) \tag{21}
\]
At time $t+1$, $A_1(t, j)$ and $S(t, j)$ are slaughtered, while $d$ pigs become susceptible by losing maternal antibody. Therefore, susceptible pigs are

$$S_2(t+1, i) = d \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \quad \ quad
for \( i = 2, \ldots, j \). And then the number of susceptible pigs, \( S_i(t+1, i) \), is
\[
S_i(t+1, i) = d - A_1(t+1, i) \tag{30}
\]
At time \( t+2 \), \( A_1(t+1, j) \) and \( S_i(t+1, j) \) are slaughtered and \( d \) pigs become susceptible by losing maternal antibody, just as seen at time \( t+1 \). Thus,
\[
S_i(t+2, i) = d \tag{31}
\]
for \( i = 1 \), and
\[
S_i(t+2, i) = S_i(t+1, i-1) \tag{32}
\]
for \( i = 2, \ldots, j \). Therefore, the number of infected pigs in age group \( i \), \( C(t+2, i) \), is given by
\[
C(t+2, i) = S_i(t+2, i) \times I(t+2) \tag{33}
\]
In this way, we can obtain successively the epizootic process in pigs by age group.

It should be noted that immune pigs are accumulated with the progress of time, accordingly the rate of immune pigs is higher in older age groups. Since the pigs to be slaughtered are in the oldest age group, the rate is the highest among all age groups. \( \frac{A_1(t)}{N} \) or \( \frac{A_2(t)}{N} \) is the rate of immune pigs among the all pigs excepting those with maternal antibody,
but it is usually different from the rate among slaughtered pigs, which is expressed by \( \frac{A_1(t, j)}{d} \)
or \( \frac{A_2(t, j)}{d} \).

**CONDITIONS GIVEN TO THE MODEL**

If certain values are given to the *constants* which have appeared in the model for JE epizootic in pigs, we can calculate the epizootic process under a given prevalence of mosquito density, starting with a given initial number of infected pigs. The *constants* mentioned here, strictly speaking, may be variable under particular conditions, or may be even subject to a rather great change. However, because it seems that JE epizootic process is influenced most profoundly by the change of the mosquito density, it was attempted in the present paper to examine how influenced by it, keeping *constants* not variable. Thus, the values for *constants* were given as follows:

1. The number of pigs in an area \( n = 1,000 \).
2. Slaughtered age of pigs \( a = 8 \) months.
3. Duration with maternal antibody in a pig from the birth \( b = 2 \) months. Therefore, the number of pigs without maternal antibody \( N = n \times \frac{a - b}{a} = 750 \).
4. Duration showing viremia in a pig \( m = 1 \) day. Although one day was here given as the value of \( m \), actually it seems to be a little longer. Strictly, therefore, \( M(t) \) should be regarded as the number of mosquitoes biting one pig in \( m \) nights, not in one night.
5. Duration from the infection of the mosquito to the time of becoming transmissi-
ble \( k = 10 \) days, duration from the infection of the pig to the time of showing viremia \( l = 4 \) days. Therefore, the duration of one infection cycle \( c = k + l + \frac{m}{2} \approx 0.5 \) month, and the number of pigs born or slaughtered in each infection cycle \( d = n \times \frac{c}{a} = 62.5 \).

(6) Probability of mosquito survival through the period for the infected mosquito to become transmissible \( p' = 0.05 \). As to this value, it may be necessary to give some comments in the below.

If the probability of mosquito survival through one day is constant irrespective of age, daily survival rate, \( p' \), can be obtained after Davidson (1955) as follows. Provided that the mosquito takes the first blood meal \( r \) days after emergence and the duration of one gonotrophic cycle (from blood feeding to next blood feeding) is \( s \) days, then,

\[
\text{nulliparous rate} = \frac{p'^r}{(p'^r + p'^{r+s} + p'^{r+2s} + \ldots)} = 1 - p'^s,
\]

therefore,

\[
\text{parous rate} = 1 - \text{nulliparous rate} = p'^s.
\]

Thus, we can estimate the daily survival rate of the mosquito in the field \( p' \) by the parous rate and the duration of one gonotrophic cycle. According to Kawai (1969), the duration of one gonotrophic cycle is 5 days at an insectary of 27°C, and we have an unpublished data that the parous rate of mosquitoes collected in summer in Nagasaki area was 0.22. Applying these figures, 0.74 was obtained as an estimate of \( p' \), and therefore the probability of mosquito survival through the period for the infected mosquito to become transmissible \( (k = 10 \) days) \( p' \) was given as

\[
p' = p'^k = 0.74^{10} = 0.05
\]

However, the assumption that the daily survival rate of the mosquito is constant irrespective of age must be examined if this is valid or not through careful studies in the field. Also studies would be necessary whether the duration of a gonotrophic cycle obtained in the laboratory can be applied to the field population. Besides these, it would be reasonable to consider that the daily survival rate itself may change under different environmental conditions and consequently the process of JE epizootic in pigs may change, too. In the present paper the stress was put on the examination of the effect by the density of the mosquito on the epizootic process, firstly the probability of mosquito survival through the incubation period \( p' \) was regarded as constant and the value \( p' = 0.05 \) was used, and then the cases when the daily survival rate of mosquitoes is changed were discussed.
The epizootic process in pigs and the density of mosquitoes

Under those assumptions and conditions described in the preceding sections, theoretical epizootic process in pigs was examined when the mosquito density is constant in relation to time, starting with one infected pig. In Fig. 1 are given the prevalences of infected pigs C(t) when the numbers of mosquitoes feeding on a pig in a night are 40, 160, 640, 2,560, and 10,240, calculations being based on the model for JE epizootic in pigs. It is apparent that generally the infected pigs are larger in number and sharper in appearance when the mosquito density is higher. However, it must be noted that the difference in the prevalences of infected pigs is very slight at the mosquito densities of 640 or more per pig per night. Also it is indicated that irrespective of the mosquito density the infected pigs do not disappear with the progress of time (in terms of infection cycle), but continue to be produced, due to the constant production of susceptible pigs by losing maternal antibody.

Fig. 2 shows the prevalences of the rates of immune pigs (infected pigs inclusive) under the same conditions as in Fig. 1. The solid lines drawn in Fig. 2 indicate the rates of immune pigs in the 750 pigs (all pigs excepting 250 with maternal antibody in a population of 1,000 pigs), \( \frac{A_2(t)}{750} \) and \( \frac{A_1(t)}{750} \), and the broken lines those in slaughtered pigs, \( \frac{A_2(t,12)}{62.5} \) and \( \frac{A_1(t,12)}{62.5} \). That “stairs-shaped” lines are drawn in Fig. 2 is due to the fact that the infection is assumed to occur discontinuously at each time; the length of the vertical line indicates the rate of infected pigs at that time and the lower end point of the
vertical line is the rate of immune pigs immediately before the infection $\frac{A_3(t)}{750}$ or $\frac{A_3(t, 12)}{62.5}$ and the upper end point the rate immediately after the infection $\frac{A_1(t)}{750}$ or $\frac{A_1(t, 12)}{62.5}$. But, as the values of constants in the model are actually more or less variable, the real situation would perhaps be given by the more smooth curve. The rate of immune pigs in the 750 pigs (solid line) shows similar prevalence to the rate in slaughtered pigs (broken line), excepting that in the former the reduction of the rate is observed between two infection cycles due to the appearance of the susceptible pigs by losing maternal antibody, while in the latter such reduction does not occur.

It is seen from Fig. 2 that the rate of immune pigs shows S-shaped increase which is in general sharper at higher mosquito density. Again it is seen that the state of the increase does not differ much when the mosquito density per pig per night is 640 or more.

Next was examined the process of JE epizootic in pigs under changing mosquito density in relation to time. Here, the prevalences of mosquitoes were tentatively given, theory was adopted merely from the reasons that the seasonally cumulated curve of the numbers of mosquitoes collected in the field can be approximated by the logistic curve (Maeda, 1970), and that this was thought to be enough for the purpose to compare the epizootic processes when the first pig infections occur at various times in relation to the mosquito prevalence.

Table 1 shows the prevalences of the number of infected pigs, the percentage of immune pigs, and the number and the percentage of transmissible mosquitoes, after one first pig is infected at time in infection cycle $t=0$ in a population of 1,000 pigs, in which 250 have maternal antibody, under changing density of mosquitoes. Four examples were
given with different mosquito prevalences of which peak densities are 175, 600, 2,125, and 5,500 mosquitoes per pig per night respectively, the peaks being situated at \( t=3 \) in all examples.

It is seen in Table 1 that the prevalences of infected pigs are very similar in all the four examples in spite of very different mosquito prevalences, though the peak time of the appearance of infected pigs is slightly earlier when the mosquito density is higher.

<table>
<thead>
<tr>
<th>Time in infection cycle</th>
<th>No. of mosquitoes per pig per night</th>
<th>No. of infected pigs</th>
<th>% immunity in whole pigs</th>
<th>% immunity in slaughtered pigs</th>
<th>No. of transmissible mosqs</th>
<th>% of transmissible mosqs</th>
</tr>
</thead>
<tbody>
<tr>
<td>( t = 0 )</td>
<td>919</td>
<td>859</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(Example 1)
<table>
<thead>
<tr>
<th>( t )</th>
<th>( M(t) )</th>
<th>( C(t) )</th>
<th>( A_2(t) \times 100/750 )</th>
<th>( A_2(t) \times 100/750 )</th>
<th>( A_2(t, 12) \times 100/62.5 )</th>
<th>( A_2(t, 12) \times 100/62.5 )</th>
<th>( T(t) )</th>
<th>( R(t) \times 100 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>68</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>0.00</td>
</tr>
<tr>
<td>1</td>
<td>111</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>0.01</td>
</tr>
<tr>
<td>2</td>
<td>155</td>
<td>11</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>14</td>
<td>0.05</td>
</tr>
<tr>
<td>3</td>
<td>175</td>
<td>59</td>
<td>0</td>
<td>2</td>
<td>11</td>
<td>1</td>
<td>11</td>
<td>0.33</td>
</tr>
<tr>
<td>4</td>
<td>155</td>
<td>275</td>
<td>0</td>
<td>11</td>
<td>11</td>
<td>50</td>
<td>512</td>
<td>1.92</td>
</tr>
<tr>
<td>5</td>
<td>111</td>
<td>387</td>
<td>45</td>
<td>50</td>
<td>95</td>
<td>95</td>
<td>2,132</td>
<td>3.16</td>
</tr>
<tr>
<td>6</td>
<td>68</td>
<td>97</td>
<td>87</td>
<td>98</td>
<td>95</td>
<td>99</td>
<td>2,151</td>
<td>3.16</td>
</tr>
<tr>
<td>7</td>
<td>38</td>
<td>21</td>
<td>90</td>
<td>93</td>
<td>99</td>
<td>100</td>
<td>330</td>
<td>0.87</td>
</tr>
<tr>
<td>8</td>
<td>20</td>
<td>4</td>
<td>84</td>
<td>85</td>
<td>100</td>
<td>100</td>
<td>39</td>
<td>0.20</td>
</tr>
<tr>
<td>9</td>
<td>10</td>
<td>1</td>
<td>77</td>
<td>77</td>
<td>100</td>
<td>100</td>
<td>39</td>
<td>0.20</td>
</tr>
<tr>
<td>10</td>
<td>5</td>
<td>0</td>
<td>68</td>
<td>68</td>
<td>100</td>
<td>100</td>
<td>0</td>
<td>0.01</td>
</tr>
<tr>
<td>11</td>
<td>3</td>
<td>0</td>
<td>60</td>
<td>60</td>
<td>100</td>
<td>100</td>
<td>0</td>
<td>0.00</td>
</tr>
<tr>
<td>Total</td>
<td>919</td>
<td>859</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5,268</td>
<td></td>
</tr>
</tbody>
</table>

(Example 2)
<table>
<thead>
<tr>
<th>( t )</th>
<th>( M(t) )</th>
<th>( C(t) )</th>
<th>( A_2(t) \times 100/750 )</th>
<th>( A_2(t) \times 100/750 )</th>
<th>( A_2(t, 12) \times 100/62.5 )</th>
<th>( A_2(t, 12) \times 100/62.5 )</th>
<th>( T(t) )</th>
<th>( R(t) \times 100 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>62</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>0.00</td>
</tr>
<tr>
<td>1</td>
<td>183</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>0.01</td>
</tr>
<tr>
<td>2</td>
<td>427</td>
<td>16</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>22</td>
<td>0.02</td>
</tr>
<tr>
<td>3</td>
<td>600</td>
<td>213</td>
<td>2</td>
<td>27</td>
<td>2</td>
<td>28</td>
<td>343</td>
<td>0.66</td>
</tr>
<tr>
<td>4</td>
<td>427</td>
<td>538</td>
<td>25</td>
<td>100</td>
<td>28</td>
<td>100</td>
<td>6,382</td>
<td>1.49</td>
</tr>
<tr>
<td>5</td>
<td>183</td>
<td>63</td>
<td>91</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>11,487</td>
<td>2.68</td>
</tr>
<tr>
<td>6</td>
<td>62</td>
<td>28</td>
<td>92</td>
<td>95</td>
<td>100</td>
<td>100</td>
<td>579</td>
<td>0.53</td>
</tr>
<tr>
<td>7</td>
<td>19</td>
<td>8</td>
<td>87</td>
<td>88</td>
<td>100</td>
<td>100</td>
<td>86</td>
<td>0.44</td>
</tr>
<tr>
<td>8</td>
<td>6</td>
<td>1</td>
<td>80</td>
<td>80</td>
<td>100</td>
<td>100</td>
<td>8</td>
<td>0.13</td>
</tr>
<tr>
<td>9</td>
<td>2</td>
<td>0</td>
<td>72</td>
<td>72</td>
<td>100</td>
<td>100</td>
<td>0</td>
<td>0.00</td>
</tr>
<tr>
<td>10</td>
<td>1</td>
<td>0</td>
<td>63</td>
<td>63</td>
<td>100</td>
<td>100</td>
<td>0</td>
<td>0.00</td>
</tr>
<tr>
<td>Total</td>
<td>1,972</td>
<td>870</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>18,910</td>
<td></td>
</tr>
</tbody>
</table>

(Example 3)
<table>
<thead>
<tr>
<th>( t )</th>
<th>( M(t) )</th>
<th>( C(t) )</th>
<th>( A_2(t) \times 100/750 )</th>
<th>( A_2(t) \times 100/750 )</th>
<th>( A_2(t, 12) \times 100/62.5 )</th>
<th>( A_2(t, 12) \times 100/62.5 )</th>
<th>( T(t) )</th>
<th>( R(t) \times 100 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>51</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>0.00</td>
</tr>
<tr>
<td>1</td>
<td>266</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>0.00</td>
</tr>
<tr>
<td>2</td>
<td>1,110</td>
<td>21</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>3</td>
<td>28</td>
<td>0.00</td>
</tr>
<tr>
<td>3</td>
<td>2,125</td>
<td>496</td>
<td>3</td>
<td>68</td>
<td>3</td>
<td>68</td>
<td>1,141</td>
<td>0.05</td>
</tr>
<tr>
<td>4</td>
<td>1,110</td>
<td>276</td>
<td>62</td>
<td>100</td>
<td>68</td>
<td>100</td>
<td>52,707</td>
<td>4.75</td>
</tr>
<tr>
<td>5</td>
<td>266</td>
<td>63</td>
<td>92</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>15,302</td>
<td>5.76</td>
</tr>
<tr>
<td>6</td>
<td>51</td>
<td>35</td>
<td>92</td>
<td>96</td>
<td>100</td>
<td>100</td>
<td>830</td>
<td>1.62</td>
</tr>
<tr>
<td>7</td>
<td>9</td>
<td>8</td>
<td>88</td>
<td>89</td>
<td>100</td>
<td>100</td>
<td>91</td>
<td>0.96</td>
</tr>
<tr>
<td>8</td>
<td>2</td>
<td>0</td>
<td>81</td>
<td>81</td>
<td>100</td>
<td>100</td>
<td>4</td>
<td>0.21</td>
</tr>
<tr>
<td>Total</td>
<td>4,990</td>
<td>902</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>70,106</td>
<td></td>
</tr>
</tbody>
</table>

(Example 4)
<table>
<thead>
<tr>
<th>( t )</th>
<th>( M(t) )</th>
<th>( C(t) )</th>
<th>( A_2(t) \times 100/750 )</th>
<th>( A_2(t) \times 100/750 )</th>
<th>( A_2(t, 12) \times 100/62.5 )</th>
<th>( A_2(t, 12) \times 100/62.5 )</th>
<th>( T(t) )</th>
<th>( R(t) \times 100 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>30</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0.00</td>
</tr>
<tr>
<td>1</td>
<td>264</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0.00</td>
</tr>
<tr>
<td>2</td>
<td>1,976</td>
<td>12</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>15</td>
<td>0.00</td>
</tr>
<tr>
<td>3</td>
<td>5,500</td>
<td>505</td>
<td>2</td>
<td>67</td>
<td>2</td>
<td>67</td>
<td>1,149</td>
<td>0.02</td>
</tr>
<tr>
<td>4</td>
<td>1,976</td>
<td>276</td>
<td>61</td>
<td>100</td>
<td>67</td>
<td>100</td>
<td>138,771</td>
<td>7.02</td>
</tr>
<tr>
<td>5</td>
<td>264</td>
<td>63</td>
<td>92</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>27,268</td>
<td>10.34</td>
</tr>
<tr>
<td>6</td>
<td>30</td>
<td>35</td>
<td>92</td>
<td>96</td>
<td>100</td>
<td>100</td>
<td>824</td>
<td>2.76</td>
</tr>
<tr>
<td>7</td>
<td>3</td>
<td>5</td>
<td>88</td>
<td>89</td>
<td>100</td>
<td>100</td>
<td>53</td>
<td>1.59</td>
</tr>
<tr>
<td>Total</td>
<td>10,043</td>
<td>898</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>168,081</td>
<td></td>
</tr>
</tbody>
</table>
This has already been indicated in the case of constant mosquito densities. It is also shown that in all the four cases the number of infected pigs becomes 0 after forming a peak, thus the epizootic is extinguished. The prevalences of immune pigs are again very similar in all the four examples. The percentages of immune pigs in the whole pigs excepting those with maternal antibody, \( A_f(t) \times 100/750 \) and \( A_i(t) \times 100/750 \), increase with time and reach 100% or nearly so, then decrease to 60–89%. The percentages in slaughtered pigs, however, do not decline after reaching 100%.

In the above, the cases in which one first pig is infected at \( t=0 \) were mentioned. Fig. 3A–D illustrates how the epizootic processes differ when one first pig is infected at \( t=0, 2, 4, \) and \( 6 \), that is, 3 infection cycles before, 1 cycle before, 1 cycle after, and 3 cycles after the peak time of the mosquito prevalence as indicated by vertical arrows in the figure. Here, the mosquito prevalence is the same as that in Example 3 of Table 1.

Fig. 3A clearly shows that when the pig epizootic has started with one infected pig before the peak time of mosquitoes, \( i.e., t=0 \) and \( 2 \), many infected pigs are produced. On the other hand, if the epizootic has started after the peak time of mosquitoes, for example at \( t=4 \), the number of infected pigs is reduced greatly, and if the epizootic has started at \( t=6 \) the reduction in the number of infected pigs is much more conspicuous. The rates of immune pigs (Fig. 3B), both in the all pigs excepting those with maternal antibody and in slaughtered pigs, are generally in parallel in prevalence with the numbers of infected pigs. If the starting time of pig epizootic is at \( t=0 \) and \( t=2 \) (before mosquito peak), the rates reach 100%, but if the starting time is at \( t=4 \) (1 cycle after mosquito peak) the rate does not exceed 80%, and if \( t=6 \) (3 cycles after peak) the rate remains at nearly 0%.

It was shown in the above that the starting time of the epizootic in relation to the mosquito prevalence is very important in determining the prevalence of infected pigs thereafter. The effect of the starting time of the epizootic is more remarkable on the prevalence of transmissible mosquitoes, which is closely related to the human epidemic, than on the prevalence of infected pigs, and this will be described later.

Before going to the next section, the pig epizootic for a long term will be mentioned. The above explained that the pig epizootic is variable in process under different mosquito prevalences, all of which have only one peak. These mosquito prevalences were modeled after the situation found in Japan where the cold winter does not allow the mosquito feeding activity, and therefore the epizootic disappears as a matter of course. The situation in more southern areas is apparently different from it; the mosquito continues to feed on animals throughout the year, though the mosquito fluctuates in number. Because considerations on such cases are thought to be worth while for understanding the nature of the pig epizootic even in Japan, two examples of the epizootic process for 72 infection cycles or 3 years are calculated by the model and illustrated in Fig. 4A – B. Here, mosquito prevalences, which were based on the sine curve, all have 3 peaks during 72 infection cycles, but with different amplitudes and different mean levels.

It is shown from Fig. 4A, in which the minimum and maximum numbers of mosqui-
toes per pig per night during the period are respectively 30 and 350, that 3 epizootics of pigs appear as in the mosquito prevalence. However, the peak of infected pigs is slightly before the corresponding peak of mosquitoes, indicating clearly that a sufficient number of susceptibles are necessary for the pig epizootic.

Fig. 4B illustrates the situation when the minimum number of mosquitoes per pig per night is the same as in Fig. 4A, i.e., 30, but the maximum number is much smaller, i.e., 50. The variation in the number of mosquitoes is as small as 20, but 3 peaks of infected pigs are again shown. This suggests that even under continuous feeding of mosquitoes throughout the year, it is probable that the infection of pigs appears in the form of epizootic owing to the slight seasonal change of mosquito density. It is also represented from Fig. 4B.

**Fig. 3.** Numbers of infected pigs (C(t)) in Fig. 3A, percentages of immune pigs (A_2 (t)×100/750, A_1(t)×100/750, A_2(t, 12)×100/62.5, and A_1(t, 12)×100/62.5) in Fig. 3B, and numbers and percentages of transmissible mosquitoes (T(t) and R(t)×100) in Fig. 3C and 3D, after one first pig is infected in a population of 1,000 pigs at various times in relation to the prevalence of mosquitoes feeding on the pigs. The time of the initiation of pig infection is indicated by an vertical arrow. As to solid and broken lines for percentages of immune pigs in Fig. 3B, see Fig. 2.
that if the level of mosquito density is generally low a large number of susceptibles remain uninfected even after the epizootic.

**THE EPIZOOTIC IN PIGS AND THE EPIDEMIC IN MAN**

JE is not considered essentially to be a disease of human beings in the sense that the infection cycle of JE can not be maintained without amplifying vertebrate animals, of which the most important one is pigs in Japan. Therefore, it is natural that the human epidemic is directly influenced by the pig epizootic, that is, the more the transmissible mosquitoes produced from the pig epizootic, generally the more the human cases. if other conditions remain the same. For this reason, the numbers of the transmissible mosquitoes under various mosquito densities and prevalences will be compared.

![Diagram 3C](image)

**FIG. 3C**

- Time in Infection Cycle: Total: 70,106

![Diagram 3D](image)

**FIG. 3D**

- Time in Infection Cycle: Total: 44,443

![Diagram 3E](image)

- Time in Infection Cycle: Total: 4

![Diagram 3F](image)

- Time in Infection Cycle: Total: 1,472
First of all, are given the cases when the number of mosquitoes is constant in relation to time. Table 2 shows the number of transmissible mosquitoes produced from various constant numbers of feeding mosquitoes per pig per night in a population of 1,000 pigs during 3, 5 and 10 infection cycles after the infection of one pig.

Table 2. Numbers of transmissible mosquitoes produced from various constant numbers of feeding mosquitoes per pig per night in a population of 1,000 pigs during 3, 5 and 10 infection cycles after the infection of one pig

<table>
<thead>
<tr>
<th>No. of mosquitoes per pig per night (A)</th>
<th>During 3 cycles</th>
<th>During 5 cycles</th>
<th>During 10 cycles</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of transmissible mosquitoes (B)</td>
<td>No. of transmissible mosquitoes (C)</td>
<td>No. of transmissible mosquitoes (D)</td>
</tr>
<tr>
<td>10</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>20</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>40</td>
<td>10</td>
<td>26</td>
<td>196</td>
</tr>
<tr>
<td>80</td>
<td>52</td>
<td>439</td>
<td>3,388</td>
</tr>
<tr>
<td>160</td>
<td>335</td>
<td>5,071</td>
<td>7,550</td>
</tr>
<tr>
<td>320</td>
<td>2,263</td>
<td>13,103</td>
<td>36,870</td>
</tr>
<tr>
<td>640</td>
<td>13,175</td>
<td>27,163</td>
<td>75,997</td>
</tr>
<tr>
<td>1,280</td>
<td>45,986</td>
<td>56,062</td>
<td>152,982</td>
</tr>
<tr>
<td>2,560</td>
<td>96,984</td>
<td>112,982</td>
<td>307,664</td>
</tr>
<tr>
<td>5,120</td>
<td>195,665</td>
<td>227,664</td>
<td>6.01</td>
</tr>
</tbody>
</table>

Fig. 4. Prevalences during 72 infection cycles of infected pigs \( C(t) \) and susceptible pigs \( S_2(t) \), after one first pig is infected in a population of 1,000 pigs, under the mosquito density, per pig per night, changing in time in infection cycle but without disappearance.
stant numbers of mosquitoes during 3, 5 and 10 infection cycles, one infection cycle being 0.5 month. It is apparent from Table 2 that the number of transmissible mosquitoes is larger when the density of mosquitoes per pig per night is higher and also when the number of infection cycles is larger. It should be noted that the prevalences of infected pigs and also of immune pigs did not differ much with the mosquito density if the number of mosquitoes per pig per night is 640 or more (see Figs. 1 and 2), nevertheless the number of transmissible mosquitoes increases steadily with the increase of the mosquito density, even if the mosquito density is 640 or more. The number of transmissible mosquitoes is considered roughly proportional to the number of human cases if other conditions are the same, therefore the higher the density of mosquitoes feeding on pigs, generally the greater the danger of man to the infection. Reversely, it may be misleading if we suppose the intensity of the human epidemic only from the state of the pig infection. Of course, we can suppose with reasonable certainty that the small number of human cases only will occur if the number of infected pigs is so small that fairly large proportion of pigs are still susceptible.

The numbers of transmissible mosquitoes when the number of feeding mosquitoes changes with time are given in Table 1 and Fig. 3C. The number of transmissible mosquitoes is, in general, larger, under higher densities of feeding mosquitoes, and also is influenced by the starting time of the epizootic. Although the number of infected pigs did not differ much if only the epizootic has started before the peak time of the mosquito prevalence, the number of transmissible mosquitoes differs clearly between the starting times $t=0$ and $t=2$, in spite of both being before the mosquito peak time. Taking the total number of transmissible mosquitoes, it is 70,106 when the epizootic has started at $t=0$, while it is 44,443 when the starting time has been at $t=2$. Thus, the latter cases yields only 63% transmissible mosquitoes of the former case. If the epizootic has started just after the peak time of the mosquito prevalence (at $t=4$), the number of transmissible mosquitoes very remarkably decreases to 1,472, and the pig epizootic which has started at $t=6$ produces only 4 transmissible mosquitoes.

As mentioned earlier, the danger of man to the infection is roughly proportional, in general to the number of transmissible mosquitoes, and therefore it is extremely important in determining the intensity of the human epidemic when the pig epizootic starts in relation to the mosquito prevalence. Also, we can say that it will be rather difficult to presume the intensity of the epidemic of man by the prevalence of infected pigs (this has already been indicated by the cases when the density of feeding mosquitoes is constant throughout the process of the pig epizootic). Here, it should be noted that the starting time of the pig epizootic is important in influencing the human epidemic only in a general sense. It would be rather meaningless to presume the intensity of the human epidemic merely through the starting time of the epizootic in pigs in relation to the mosquito prevalence, without the consideration of the level of mosquito density. It is apparent from Table 1 that even when the pig epizootic has started at the same time in relation to the peak time of the mosquito prevalence, $t=0$, the number of transmissible mosquitoes (and therefore the danger of infection in man) is greatly different with the abundance of mosquitoes, and in the extreme case the number of transmissible mosquitoes may be even larger when the pig epizootic has started after the
peak of the mosquito prevalence than when the epizootic has started before the peak (compare Table 1 and Fig. 3C).

**INFECTION RATE OF MOSQUITOES**

Two kinds of infection rate of mosquitoes will be taken into consideration. One is the infection rate among those mosquitoes which are fresh from feeding on pigs (referred to as immediate infection rate hereinafter), and the other is the rate of transmissible mosquitoes among those mosquitoes which are about to feed on pigs (transmissible mosquito rate). The latter will be mentioned first.

When the number of mosquitoes per pig per night is constant in relation to time, it is apparent from Expression (7) that

\[ R(t+1) = \frac{C(t) \times \rho'}{n} \]

\[ = 0.00005 \times C(t). \]

Therefore, the transmissible mosquito rate is directly proportional to the number of infected pigs one infection cycle before. However, the constant mosquito density is usually not realized in nature, and the situation becomes more complex when the mosquito density changes with time.

The prevalence of transmissible mosquito rates after one first pig is infected in a population of 1,000 pigs at various times in relation to the mosquito prevalence is given in Table 1 and Fig. 3D. It is apparent from Table 1 that the transmissible mosquito rate in percentage, \( R(t) \times 100 \), does not differ so remarkably as the number of transmissible mosquitoes does. It is also shown that in each example the highest percentage does not always appear at the time when the number is largest. For instance, in Example 4 the number of transmissible mosquitoes at \( t=3 \) is 138,771 which is largest, but the highest percentage of 10.34 appears at \( t=4 \), not at \( t=3 \). Fig. 3D shows that though the transmissible mosquito rate in percentage differs with different times of the first pig infection, the difference in the rate is not so great as in the number of transmissible mosquitoes (see Fig. 3C), and in fact the prevalences of transmissible mosquito rate when the first pig infection has occurred at \( t=0, 2 \) and 4 are similar one another.

The above can be understood by observing the model. The transmissible mosquito rate \( R(t+1) \) is given by Expression (7)

\[ R(t+1) = T(t+1) \frac{T(t+1)}{M(t+1) \times n}, \]

and by Expression (6), \( T(t+1) \) is

\[ T(t+1) = M(t) \times C(t) \times \rho', \]

therefore

\[ R(t+1) = \frac{M(t) \times C(t) \times \rho'}{M(t+1) \times n}. \]
From this, it is clear that $R_{t+1}$ is related proportionally to the ratio of $M(t)$ to $M(t+1)$ and to the number of infected pigs. This implies that when the mosquito population is increasing the transmissible mosquito rate is lower than when decreasing, under the presence of the same number of infected pigs. This, in turn, indicated that the intensity of the epizootic can not be estimated only by the transmissible mosquito rate.

Unlike the transmissible mosquito rate, the immediate infection rate of mosquitoes is independent on the mosquito prevalence. For example, if there are 4 infected pigs showing viremia in a population of 1,000 pigs, the immediate infection rate is $4/1,000$, since the number of feeding mosquitoes per pig per night is the same in any pigs. In other words, it can be said that the immediate infection rate of mosquitoes is the same as the rate of infected pigs.

**Threshold Densities of Mosquitoes and Susceptible Pigs**

It may be understandable that the epizootic will not expand further, if the number of mosquitoes or susceptible pigs is extremely small. Thus, we can assume the threshold density of mosquitoes or susceptible pigs below which the epizootic tends to disappear. This problem can also be analyzed by the aid of the model for the epizootic in pigs.

The threshold density of mosquitoes will be firstly considered. For the sake of simplicity is taken the case when the mosquito density is constant in relation to time. Under the conditions of $p'=0.05$ and $n=1,000$, Expression (13) becomes

$$C(t+1) = S_2(t+1) \times (1 - (1 - \frac{C(t)}{n} \times \frac{p'}{n})^W)$$

$$= S_2(t+1) \times (1 - (1 - 0.00005 \times C(t))^W)$$

where $M$ is the number of mosquitoes per pig per night. The threshold density of mosquitoes is given as the maximum value of $M$ when $C(t) \leq C(0)$, regarding $t=0$. From Expressions (10), (9) and (4)

$$S_2(1) = N - A_2(1)$$

$$= N - A_1(0) \times (1 - \frac{d}{N})$$

$$= N - C(0) \times (1 - \frac{d}{N})$$

Since $N=750$ and $d=62.5$,

$$S_2(1) = 750 - 0.9167 \times C(0)$$

and therefore

$$C(1) = (750 - 0.9167 \times C(0)) \times (1 - 0.00005 \times C(0))^W$$

From this, the maximum values of $M$ for $C(1) \leq C(0)$, when various numbers of pigs are initially infected, were calculated and shown in Table 3.

It is indicated from Table 3 that the threshold density of mosquitoes given as the number of mosquitoes per pig per night is slightly less than 30, unless the initial number of infected pigs is very large. If the mosquito is below the threshold density, we can expect that no epizootic in pigs nor epidemic in man will occur, though an extremely small
Table 3. Threshold density of mosquitoes for the extinguishment of pig epizootic after a certain number of pigs are initially infected in a population of 1,000 pigs, expressed as the number of feeding mosquitoes on one pig in one night

<table>
<thead>
<tr>
<th>Initial No. of infected pigs</th>
<th>1</th>
<th>2</th>
<th>4</th>
<th>8</th>
<th>16</th>
<th>32</th>
<th>64</th>
<th>128</th>
<th>256</th>
</tr>
</thead>
<tbody>
<tr>
<td>Threshold No. of mosquitoes per pig per night</td>
<td>26</td>
<td>26</td>
<td>26</td>
<td>27</td>
<td>27</td>
<td>28</td>
<td>30</td>
<td>35</td>
<td>53</td>
</tr>
</tbody>
</table>

scale of the epizootic or the epidemic may reappear as a result of the accumulation of susceptible pigs which have lost maternal antibody.

As for the threshold density of susceptible pigs, the model for JE epizootic when the mosquito density is constant was again applied. Suppose that among 750 pigs, which are without maternal antibody, x pigs are immune. Substituting C(0)+x for C(0) in Expression (34), and regarding C(0)=1 (the initial number of infected pigs is usually very small), Expression (35) becomes

$$C(1) = \left( 750 - 0.9167 \times (C(0) + x) \right) \times \left( 1 - \left( 1 - 0.00005 \times C(0) \right)^{M} \right)$$

$$= \left( 750 - 0.9167 \times (1+x) \right) \times \left( 1 - 0.99995^{M} \right). \tag{36}$$

The threshold number of immune pigs under a given mosquito density of $M$ is obtained as a maximum value of $x$ for $C(1) \leq 1$ in Expression (36). The threshold densities of immunized pigs under various mosquito densities were calculated in this way and given in Table 4. The threshold density of susceptible pigs below which the epizootic tends to disappear can be obtained by subtracting the threshold number of immune pigs shown in Table 4, from 749, since one pig is infected and $x$ pigs are immune among 750 pigs which are involved in the infection cycle.

From Table 4, it is seen that the threshold number of immune pigs is 273 when the number of mosquitoes per pig per night is 40, and the threshold number becomes large with

Table 4. Threshold density of immune pigs for the extinguishment of pig epizootic after one pig is initially infected in a population of 1,000 pigs under various constant numbers of mosquitoes feeding on one pig in one night

<table>
<thead>
<tr>
<th>No. of mosquitoes per pig per night*</th>
<th>40</th>
<th>80</th>
<th>160</th>
<th>320</th>
<th>640</th>
</tr>
</thead>
<tbody>
<tr>
<td>Threshold No. of immunized pigs</td>
<td>273</td>
<td>545</td>
<td>680</td>
<td>748**</td>
<td>782***</td>
</tr>
</tbody>
</table>

* When the number of mosquitoes per pig per night is 26 or less, pig epizootic will tend to disappear even if no pigs are immune (See Table 3).

** In the infection cycle 750 pigs are involved, in a population of 1,000 pigs, because remaining 250 pigs have maternal antibody, therefore if the number of mosquitoes per pig per night is 320, almost all pigs must be immune for epizootic extinguishment.

*** From the reason seen in**, 782 is actually a non-existent figure; this must be understood to show that even if the all pigs are immune, a small number of pigs will be infected owing to the addition of susceptible pigs to the pig population by the disappearance of maternal antibody.
the increase of the mosquito density. When the mosquito density is 640 per pig per night, 748 pigs, i.e., almost all pigs must be immune for the epizootic to tend to disappear. When the mosquito density is 640, 782 was calculated as the threshold number of immune pigs, but 782 is actually a non-existent value. This must be understood to show that even if the all pigs are immune a small number of pigs will be infected owing to the introduction of susceptible pigs to the pig population by the disappearance of maternal antibody. Of course, if the mosquito is below the threshold mosquito density, the epizootic will tend to disappear without any immune pigs.

**ARTIFICIAL PIG IMMUNIZATION AND PREVENTIVE MEASURES FOR JE**

As the preventive measures for JE, three can be considered, (1) against humans, (2) against amplifying animals, and (3) against vector mosquitoes. The second one is the artificial immunization of pigs which are the most important amplifying animal, and the considerations on this will be given firstly. And then, the preventive measures for JE will be collectively mentioned.

Buescher & Scherer (1959) pointed out that measures against amplifying animals should be considered as preventive ones for JE from a result of epidemiological surveys in Japan. Since then, the pig has taken a position as the most important amplifying animal, and studies have been carried out on the effects of artificial pig immunization (Oya, 1967; Takahashi et al., 1968; Tsuchiya et al., 1970; Ueba et al., 1972). However, it is not easy to evaluate the effects from the data obtained in the field, because the epizootic of pigs or the epidemic of man is controlled by many complicated factors. In such a case, it was thought that again the model for JE can effectively be used.

Suppose that at time $t$, $C(t)$ pigs are firstly infected and $x$ pigs are artificially immunized, Expression (20) becomes

$$A_1(t, i) = C(t, i) + \frac{x}{j}.$$  

(37)

Using Expression (37) in place of Expression (20), we can calculate the epizootic process of pigs. Fig. 5 shows the effect of the pig immunization on the prevalence of infected pigs. In Fig. 5, under the mosquito densities of 40, 160, 640 and 2,560 per pig per night, the prevalences of infected pigs when all and a half pigs have been immunized artificially are compared with the prevalence when no pigs have been immunized. In a population of 1,000 pigs at $t=0$, 250 have maternal antibody and one is infected, therefore all pigs and a half pigs mean 749 and 374.5 pigs, respectively.

It is clearly seen from Fig. 5 that when no pigs are artificially immunized, generally the infected pigs are larger in number and earlier in appearance under higher mosquito density (see Fig. 1), and when all pigs are immunized the number and the appearance of infected pigs becomes smaller and later, respectively, the situation by the immunization of a half pigs being the intermediate between the two cases.

According to the previous section where the threshold density of immune pigs was
mentioned, the pig epizootic will tend to disappear, if 273 or more pigs are immune, or artificially immunized, when the mosquito density per pig per night is 40, or if 680 or more are immunized when the mosquito density is 160. Therefore, it is expected that the epizootic tends to disappear if a half pigs and all pigs are immunized when the mosquito density is 40 and 160, respectively. However, this is the tendency only between \( t=0 \) and \( t=1 \), and as new susceptible pigs are continuously added to the pig population with the progress of time, the epizootic occurs before long, as seen in Fig. 5. When the mosquito density is 640
or more per pig per night, the immunization of all pigs is still not enough for the pig epi-
zootic to proceed toward the extinguishment, but it should be noted that the number of in-
fected pigs becomes smaller by the pig immunization.

The prevalences of the rates of immune pigs (infected pigs inclusive) are shown in
Figs. 6 and 7, when a half (374.5) pigs and all (749) pigs are artificially immunized at
\( t=0 \), respectively. The solid lines drawn in Figs. 6 and 7 indicate the rates of immune
pigs in all the 750 pigs (all pigs excepting 250 with maternal antibody in a population of
1,000 pigs), \( \frac{A_2(t)}{750} \) and \( \frac{A_1(t)}{750} \), and the broken lines those in slaughtered pigs, \( \frac{A_2(t,12)}{62.5} \)
and \( \frac{A_1(t,12)}{62.5} \).

If a half pigs are immunized at \( t=0 \), as seen from Fig. 6, the prevalences of the
immune pig rate all start from the point of ca. 50%, but the prevalences thereafter are quite
different with the mosquito density. As the pig infection hardly occurs when the mosquito
density is 40 per pig per night (see Fig. 5), the rate of immune pigs in the 750 pigs de-

---

**FIG. 6. A HALF PIGS ARE IMMUNIZED**

**FIG. 7. ALL PIGS ARE IMMUNIZED**

Figs. 6 and 7. Effect of pig immunization at \( t=0 \) on the prevalence of immune pigs,
after the infection of one pig at \( t=0 \) in a population of 1,000 pigs on which various constant numbers of mosquitoes feed. Fig. 6: A half pigs are immunized; Fig. 7: All pigs are immunized. See Fig. 2 for solid and broken lines, and see Fig. 5 for a half pigs and all pigs.
creases constantly and finally reaches nearly 0% at \( t=12 \). On the other hand, the rate in slaughtered pigs keeps ca. 50% till \( t=11 \) and sharply drops to ca. 0% at \( t=12 \). This means that all the artificially immunized pigs have been slaughtered by that time.

As for the prevalences of the immune pig rates when the mosquito densities are 160 or more per pig per night, it is apparent that the rise in the rate toward 100% is earlier under the higher mosquito density. The rates in the 750 pigs and in slaughtered pigs change generally in parallel, but such decreases as seen in the rate in the 750 pigs (solid lines) between two succeeding times in infection cycle, particularly at the mosquito density of 160, are not observed in the rate in slaughtered pigs (broken lines).

Fig. 7 shows the prevalences of the rates of immune pigs when all pigs are artificially immunized at \( t=0 \). The general tendency of the prevalences is similar to the cases when a half pigs are immunized, excepting that conspicuous reduction of the immune pig rate in the 750 pigs (solid line) is observed at the mosquito density of 160.

Turning to the preventive measures for JE, the effect can be assessed by the total number of transmissible mosquitoes produced, \( T(t) \), which is considered proportional to the number of human cases if other conditions remain constant. Therefore, in Table 5 is shown the reduction in the total number of transmissible mosquitoes produced in the process of pig epizootic by the pig immunization.

The number of transmissible mosquitoes produced from pig infection at time \( t \), \( T(t+1) \) is given by Expression (6) as

\[
T(t+1) = M(t) \times C(t) \times p^t
\]

which indicates that the number of transmissible mosquitoes is proportional to the number of infected pigs, \( C(t) \). The infected pigs change by the pig immunization not only in the number, but also in the prevalence, as seen in Fig. 5. For this reason, the total numbers of transmissible mosquitoes produced during 3 and 5 infection cycles are given in Table 5.

It has already been indicated in the earlier section that when no pigs are immunized the larger number of transmissible mosquitoes are produced under the higher mosquito density. Also, it is easily understood that the total number of transmissible mosquitoes is larger during longer time. Keeping these in mind, Table 5 clearly shows that the total

---

**Table 5.** Decreases of the numbers of transmissible mosquitoes during 3, 5, and 10 infection cycles by immunizing a half and all pigs at \( t=0 \) when a first pig is infected in a population of 1,000 pigs

<table>
<thead>
<tr>
<th>No. of mosquitoes per pig per night</th>
<th>No. of transmissible mosquitoes during 3 cycles when the indicated No. of pigs* are immunized</th>
<th>No. of transmissible mosquitoes during 5 cycles when the indicated No. of pigs* are immunized</th>
<th>No. of transmissible mosquitoes during 10 cycles when the indicated No. of pigs* are immunized</th>
</tr>
</thead>
<tbody>
<tr>
<td>40</td>
<td>0 374.5 749</td>
<td>0 374.5 749</td>
<td>0 374.5 749</td>
</tr>
<tr>
<td>80</td>
<td>10 5 2</td>
<td>26 8 2</td>
<td>196 19 3</td>
</tr>
<tr>
<td>160</td>
<td>335 123 16</td>
<td>5,071 1,400 34</td>
<td>7,550 5,634 31</td>
</tr>
<tr>
<td>320</td>
<td>2,263 799 64</td>
<td>13,103 8,204 497</td>
<td>17,334 12,515 81</td>
</tr>
<tr>
<td>640</td>
<td>13,173 5,031 337</td>
<td>27,163 17,403 6,137</td>
<td>36,870 27,108 17,672</td>
</tr>
<tr>
<td>1,280</td>
<td>45,988 22,918 2,025</td>
<td>56,062 35,790 16,062</td>
<td>75,997 55,716 35,990</td>
</tr>
<tr>
<td>2,560</td>
<td>96,984 56,462 10,416</td>
<td>112,902 72,547 32,128</td>
<td>132,982 112,547 72,125</td>
</tr>
<tr>
<td>5,120</td>
<td>195,665 113,960 31,505</td>
<td>227,664 145,960 64,255</td>
<td>307,664 225,959 144,255</td>
</tr>
</tbody>
</table>

* In a population of 1,000 pigs, 250 have maternal antibody and one is infected, therefore 749 and 374.5 pigs mean the immunization of all and a half pigs, respectively.
number of transmissible mosquitoes, and therefore the danger of infection in man, can be re-
duced greatly by the pig immunization. Of course, the degree of the reduction is grea-
ter when the number of artificially immunized pigs is larger. Thus, the pig immunization
can be considered very effective to reduce the number of transmissible mosquitoes, neverthe-
less it should be pointed out that the reduced number is still fairly large when the mosquito
density is very high.

It was mentioned that the number of transmissible mosquitoes are reduced by two me-
thods, the decrease of the mosquito density and the pig immunization. The effects in
reducing the number of transmissible mosquitoes are compared, in the below, between these
two methods. For example, when the mosquito density is 5,120 per pig per night, the
number of transmissible mosquitoes during 3 infection cycles if all pigs are immunized is
31,505, which lies between 13,175 and 45,986 (the numbers of transmissible mosquitoes
under the mosquito densities of 640 and 1,280 respectively, when no pigs are immunized).
Namely, the immunization of all pigs has the same effect as seen when the mosquito density
is reduced to 1/8–1/4. Examining Table 5 in this way, it can be said that the immu-
niazation of all pigs will reduce the number of transmissible mosquitoes to such an extent as
the mosquito density is lowered to 1/8–1/4 during 3 infection cycles and to 1/4–1/2 during
5 cycles, and about 1/2 during 10 cycles. As the duration of the pig epizootic is usual-
ly less than 2 months (4 infection cycles) in Japan, the effect of the immunization of all pigs
is approximately equal to the effect of the reduction in mosquito density to about 1/2.

In the above is considered the case when one pig is infected at t=0 and simul-
taneously pigs are immunized. The pig immunization has little effect on the prevention
of JE epizootic or epidemic, if it is done after many pigs have already been infected. How-
ever, the prediction of the starting time of pig epizootic in nature is very difficult at the
present time, and for this reason, there is no way except for immunization of pigs suffi-
ciently before the actual epizootic. If they do so, some immunized pigs will be slaughtered
and the same number of susceptible pigs will be added to the pig population between the
period from the immunization to the start of the epizootic. Because this seems unavoidable,
the actual effect of the pig immunization has to be rather discounted.

When no pigs are immunized, larger number of transmissible mosquitoes are produced
under higher density of feeding mosquitoes, as repeatedly mentioned. This is clearly shown
in Fig. 8, in which the numbers of transmissible mosquitoes during 3 and 5 infection
cycles are plotted against the number of feeding mosquitoes per pig per night, based on the
figures given in Table 5. It is interesting that the number of transmissible mosquitoes
is very small and may be regarded actually as zero, if the density of feeding mosquitoes per
pig per night is 80 or less. Since the threshold density of mosquitoes below which the pig
epizootic will tend to extinguish is 26 (see Table 3), it is shown that even when the number
of feeding mosquitoes is slightly higher than the threshold density, the number of transmissi-
ble mosquitoes is negligibly small, and therefore human cases are scarcely expected, if the
mosquito density per pig per night is 80 or less. Such a mosquito density is invariably shown
also when pigs are immunized, though the value may be a little different.
Lastly, the effect of the vaccination to man will be mentioned. In the case of an infectious disease of man itself, the introduction of infectious persons into a community would not give rise to an epidemic outbreak if the density of susceptibles were below a certain critical value, and on the other hand, if the critical value were exceeded then there would be an epidemic of magnitude sufficient to reduce the density of susceptibles as far below the threshold as it originally was above (Bailey, 1957). But, in JE there does not exist the threshold density of susceptible persons, because, as stated in the earlier section, JE is not considered a disease of man in the sense that man is not involved in the infection cycle of JE. The vaccination to man, however effective to the protection from the infection, can not influence the infection cycle between pigs and mosquitoes.

OTHER FACTORS THAN MOSQUITO DENSITY

Because it seems that the epizootic in pigs and the epidemic in man are most greatly influenced by the mosquito density, other factors have been kept constant in the preceding sections. However, some of them are apparently variable and according to their values the epizootic or the epidemic is appreciably affected. Among those factors, the survival rate of mosquitoes is firstly considered.

Theoretical values for the pig epizootic and the human epidemic were calculated in the previous sections by using 0.05 as the mosquito survival rate \( p' \) through the period of 10 days \( k \) for the infected mosquito to become transmissible. This is based on the relation \( p' = p^k \), where \( p \) is the daily survival rate of mosquitoes. Therefore, a slight increase or decrease in \( p \) gives rise to a great change in \( p' \), as given in Table 6. If \( p \) increases by 0.1 within the range from 0.6 to 0.9, \( p' \) becomes approximately 4 times large. And the
Table 6. Relation between survival rates of mosquitoes in one day ($p$) and in 10 days ($p'$).

<table>
<thead>
<tr>
<th>In one day ($p$)</th>
<th>In 10 days ($p'$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.6</td>
<td>0.0060</td>
</tr>
<tr>
<td>0.7</td>
<td>0.028</td>
</tr>
<tr>
<td>0.8</td>
<td>0.11</td>
</tr>
<tr>
<td>0.9</td>
<td>0.35</td>
</tr>
</tbody>
</table>

*Period from the infection of the mosquito to the time of becoming transmissible (= $k$)*

increase of $p'$ results in the increase of the number of transmissible mosquitoes, which is directly proportionate to $p'$ as given by Expression (6), thus the epizootic in pigs is affected.

Table 7 shows the numbers of transmissible mosquitoes when the daily survival rate of mosquitoes ($p$) is different. It is apparent that much larger number of transmissible mosquitoes are produced with the increase in the daily survival rate as well as in the number of mosquitoes. It can be said that in producing transmissible mosquitoes the increase of the daily survival rate by 0.1 is approximately equivalent to four times the increase of the number of feeding mosquitoes. A larger number of transmissible mosquitoes are responsible for larger numbers of infected pigs and of human cases as well, since the number of human cases is proportional to the number of transmissible mosquitoes if other conditions are same.

It is also implied from Table 7 that the change in the daily survival rate of mosquitoes will affect the threshold densities of mosquitoes and immune pigs for the extinguishment of pig epizootic. Therefore, threshold densities were calculated as before, and shown in Tables 8 and 9. The threshold density of mosquitoes (Table 8) is very greatly different with the value of the daily survival rate ($p$); it is as large as 200 odd when $p$ is 0.6, but only 4 when $p$ is 0.9. A remarkable change is also seen in the threshold density of immune pigs (Table 9). When $p$ is 0.6, the pig epizootic tends to disappear even if no pigs are immune at mosquito density of 160 or less, but when $p$ is 0.9, 504 or more pigs have to be immune for the epizootic extinguishment at mosquito density of only 10. Also, a small number of pigs are infected owing to the addition of susceptible pigs to the pig popu-
Table 8. Threshold density of mosquitoes, when the daily survival rate of mosquitoes (p) is different, for the extinguishment of pig epizootic after a certain number of pigs are initially infected in a population of 1,000 pigs, expressed as the number of feeding mosquitoes on one pig in one night

<table>
<thead>
<tr>
<th>Initial No. of infected pigs</th>
<th>Threshold density of mosquitoes when p′ is</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.6</td>
<td>0.7</td>
</tr>
<tr>
<td>1</td>
<td>202</td>
</tr>
<tr>
<td>2</td>
<td>213</td>
</tr>
<tr>
<td>4</td>
<td>220</td>
</tr>
<tr>
<td>8</td>
<td>221</td>
</tr>
</tbody>
</table>

Table 9. Threshold density of immune pigs, when the daily survival rate of mosquitoes (p) is different, for the extinguishment of pig epizootic after one pig is initially infected in a population of 1,000 pigs under various constant numbers of mosquitoes feeding on one pig in one night

<table>
<thead>
<tr>
<th>No. of mosquitoes per pig per night</th>
<th>Threshold density of immune pigs when p is</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.6</td>
</tr>
<tr>
<td>10</td>
<td>*</td>
</tr>
<tr>
<td>20</td>
<td>*</td>
</tr>
<tr>
<td>40</td>
<td>*</td>
</tr>
<tr>
<td>80</td>
<td>*</td>
</tr>
<tr>
<td>160</td>
<td>*</td>
</tr>
<tr>
<td>320</td>
<td>253</td>
</tr>
<tr>
<td>640</td>
<td>534</td>
</tr>
<tr>
<td>1,280</td>
<td>675</td>
</tr>
<tr>
<td>2,560</td>
<td>746</td>
</tr>
<tr>
<td>5,120</td>
<td>**</td>
</tr>
</tbody>
</table>

* Pig epizootic intends to disappear even if no pigs are immune.
** Even if all pigs are immune, a small number of pigs are infected owing to the addition of susceptible pigs to the pig population by the disappearance of maternal antibody.

The importance of the daily survival rate of mosquitoes in affecting the pig epizootic as well as the human epidemic is clearly indicated in the above. The survival rate of mosquitoes is also an important factor controlling the mosquito population itself. Thus, studies on the mosquito survival rate in the field are highly required. In this connection, the effect of light traps set at pigsties should be reassessed in that they kill many engorged mosquitoes which include potentially infected ones, and in this way reduce the number of transmissible mosquitoes.

It was assumed in the present theoretical model for pig epizootic that mosquitoes feed only on pigs. However, this is not the case in the field. Generally speaking, how frequently a particular animal is fed on by the vector mosquito of JE depends, besides the mosquito density, on the host preference of the mosquito as well as the availability of that animal in the area concerned, therefore the frequency of mosquito bites in pigs very likely varies from area to area. It is necessary in analyzing field data to adjust the number of mosquitoes according to the rate of mosquitoes feeding on pigs.
It was also assumed in the model that the probability of being bitten by a mosquito is the same in any pig. This is again not valid in nature, and the number of feeding mosquitoes is subject to a great change among pigs, particularly at different pigsties. Its effect on the pig epizootic would be significant, when the general mosquito density is low, and a small scale of epizootic may occur even when the density of mosquitoes is lower than the threshold density. It is extremely difficult to estimate the frequency distribution of mosquito bites in pigs, however the distribution pattern is probably an aggregated type.

The size of pig population in the area concerned is very important in determining the absolute number of transmissible mosquitoes, and accordingly the danger for human infection. Therefore, it seems advisable to compare the general densities of vector mosquitoes and amplifying pigs in different countries in relation to JE epidemiology. Other factors affecting the number of human cases are such as the rate of mosquitoes that feed on man in one bite, the rate of people in the area who are susceptible to the virus, and the rate of people among the susceptible population who develop clinical symptoms after infection (Sasa, 1971). Those factors are important to understand the human epidemic, however the knowledge of pig epizootic is apparently prerequisite for it.

DISCUSSIONS

It has been mentioned in the present paper how the epizootic in pigs and accordingly the epidemic in man is influenced by the mosquito density, and other factors, by using a mathematical model for JE. It must be evaluated by the comparison with field data whether the results obtained by simulation studies with the model are true or not. If there is a great deviation between the actual epizootic or epidemic process observed in the field and the theoretical process obtained by the model, it is considered that some of the assumptions and/or conditions used in developing the model are not appropriate, and by modifying them we shall become to understand the natural events more rightly. The threshold density of mosquitoes or artificially immunized pigs, of course, can not be an exception. However, the concept of the threshold density is very important to understand the epidemiology.

In the present paper, it has been dealt with how the epizootic or epidemic will proceed after a pig or pigs are once infected. When the epizootic and subsequently the epidemic will begin in each year is another problem to be studied, which is probably related to the overwintering of JE virus.

The number of JE human cases has decreased greatly in recent years in Japan. The model for JE clearly shows that the number of human cases will decrease if the mosquito density becomes low, and in fact there is a very clear relation between the mosquito densities and the numbers of human cases, at least in Nagasaki Prefecture from 1965 to 1972. The vaccination to man may have probably played a role to a certain extent in reducing human cases, but this can not be an only reason, since some changes have occurred also in the epizootic in pigs. Thus, it seems certain that the lowered mosquito density is, at least partly, responsible for the reduction in the number of human cases. The results of analyses of field data by the model will be published in other papers in preparation.
REFERENCES


日本脳炎流行の理論的考察
和田義人（長崎大学医学部医動物学教室）

日本脳炎は本質的には豚の病気であるとの考えから、豚における流行の数学的モデルを作って、これを人における流行と関連づけた。このモデルを用いてのシミュレーションから、以下に述べるような結論が明らかとなったが、その多くは他の方法では理解が容易でないものである。伝搬動物の密度と人との感染を受ける危険性との相関があるが、逆に豚の感染状況から人感染の危険性を論ずるのは、多くの場合無理である。豚の消化のどの時期から豚の感染が始まるかによって、その後の豚及び人での流行様相は大きく変わる。豚での流行がそれ以上拡大したための、豚及び感染性豚の限界密度が存在する。豚の日生存率が大きくなると、豚の密度が高くなった場合と同じように、豚及び人での流行は
大きく影響を受ける。豚の人工免疫によって、感染を起す豚数は減少し、抗体保有豚の率は高くな る。また、人工免疫によって、人が感染を受ける危険性を大きく減少させることができるが、日本脳 炎の予防対策としては、特に伝搬経の密度が高い場合には、それだけでは不充分である。

熱帯医学、第16巻第4号171-199頁、1975年2月。