ON THE ANALYSIS OF EPIDEMIC MODEL I (THEORETICAL APPROACH)

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1. Introductory remarks and basic ideas.

The deterministic continuous-time model of epidemic was studied by Mckendrick and Kermack (1927). A new impetus has been given to the study of epidemic since 1940. Bartlett (1949) and Bailey (1950, 1955) have introduced the idea of stochastic process to their deterministic model and developed a partial differential equation for the probability generating function of two variables, viz., the numbers of susceptibles and infectives.

They take the assumption that infection and removal from circulation occur as random events in continuous time, and a newly infected indiuidual is infectious to other susceptibles until the infective is removed by death, recovery or isolation. So various results have been developed by Bailey (1950, 1953, 1955) both for this general case and the simpler one involving no removal.

The social group in the epidemic model must be based on the assumption of homogeneous mixing. The most epidemiological phenomena must depend only on the homogeneous contact between hosts and infecting organisms. As the actual problem, we have some doubts about this assumption, but we cannot help doing so theoretically because the reliable information on the nature, density and the mode of transmission of infective agent is fairly meagre. This kind of information must be more precisely studied by medical experimentation. If this problem is solved, we shall be able to give a more exact epidemic model.

In any case, it seems reasonable that epidemics actually occur in several relatively small social groups in which the assumption of homogeneous mixing is satisfied. Bailey (1956) concentrated his attention to the small scale epidemics in family groups after one of the members contracted the disease from outside, and analysed the data of Hope Simpson. As the chances of cross-infection within the family are usually fairly high, it will perhaps be practical to study these

intra-familial epidemics. From his data on the measles, we can see that the cases of cross-infection within the family are not so many and, rather, of rare occurrences in that area. But such phenomena may originate in the incomplete report of measles.

In the present paper, we shall construct some epidemic models in the community where the assumption of homogeneous mixing is satisfied.

We treat only the diseases that are infectious in the sense of being communicable at the appropriate stage of the development in an infected individual by adequate contact with a susceptible. As soon as infecting organisms enter into the body by the adequate contact, the infecting organisms undergo certain biological developments within the body during the latent period, but any kind of infectious material is is not exhaled. The receipt of infection by adquate contact is a rather broad concept used to introduce the probability element. And at some time in the individual's history of infection, recognizable symptoms may appear. With acute infections these symptoms are the signals for isolating the case from the community until patient recovers and is, at least temporarily, immune from further infection.

We, also, assume *the chance of contact* to be the same for any pair of one infective and one susceptible. But we cannot state that the *chance of attack* is the same for all contact members because all ostensibly contact members are not necessarily all infective. That is, if the infective organisms enter into the body, the antigen and antibody will be constructed within the body. And if the quantity of the antibody constructed within the body is less than a fixed level, the body will be infected and some symptoms will appear. Inversely if the antibody is more, the body will not be infected by the infectious disease. Then, temporary immunity is conferred by attack of the infectious bacillus or virus, though its immunity is not generally permanent. And the body will not be infected for at least a certain length of period owing to the temporary immunity. We are supposing that this will be the characteristic of many infectious diseases. Surely, a very few persons will be infected for a period of the epidemic. For example, when we consider the epidemic period of such infectious diseases that encephalitis, infantile paralysis and etc., a very few may be infected.

Now, for simplicity, we shall assume the latent period to be reduced to zero in comparison with the infectious period when we construct our model. On this idea, we shall construct the stochastic epidemic model in Section 2, and illustrate numerically this model by Monte Carlo Method in Section 3.

The second idea in Section 4 is that we introduce the conditional probability which a infective infected at the time τ will recover in the time interval $(t, t+\Delta t)$. Our model in Section 2 contains some disadvantage that every infective recovers independently of the infected time.

In Section 5, we shall revise some classical treatments of epidemic from some practical stand points.

2. Stochastic treatment (I).

Let us assume that the epidemic is started by the introduction of a infectious individuals into a population of n susceptibles. We take the community, the size of which is $n+a$ individuals at $t=0$. Suppose that, at the time t, there are r susceptibles, s infectious cases in circulation and q individuals who are isolated, dead or recovered.

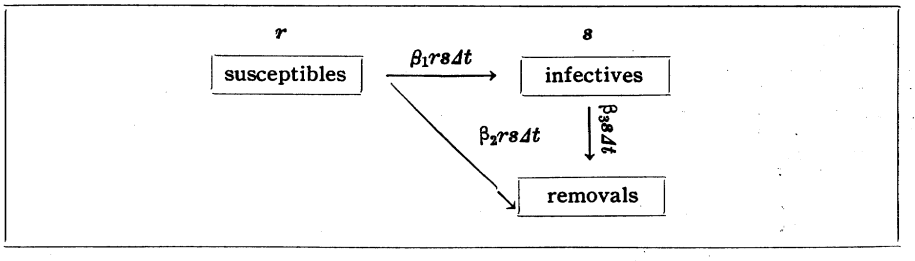


Fig. 1. Diagram is showing the relation between infections probability and removal probability in the time interval $(t, t+\Delta t)$ when there are r susceptibles and s infectives at the time t.

$\beta_2 r s \Delta t$ is the conditional probability that a susceptibe which accepted infectious baccilus from one infetive is not infected, but falls in the removal group (immune group) in the infinitesimal time interval $(t, t+\Delta t)$ when there are r susceptibles and s infectives at the time t.

Thus we have

$$r+s+q=n+a \qquad (1)$$

where

$$\begin{aligned} 0 &\leqq r \leqq n, \\ 0 &\leqq s \leqq n+a, \\ 0 &\leqq s+s \leqq n+a, \\ 0 &\leqq q \leqq n+a. \end{aligned} \tag{2}$$

Now suppose that, on the assumption of the homogeneous mixing of the susceptibles and infectious cases in circulation, the conditional probability of one new infection taking place in the infinitesimal time interval $(t, t+\Delta t)$ is given by $\beta_1 rs\Delta t+o(t)$. That is,

$$\Pr\{r-1, s+1 \text{ at the time } t+\Delta t/\text{given } r, s \text{ at the time } t\} = \beta_1 rs\Delta t+o(\Delta t), \tag{3}$$

where β_1 shows constant infection rate and the symbol $o(\Delta t)$ stands for a quantity which tends zero faster than Δt. Similarly, suppose that that, the conditional probabilty of one new removal (one immune case) from the group of r susceptibles taking place in the infinitesimal time interval $(t, t+\Delta t)$ is given by $\beta_2 rs\Delta t+to(\Delta t)$. As stated in Section 1, when a susceptible is very healthy, he would not be infected if the infectious organisms entered into his body. Then, he has, rather, temporary immune. That is, $\beta_2 rs\Delta t$ expresses the conditional probability by which one susceptible falls in the removal group directly from susceptible group in the time interval $(t, t+\Delta t)$. Next, Suppose the conditional probabitity of one infected person being removed from circulation in the time interval $(t, t+\Delta t)$ is given by $\beta_3 s\Delta t+o(\Delta t)$.

That is,

$$\Pr\{r-1, s \text{ at the time } t+\Delta t/\text{given } r, s \text{ at the time } t\} = \beta_2 sr\Delta t+o(\Delta t), \tag{4}$$

where β_2 shows contant removal rate from susceptibles, and

$$\Pr\{r, s-1 \text{ at the time } t+\Delta t/\text{given } r, s \text{ at the time } t = \beta_3 s\Delta t+o(\Delta t),$$

where β_3 constant removal rate from infectious cases.

Furthermore, let $P_{rs}(t)$ be the probability that at the time t there are r susceptibles still uninfected and s infectious cases in circulation. Putting $\Delta t \to 0$, the well-known procedure gives the following differential-difference equation from (3), (4), (5),

$$\left.\begin{aligned}\frac{dP_{rs}(t)}{dt}&=\beta_1(r+1)(s-1)P_{r+1,s-1}(t)+\beta_2(r+1)sP_{r+1,s}(t)\\&\quad-(\beta_1rs+\beta_2rs+\beta_3s)P_{rs}(t)+\beta_3(s+1)P_{r,s+1}(t)\ ,\\\frac{dP_{na}(t)}{dt}&=-(\beta_1n+\beta_2n+\beta_3)aP_{na}(t)\ ,\end{aligned}\right\}\qquad(6)$$

where the initial condition is $P_{na}(0)=1$. Using the probability generating function $\pi=\pi(u, v, t)=\sum_{r,s}u^r v^s P_{rs}(t)$, it is now easy to show that the whole process can be characterized by the partial differential equation for π:

$$\frac{\partial\pi}{\partial t}=(\beta_1v^2-\beta_1uv-\beta_2uv+\beta_2v)\frac{\partial^2\pi}{\partial u\partial v}+\beta_3(1-v)\frac{\partial\pi}{\partial v}\qquad(7)$$

with the boundary condition

$$\pi(u, v, \theta)=u^n v^a\ .\qquad(8)$$

If r, s are the independent random variables and thc mean of susceptibles is given by $\bar{\gamma}_t=e^{-\rho(t)}$ at the time t, the number of infections at the time t becomes the branching process. And the number of infectives s at the time t can be calculated easily. But it is, generally, difficult to to derive, rigorously, the solution of the partial differential equation (7) of hyperbolic type corresponding to the boundary condition (8). By the way, we shall illustrate numerically this model by the Monte Carlo Method.

3. Numerical illustration.

Let the time interval Δt in (3), (4), (5), be equal to a infinitesmal unit time. Then, we shall be able to derive the next relations from (3), (4), (5):

$$\begin{aligned}\lambda_1&=\Pr\{r-1,\ s+1 \text{ at the time } t+1/\text{giving } r, s \text{ at the time } t\}\\&=\beta_1rs\ ,\end{aligned}\qquad(9)$$

$$\begin{aligned}\lambda_2&=\Pr\{r-1,\ s \text{ at the time } t+1/\text{giving } r, s \text{ at the time } t\}\\&=\beta_2rs\end{aligned}\qquad(10)$$

$$\begin{aligned}\lambda_3&=\Pr\{r\ ,\ s-1 \text{ at the time } t+1/\text{giving } r, s \text{ at the time } t\}\\&=\beta_3s\end{aligned}\qquad(11)$$

$$\begin{aligned}\lambda_4&=1-(\lambda_1+\lambda_2+\lambda_3)\\&=\Pr\{r\ ,\ s \text{ at the time } t+1/\text{giving } r, s \text{ at the time } t\}\end{aligned}\qquad(12)$$

where r susceptibles and s infectives in carculation are at the time t. When we construct four domains, $(0, \lambda_1]$, $(\lambda_1+\lambda_2]$, $(\lambda_1+\lambda_2, \lambda_1+\lambda_2+\lambda_3]$, $(\lambda_1+\lambda_2+\lambda_3, 1)$ and we take a non-negative random number X_t from a uniform distribution on $(0, 1)$, we can decide r, s by finding a domain which X_t is belonging to ;

$r-1$ susceptibles and $s+1$ infectives at the time $t+1$
if $X_t \leqq \lambda_1$

$r-1$ susceptibles and s infectives at the time $t+1$
if $\lambda_1 \leqq X_t < \lambda_1+\lambda_2$

r susceptibles and $s-1$ infectives at the time $t+1$
if $\lambda_1+\lambda_2 \leqq X_t < \lambda_1+\lambda_2+\lambda_3$

or

r susceptibles and s infectives at the time $t+1$
if $\lambda_1+\lambda_2+\lambda_3 \leqq X_t$.

For example, if X_t belongings to the domain $(0, \lambda]$, we can derive λ_t at the time $t+1$.

That is,

$$\lambda_1=\beta_1(r-1)(s+1) \qquad (9')$$
$$\lambda_2=\beta_2(r-1)(s+1) \qquad (10')$$
$$\lambda_3=\beta_3(s+1) \qquad (11')$$
$$\lambda_4=1-(\lambda_1+\lambda_2+\lambda_3) \qquad (12')$$

where $r-1$ susceptibles and $s+1$ infectives in circulation are at the time $t+1$. Furthermose, we take a random number X_{t+1} and repeat the above procedure. And this procedure stops if $s=0$. By the method mentioned above, we calculated the 10 epidemic curves of r, s as the initial values $n=400$ susceptibles, $a=20$ infectives and the infection rates $\beta_1=10^{-5}$, removal rates $\beta_2=5\times10^{-6}$, $\beta_3=2\times10^{-3}$. By this method, we gave the mean epidemic curves $\bar{r}_t, \bar{s}_t$ of r susceptibles and s infectives at the time t as Fig. 2 and Fig. 3. The mean susceptible curve $\bar{r}_t$ decrerses monotonously, but the mean infective curve $\bar{s}_t$ rises fast up to the maximum point $\bar{s}_{t_m}$ and descends slowly from its point.

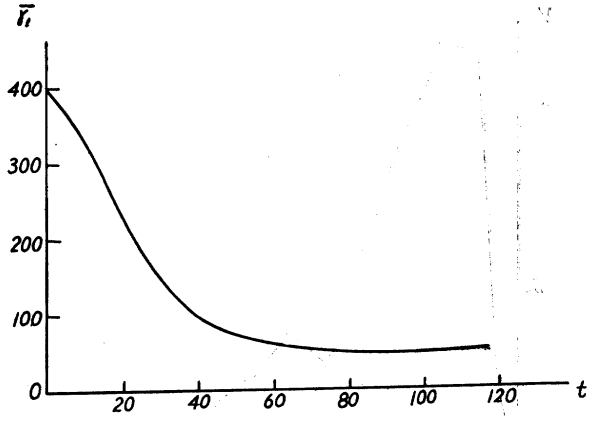
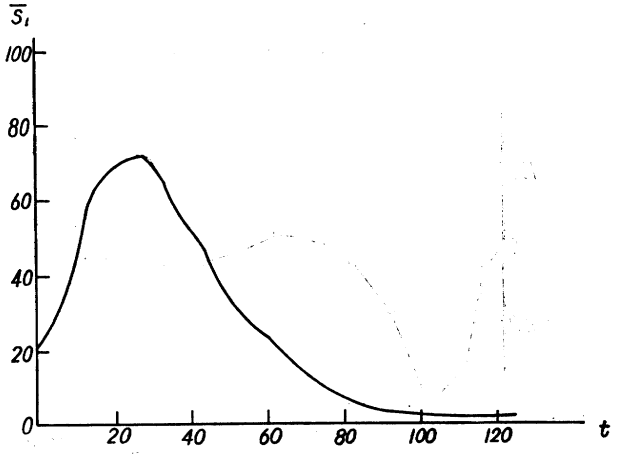


Fig. 2. r_t shows the mean number of susceptibles at the time t.



Eig. 3. $\bar{s}_t$ shows the mean number of infectives at the time t.

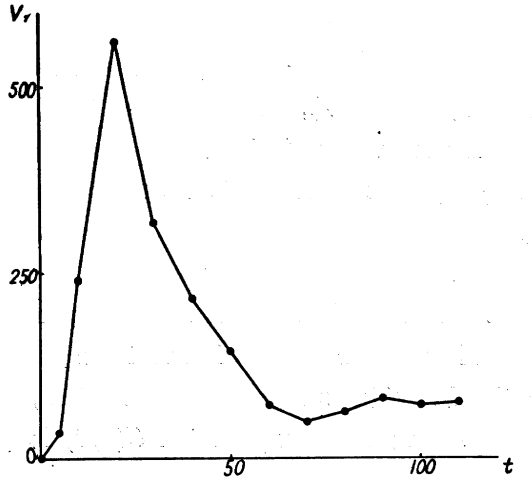


Fig. 4. V_r expresses the variance gf r at the time t.

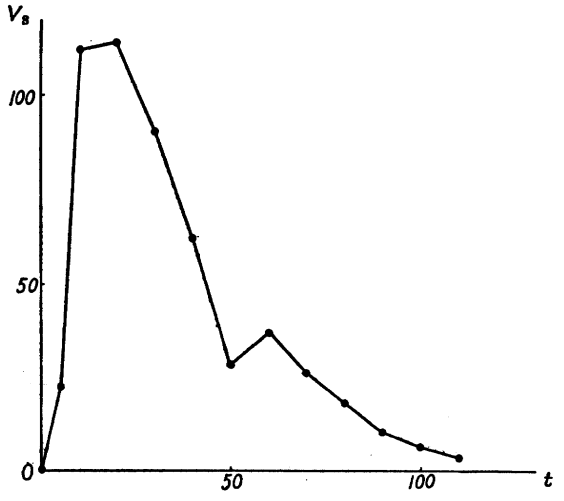


Fig. 5. V_s expresses the variance of s at the time t.

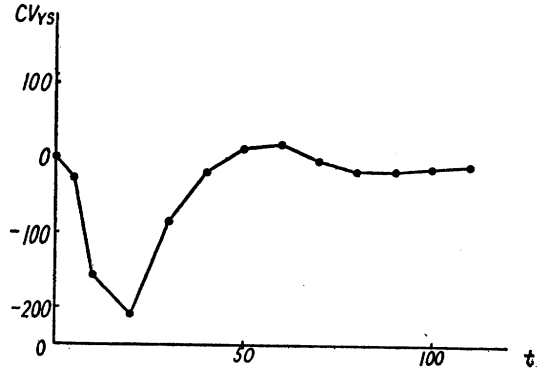


Fig. 6. CV_{rs} expresses the covariance of r, s at the time t.

Let us denote the variance of r by V_r, the variance of s by V_s and the covariance of r, s by CV_{rs} at the time t. Then, the graphs of V_r, V_s CV_{rs} are shown in Fig. 4, 5, 6.

This result (r, s) was a numerical illustration of the differential equation (6) by Monte Carlo Method, but we must compare its solution with epidemiological data whether this model fits or not. But theoretical defect of this model is that the conditional probability of recovering is only proportional to the number of infectives at that time and does not depend on the individuals infected time. And, therefore, we shall introduce the recovering time distribution $G(\tau, t)$ in Section 4 so as to remove this defect.

4. Stochastion treatment (II)

We can understand that the probability of a new recovering does not depend on the number of infectives, but must depend only on the individual's infected time from epidemiological phenomenon. So, we must consider the recovering time distribution $g(\tau, t)$ from the infected time τ. The conditional probability $\beta_3 s\Delta t$ means that every infective has an equal chance of recovering independent of the individual's infected time.

Bartlett or Bailey, also, do the same restricted assumption as we do. In reality, the probability of recovering of one infective in the time interval $(t, t+\Delta t)$ depends only on the infeted time, so that the model (6) is so restricted. If we can improve this point, the epidemic model will be very useful so as to analyse the epidemic expansion.

Let $g(\tau, t)\Delta t$ be the conditional probability that one infective which was infected at the time τ recovers in the interval $(t, t+\Delta t)$. And, therefore, the propabtlity that one infective which was infected at the time τ will recover before the time t is given by:

$$G(\tau,\ t)=\int_{\tau}^{t} g(\tau, u)du \tag{13}$$

where $\tau \leqq t$.

As shown in Section 2, the conditional probability of one new infection (or one new removal from the group of susceptibles) taking place in the time interval $(t, t+\Delta t)$ is given by $\beta_1 rs\Delta t$ (or $\beta_2 rs\Delta t$) when there are r susceptibles and s infectives at the time t.
Let us assume that

$$\int_0^t E_t(\beta_1 r_\tau s_\tau)g(\tau, t)d\tau = K(t) \leqq K < \infty \tag{14}$$

where r_τ is the number of susceptibles and s_τ the number of infectives at the time τ. And E_t expresses the expectation of r_τ, s_τ at the time τ so as to gain r_t, s_t at the time t. Then, we can give (15) as the approximation of the probability $W(t)dt$ of one new recovering taking place in the time interval $(t, t+\Delta t)$.

That is, it is:

$$W(t)\Delta t=\left\{\int_0^t E_t(\beta_1 r_\tau s_\tau)g(\tau, t)d\tau\right\}\left\{\exp\left[-\int_0^t E_t(\beta_1 r_\tau s_\tau)\, g(\tau, t)d\tau\right]\right\}\Delta t \tag{15}$$

This idea is the essential point in this section

If we can replace the conditional probability (5) by the equation (15) and derive the corresponding differential equation to (6), (7), this problem is vey simle. But it is not easy. We shall publish the epidemic model basing on the equation (15) in future. Surely the model in section 2 contains the contradiction that the probability of recovering of a infective depends only on the number of infectives and does not depend on the infected time. We understand that this idea predominates the treatment of epidemiology up the present. But such idea must be improved according to the idea of (15).

5. Classical approach.

By the idea of Section 4, we shall revise a classical treatment from our stand point.

Model (A)

Let $G(\tau, t)$ be the probability that one infective infected at the time τ will recover before time t. Then, we can give the probability $G_1(\tau, t)=1-G(\tau, t)$ that one infective infected at the time τ will not recover before the time t. That is, $G(\tau, t)$ shows the probability of duration of disease to the time t from the infected moment τ.

Also, let $h(t)\Delta t$ denote the number of infectives infected in the infinitesimal time interval $(t, t+\Delta t)$, and let $F(t)$ be the number of infectives at the time t which the initial number of infectives equals to $F(0)=a$. Then, we get the equation;

$$F(t)=\int_0^t h(\tau)G_1(\tau, t)d\tau+aG_1(0, t)+\varepsilon_t\ . \tag{16}$$

The first expression of the right term of (16) is the number of infectives which were infected in the time interval $(0, t)$ and are, yet, infectious at the time t. The second expression is the number of infectives which are, yet, infections at the time t among the initial infectives a. The third expression ε_t is the independent random variable which $E(\varepsilon_t)=0$, $E(\varepsilon_t^2)=\sigma_t^2$ at the time t and $E(\varepsilon_t)=E(\varepsilon_t^2)=0$ at the time $t=0$.
And, therefore, we can calculate the number of infectives $F(t)$ at the time t if the function $h(t)$ is known.

For this pupose, if the number of infectives $h(t)\Delta t$ occurred in the time $(t, t+\Delta t)$ is the proportional to the product of the numbers of infectives and susceptibles at the time t, we shall be be able to give the non-linear integral equation:

$$h(t)=\gamma\left\{\int_0^t h(\tau)G_1(\tau, t)d\tau+aG_1(0, t)+\varepsilon_t\right\}\left(n-\int_0^t h(u)du\right) \tag{17}$$

where n is the number of susceptibles at the initial time $t=0$, and γ is the proportional coefficient.

If, in Section 2, constant infection rate β_1 corresponds to $\beta_1(t)$, we have the relation $\beta_1(t)=\gamma\Big/\left(n-\int_0^t h(u)du\right)$ between $\beta_1(t)$ and γ.

Now, Putting

$$R(t, \tau, h(\tau))=\gamma\left\{\left(n-\int_0^t h(u)du\right)h(\tau)G_1(\tau, t)-aG_1(0, t)\,h(\tau)-\varepsilon_t h(\tau)\right\}, \tag{18}$$

$$f(t)=\gamma naG_1(0, t)+\gamma\varepsilon_t n \tag{19}$$

we can give the non-linear integral equation of Volterra's type from (17):

$$h(t)=\int_0^t R(t, \tau, h(\tau))d\tau+f(t). \tag{20}$$

where n, a are finite from assumption. If we assume $h(t)\Delta t$, $F(t)$ in the equation (16) to be the expected numbers, that is, we make the deterministic model of (16), the equations (18), (19) are rewritten as next;

$$R(t\,\tau, h(\tau))=\gamma\left\{\left(n-\int_0^t h(u)du\right)h(\tau)G_1(\tau, t)-aG_1(0, t)h(\tau)\right\}, \tag{18'}$$

$$f(t)=\gamma naG_1(0, t). \tag{19'}$$

$f(t)$ satisfies the Lipshitz condition in the finite interval $0\leqq t\leqq\eta$. And then it is, easily, shown that $R(t, \tau, h(\tau))$ satisfies the Lipshitz condition in the domain $D(0\leqq t\leqq\eta,\ 0\leqq\tau\leqq t,\ 0\leqq h\leqq h_\omega)$ as $h(t)$ is bounded in the finite interval $0\leqq t\leqq\eta$.

That is,

$$\begin{aligned}
|R(t, \tau, h_1)-R(t, \tau, h_2)| &\leqq\gamma|h_1(\tau)-h_2(\tau)|\cdot|nG_1(\tau, t)-aG_1(0, t)| \\
&+\gamma|(h_1(\tau)-h_2(\tau))|-\int_0^t h_1(u)du+\gamma h_2(\tau)\int_0^t|h_1(u)-h_2(u)|du \\
&\leqq K_1|h_1(\tau)-h_2(\tau)|+K_2 t\sup_{0<u<t}|h_1(u)-h_2(u)| \\
&\leqq(K_1+K_2\gamma)\sup_{0<u\wedge t}|h_1(u)-h_2(u)| \\
&=(K_1+K_2\gamma)|h_1(u^*)-h_2(u^*)| \\
&=K|h_1(u^*)-h_2(u^*)|
\end{aligned} \tag{21}$$

in the domain D, and so $R(t, \tau, h(\tau))$ satisfies the Lipshitz condition on $h(\tau)$. So, we can derive the numerical solution by the successive approximation. That is, when we make

$$\left.\begin{aligned} &h_0(t)=f(t)\\ &h_1(t)=h_0(t)+\int_0^t R(t, \tau, h_0(\tau))d\tau\\ &\cdots\cdots\cdots\cdots\cdots\cdots\\ &\cdots\cdots\cdots\cdots\cdots\cdots\\ &h_n(t)=h_0(t)+\int_0^t R(t, \tau, h_{n-1}(\tau))d\tau\ , \end{aligned}\right\} \tag{22}$$

the relation

$$\lim_{n\to\infty} h_n(t)=h(t) \tag{23}$$

exists uniformly on t, and $h(t)$ is the unique solution of the deterministic non-linear integral equation of (20).

Using this solution $h(t)$, we can calculate the number of infective $F(t)$ at the time t from (16).

Bodel (B).

Let $G(\tau, t)$ be the probability that one infective infected at the time τ will recover before the time t as model (A). And, therefore, $G_1(\tau, t)=1-G(\tau, t)$ expresses the probability that one infective infected at the time t. Furthermore, let $\delta(\tau, t)\Delta t$ denote the number of infectives which was infected in the time interval $(t, t+\Delta t)$ from one infective infected at the time τ. The definition of $h(t)\Delta t$ are the same as stated in model (A).

Assume that, at the initial time $t=0$, a infectives entered into the homogeneous social group which had no infective.

When ε_t, ε_t' express the random variables which have $E(\varepsilon_t)=E(\varepsilon_t')=0$.

$$F(t)=\int_0^t h(t)G_1(\tau, t)d\tau+aG(0, t)+\varepsilon_t\ , \tag{24}$$

$$h(t)=\int_0^t h(\tau)G_1(\tau, t)\delta(\tau, t)d\tau+aG_1(0, t)\delta(0, t)+\varepsilon_t' \tag{25}$$

the last expression being completely determined if the structure of $\delta(\tau, t)$ is known.

For example, suppose that

$$G(\tau, t)=\frac{\lambda^s}{\Gamma(s)}\int_\tau^t (u-\tau)^{s-1}e^{-\lambda(u-t)}du \tag{26}$$

where λ, s are parameters.

It is assumed here, for simplicity, that $s=2$, $\delta(\tau, t)=c$(constant). $\delta(\tau, t)=c$ means that the fixed number c of susceptibles are infected independently of the time t from one infective.

If we assume $F(t)$ to be the expected number of infectives at the time t, $h(t)\Delta t$ to be the expected number of occurrences of infectives in the time interval $(t, t+\Delta t)$, we get $\varepsilon_t, \varepsilon_t'=0$ in (28), (29).

This is admissible as a first approximation.

Then, as

$$G_1(\tau, t)=\lambda(t-\tau)e^{-\lambda(t-\tau)}+e^{-\lambda(t-\tau)} \tag{27}$$

the linear integral equation (25) can be rewritten in the form

$$h(t)e^{\lambda t}=(\lambda t+c)\int_0^t h(\tau)e^{\lambda\tau}d\tau-c\lambda\int_0^t h(\tau)\tau e^{\lambda\tau}d\tau+ac(\lambda+1)\ . \tag{28}$$

When we differentiate the above equation (28) twice with respect to time t, we can derive the next homogeneous differential equation of Euler's type:

$$\frac{d^2h(t)}{dt^2}+(2\lambda-c)\frac{dh(t)}{dt}+(\lambda^2-2c\lambda)h(t)=0\ . \tag{29}$$

And the characteristic equation of (29) is

$$f(r)=r^2+(2\lambda-c)r+(\lambda^2-2c\lambda)=0\ . \tag{30}$$

As $\lambda, c>0$, the characteristic equation (30) has mutually different real roots. When we express their roots by $-A$, $-B$, we can give the general solution of (29)

$$h(t)=C_1e^{-At}+C_2e^{-Bt} \tag{31}$$

where C_1, C_2 are constants.

Therefore, we obtain

$$\begin{aligned}F(t)=&\int_0^t(C_1e^{-A\tau}+C_2e^{-B\tau})\{\lambda(t-\tau)e^{-\lambda(t-\tau)}+e^{-\lambda(t-\tau)}\}d\tau\\&+a(\lambda te^{-\lambda t}+e^{-\lambda t})\ .\end{aligned} \tag{32}$$

And

$$\begin{aligned}F'(t)=&(\lambda t+1)e^{-\lambda t}\int_0^t(C_1e^{-(A-\lambda)\tau}+C_2e^{-(B-\lambda)\tau})d\tau\\&-\lambda e^{-\lambda t}\int_0^t(C_1e^{-(B-\lambda)\tau}+C_2e^{-(B-\lambda)\tau})\tau d\tau+a(\lambda t+1)e^{-\lambda t}\\=&\left\{\frac{C_1}{A-\lambda}-\frac{C_1\lambda}{(A-\lambda)^2}+\frac{C_2}{B-\lambda}-\frac{C_2\lambda}{(B-\lambda)^2}+a+\frac{aC_1\lambda t}{A-\lambda}+\frac{C_2\lambda t}{B-\lambda}+a\lambda t\right\}e^{-\lambda t}\\&+\left\{\frac{C_1\lambda}{(A-\lambda)^2}-\frac{C_1}{(A-\lambda)}\right\}e^{-At}+\left\{\frac{C_2\lambda}{(B-\lambda)^2}-\frac{C_2}{(B-\lambda)}\right\}e^{-Bt}\ .\end{aligned} \tag{33}$$

Furthermore, (33) can be rewritten in the next form

$$F(t)=(C_1'+C_2't)e^{-\lambda t}+C_3e^{-At}+C_4e^{-Bt} \tag{34}$$

Where $C_1'+C_3+C_4=a$ from a relation $F(0)=a$.

If we estimate the parameters $C_i'(i=1, 2)$, $C_j(j=1, 2, 3, 4)$, λ, A, B from data, we can calculate the number of infections patients at the time t, $h(t)$ and $G(\tau, t)$.

6. Summing-up of our Result.

The practical usefulness of the model stated in Section 2, 4, 5, depends, to a considerable extent, on non-statistical consideration (for example, the nature of infectious bacillus or virus, social environment and etc.). If we do not settle from a medical point of view, we cannot construct a real epidemic model. Whether we can apply our epidemic model to the epidemic expansion or not, we must consider sufficiently the social enviroment. However, we fancy, from the phenomenistic consideration of Section 4 that our method will be capable of estimating the epidemic structure with much more validity if such stochastic model is constructed.

As described in Section 2, the essential problem in our model is that we considered removal rate β_2 from a group of susceptibles to a group of immune cases. There may, surely, be some doubt whether all ostensibly attacked members are really infective. Judging from epidemiological phenomenon, β_2 is not zero generally. Bartlett and Bailey take the same assumption that every susceptible has an equal chance of infection. But as mentioned in the Section 1, 2, the probability of infection is not necessarily equally probable for every susceptible. We considered that the probability of receiving the infectious virus or bacillus was equally probable for every susceptible, but the probability of infection was not equally probable.

The essential point in Section 4 is that the conditional probability of recovering is not $\beta_3 s\Delta t$, but is given by (15). $\beta_3 s\Delta t$ means that every infective has an equal probability of recovering in the time interval $(t, t+\Delta t)$. It is thoretically erroneous that the probability of recovering does not depend on the infected time.

In the classical approach of Section 5, we introduced the time distribution of recovering and derived the non-linear integral equation (A)

of Volterra's type and linear integral equation (B). In the following paper, we shall verify the validity of our stochastic models by the data of influenza obtained in a community.

7. Acknowledgement

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REFERENCES

[1] Kermach, W. O. and McKendrick, A. G. "Contributions to the mathematical theory of epidemics. *Proc. Roy. Soc.*, A115 (1927), p. 700-721.

[2] Bartlett, M. S. "Some evolutionary stochastic processes. *J. R. S. S.* B, 11 (1946). p 211~229.

[3] Bartlett, M. S. "Deterministic and stochastic model for recurrent epidemics." *Proceeding of the Third Berkeley Symposium on Mathematical Statistics and Probability*. Vol. IV (1956), pp. 81-109.

[4] Bailey, N.T.J. "A simple stochastic epidemic." *Biometrika*, Vol. 37 (1950), pp. 193-202.

[5] Bailey, N. T. J. "The total size of a general stochastic epidemic. *Biometrica* 40 (1553), pp. 177-185.

[6] Bailey, N. T. J. "Some problems in the statistical analysis of epidemic data." *J.R.S.S.* B, 17 (1955) pp. 35-58.

[7] Bailey, N. T. J. "On estimating the latent and infectious periods of measles." I p. 15, II p. 322, *Biometrika* 34 (1956).

ERRATA

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Page	Line	Read	Instead of
228	18	empirical	emprical
233	29	fundamental	foundamental
239	17	$\|\varepsilon(z)\| \leqq 2\|z\|$	$\|\varepsilon(z)\| \leqq \|z\|$
"	23	$2\|z\| < \frac{1}{1+a}$	$\|z\| < \frac{1}{1+a}$
240	1	h	z
"	9	coefficient	coefficients
"	9	$2B(h,\ p,\ \varepsilon)$	$B(h,\ p,\ \varepsilon)$
241	17, 24		
"	12	function $\frac{\partial^2 V_{p+\tau\xi}(z)}{\partial\tau^2}\Big\|_{\tau=\theta}$	generating function for $B(h,\ p,\ \varepsilon)$
242	25	$\sum_{\nu=0}^{\infty} q_{i,\nu} z^\nu$	$\sum_{\nu=1}^{\infty} q_{i,\nu} z^\nu$
252	3	$\{p_i\}$	$\{p\}$
253	1, 29, 31	$\frac{1}{k}\bar{\nu}_i^{(e)}(h)$	$\bar{\nu}_i^{(e)}(h)$
"	29, 32	$\frac{1}{k}V(h)$	$V(h)$
269	29	$W(t)\Delta t$	$W(t)dt$
271	27	$+\gamma\|(h_1(\tau)-h_2(\tau)\| \cdot \int_0^t h \cdots$	$+\gamma\|(h_1(\tau)-h_2(\tau)-\int_0^t h \cdots$
"	29	$\leqq (K_1+K_2\gamma) \sup_{0<u<t} \|+$	$\leqq (K_1+K_2\gamma) \sup_{0<u\wedge t} \|+$