PROBLEMS IN CONTAGION

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1. Introduction

The purpose of this paper is to present some actual problems involving contagion together with a review of recent work on the subject. Attention is focused on two avenues of investigation which have been followed in England and the United States for the past four or five years. This is not meant to be a comprehensive review, since the results given are selected primarily from applications of stochastic process techniques to accident and epidemic studies.

In studying contagion, we first consider the idea of the "risk" of occurrence of one of a class of events. We assume that if one situation has more risk than a second, then the former would be expected to have more occurrences of the type of events in question than the latter. We do not try to define risk in general terms; however, by observing the number and times of occurrences of events, we can define various specific risk measures. The word "proneness" is usually used in connection with an individual and may be interpreted as the risk associated with an individual in a particular situation. Sometimes one speaks of an underlying proneness, independent of situation, which is supposed to exist in each person. This notion will not concern us at this time, and we constrain our viewpoint to the one which involves both the individual and the situation. When risk is measured for an individual we have a measure of proneness.

Suppose a process is such that proneness depends upon the number of events previously incurred by a person. Then we say there is *individual contagion* in the process. Perhaps the first event serves as a reminder and reduces risks of future events. This would be called *negative contagion*. If, on the other hand, an event were to change a person somehow, so that he became more susceptible to future events, *positive contagion* would exist. When a person's proneness depends on the events which have occurred to others in his environment we say there is *between individual contagion* with positive and negative connotations as just given.

In order to fix ideas somewhat, a few illustrations of situations involving contagion will be given. Of particular interest are cases in which contagion is observed in connection with epidemics and the spread of disease. Some of these situations have recently been brought to the attention of the author by members of the Commission on Respiratory Diseases of the Armed Forces Epidemiological Board.

2. Some problems involving contagion

2.1. *Living conditions.* Presently under investigation at two United States Air Force bases is the epidemiology of upper respiratory diseases. These are contagious diseases which present a serious problem to the armed forces because of the great amount of time lost due to them. It has been noticed in some preliminary work that

the courses of these diseases are apparently associated with housing conditions, that is, when military personnel, instead of being housed in the conventional "open bay" style barracks are housed in barracks which have been partitioned into small rooms, changes in incidence of upper respiratory diseases seem to occur. The problem is to compare the incidence of disease in the two barracks types under various conditions of crowding, heating methods, location, and climate. Further discussion of this is given in a later section.

2.2. *Experimental epidemics.* Over 20 years ago, a series of experimental mouse epidemics was begun in England and carried on for several years. Various conditions were established to provide for different types of immunity, rates of immigration, endemic and epidemic situations, and so forth. Papers by Greenwood, Hill, Topley, and Wilson [21] described the findings. More recently, Webster [33] in this country, and Fenner [17] in Australia, have done more work along this line. In addition, and less well known, is the fact that it has been possible on several occasions to establish experimental human epidemics and to observe in detail the spread of infection by inserting a carrier of a certain disease, for example, streptococcal disease, into an environment which had 50 or 60 individuals who were free from the disease. It is interesting to note that upon repetition there was a lack of uniformity of response, which indicates a need for a probabilistic treatment of the problem.

2.3. *Family studies.* Data on the occurrence of disease within families are quite rare. Observations from an outbreak of measles in England in the 1920's were the subject of considerable study by Greenwood [20]. Data from Providence, Rhode Island, published in 1938 [35] have been utilized in papers as recently as 1953 [5]. Of importance to knowledge in this field is a family study which has been underway in Cleveland continuously since 1947 [2]. From this Cleveland work, new information on within-family contagion has recently become available. Many questions concerning upper respiratory and gastro-intestinal diseases can now be attacked. Models describing the course of such diseases through families of various sizes can be constructed and checked.

2.4. *Accident proneness.* Another problem involving contagion is the question confronting workers in the field of accident research as to whether the occurrence of an accident changes a person's proneness to future accidents. This will be discussed in some detail in the next section.

3. Contagion and accidents

This section will be devoted to reporting on recent research on the theory of accidents. The reader is referred to writings by Lundberg [28], Feller [16], and Arbous and Kerrich [1] for critical comment on the early stages of research on this theory.

3.1. *The data.* A population of aircraft pilots is the object of study. When a flyer receives his pilot rating, records are begun which list each flight he makes throughout his flying carerr. Recorded are the following:

(i) Date of flight
(ii) Type of aircraft
(iii) Mission of flight
(iv) Number of landings
(v) Number of flying hours under various conditions of weather and time of day

From such records a detailed account of exposure to the risk of aircraft accidents is available for each pilot. In addition to the above, there exists an extensive account of each accident occurring to these pilots. Over a period of years, a pilot usually has a variety of experience. For example, suppose he has a flying record as given in figure 1.

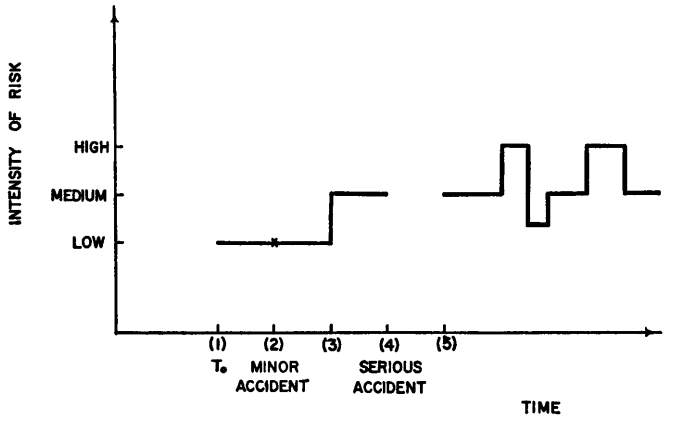

FIGURE 1

He begins flying aircraft of low risk (1) at time T_0; has a minor accident (2) but continues flying with no loss of time. He transfers to a higher risk job (3), has a serious accident (4), and does no flying for a while. He returns to flying (5) and works in all three risk situations from time to time. While all this is going on, the pilot himself changes, because of both increasing experience and increasing age.

3.2. *The problems considered.* Somehow we wish to study flyers like the above in order to describe the course of accidents among them and test hypotheses concerning them. We begin by considering an individual exposed only to a particular risk situation. Let t represent time, and suppose in the interval $0 \leqq t < T_1$ that m accidents occur to an individual at times $\tau_1, \tau_2, \cdots, \tau_m$. Denote the conditional probability that n further accidents occur to this person during interval (T_1, T_2), given this previous experience, by $P_n(T_1, T_2 | \tau_m)$ where τ_m is the vector $(\tau_1, \cdots, \tau_m)$.

We shall consider various special circumstances under which this probability has been studied. Suppose that previous accident experience is adequately summarized by the number of previous accidents. In this case we assume $P_n(T_1, T_2 | \tau_m) = P_{m,n}(T_1, T_2)$.

In accordance with the work of Bates and Neyman, we proceed as follows by accepting certain postulates.

POSTULATE P_1: The individual I cannot die or otherwise cease to be exposed to accidents. This postulate is obviously unrealistic, and there is a real problem to develop the theory in order to include the possibility of death. Comments on some early steps in this direction appear later in this paper.

POSTULATE P_2: Whatever the time interval (T_1, T_2), with $0 \leqq T_1 < T_2$, the number of accidents, say $X(T_1, T_2)$, that individual I will incur and survive in (T_1, T_2) is a random variable whose distribution depends on T_1 and T_2 and on the number of accidents incurred in the time interval $(0, T_1)$, but not on the precise times when those accidents took place.

POSTULATE P_3: If $T_2 \to T_1$, then all the probabilities $P_{m,n}(T_1, T_2)$ converge to limits $P_{m,n}(T_1, T_1)$.

$$P_{m,0}(T_1, T_1) = 1 \tag{1}$$

$$P_{m,n}(T_1, T_1) = 0 \quad , \qquad n \geqq 1. \tag{2}$$

POSTULATE P_4: At least at $T_2 = T_1$, the probabilities $P_{m,n}(T_1, T_2)$ are differentiable with respect to T_2, and specifically,

$$\left.\frac{\partial P_{m,n}(T_1, T_2)}{\partial T_2}\right|_{T_2=T_1} = \begin{cases} -\phi(m, T_1) & \text{if } n = 0 \\ \phi(m, T_1) & \text{if } n = 1 \\ 0 & \text{if } n \geqq 2 \ . \end{cases} \tag{3}$$

This postulate implies that, as $T_2 \to T_1$, the probability of more than one accident in (T_1, T_2) decreases faster than the difference $T_2 - T_1$.

3.3. *The risk function.* The function $\phi(m, t)$ is important in that it defines the process. We call it the intensity of risk or the risk function and we identify it with the *accident proneness* of individual I. If $\phi(m, t)$ varies from person to person, we say there is variable proneness. If $\phi(m, t)$ varies with m, as implied by its form, then there exists a form of *individual contagion*. Finally, if $\phi(m, t)$ varies with time, t, then we speak of a *time effect*, thus introducing the effects of age and experience.

3.4. *Pólya Model* (P). Let $\phi(m, t) = \lambda(1 + \mu m)/(1 + \nu t)$. We see that μ and ν control individual contagion and time effect; for example, if $\mu = 0$, we say there is no contagion; if $\mu \geqq 0$, positive contagion; and so forth. Work of Bates and Neyman [9] attacks a problem associated with observations of successive time intervals under this model. Consider $S + 1$ consecutive periods of time, the ith period being (t_{i-1}, t_i) with $t_{i-1} \geqq 0$, $t_0 = 0$, $t_{S+1} = \infty$. Let X_i be a random variable defined as the number of accidents incurred after the moment t_{i-1} and up to and including t_i. (For simplification we here assume all accidents are survived.)

The problem is to deduce the joint probability generating function of the variables $X_1, \cdots, X_{S+1}$. Let $G_{X_1,\cdots,X_{S+1}}(U_1, \cdots, U_{S+1}|P)$ denote this generating function where p stands for the Pólya model as given by the above assumptions.

Consider first the generating function, $g_m(t_{i-1}, t_i, U)$ corresponding to interval (t_{i-1}, t_i),

$$g_m(t_{i-1}, t_i, U) = \sum_{n=0}^{\infty} U^n P_{m,n}(t_{i-1}, t_i) \ . \tag{4}$$

By conventional methods (for example, see [6]), the solution is obtained;

$$g_m(t_{i-1}, t_i, U) = \left\{\frac{A(t_{i-1})}{A(t_{i-1}) + [A(t_i) - A(t_{i-1})](1 - U)}\right\}^{\gamma+m} \tag{5}$$

where $A(t) = (1 + \nu t)^{\lambda\mu/\nu}$ and $\gamma = 1/\mu$. Let $A(t_i) = A_i$.

Using g_m as written above we can obtain by a simple recurrence relationship the desired expression for the generating function,

$$G_{X_1, \cdots, X_{S+1}}(U_1, \cdots, U_{S+1}|P) = \left[1 + \sum_{i=1}^{S+1} (A_i - A_{i-1})(1 - U_i)\right]^{-\gamma} . \tag{6}$$

This is the generating function of the $S + 1$ variate negative binomial distribution and is called the generalized Pólya distribution.

3.5. *The Greenwood-Yule-Newbold model* (N). We refer back to $\phi(m, t)$ as given in the last section, and put $\mu = \nu = 0$. Then we assume that individual proneness, λ, is a specific value of a random variable, Λ, which has a probability density function $p_\Lambda(\lambda) = [\beta^\alpha/\Gamma(\alpha)]\lambda^{\alpha-1}e^{-\beta\lambda}$ for $\lambda \geqq 0$. This, of course, is the familiar assumption made by Greenwood and Yule [19]. We denote this model by N. The multivariate probability generating function corresponding to this model may be written as

$$G_{X_1, \cdots, X_{S+1}}(U_1, \cdots, U_{S+1}|N) = \int_0^\infty G_{X_1, \cdots, X_{S+1}}(U_1, \cdots, U_{S+1}|\mu = \nu = 0, P)p_\Lambda(\lambda)d\lambda \,. \tag{7}$$

This leads by an easy limit procedure and an integration to

$$G_{X_1, \cdots, X_{S+1}}(U_1, \cdots, U_{S+1}|N) = \left[1 + \beta^{-1}\sum_{i=1}^{S+1}(t_i - t_{i-1})(1 - U_i)\right]^{-\alpha}. \tag{8}$$

When $S + 1 = 1$, we find

$$G_{X_1}(U_1|P) = [(1 + (A_1 - 1)(1 - U_1)]^{-\gamma} \tag{9}$$

$$G_{X_1}(U_1|N) = [1 + \beta^{-1}t_1(1 - U_1)]^{-\alpha} \,. \tag{10}$$

If $\alpha = \gamma$ and $(A_1 - 1) = \beta^{-1}t_1$, these two expressions are identical. Hence we reach the well-known result that one cannot distinguish the Pólya contagion model and the Greenwood-Yule-Newbold no contagion model on the basis of empirical observations made over a single time interval. This is, of course, the point made by Lundberg [28], Feller [16], and many others. When more than one time interval is observed, however, it is easily seen that the two generating functions are equal only if $\lambda\mu = \nu$.

3.6. *Tapering contagion.* The notion of a contagion effect which diminishes with time has been considered by Neyman in lectures and in unpublished reports prior to 1954. He called this a "tapering" contagion. Where the Pólya model imposes a lasting effect of an accident upon the proneness of the pilot, Neyman postulates that this effect may decrease, that is, taper off, with time. An interesting model of this has been generalized by Le Cam [26]. Let $x(t)$ be a function of time such that $x(0) = 0$, and $x(t)$ has a unit jump each time an accident occurs (which is when time equals one of the τ's mentioned earlier).

Now let

$$\phi(x(t), t) = a(t) + \rho\int_0^t k(t, s)dx(s) \,. \tag{11}$$

For example, taking $k(t, s) = \exp[-\alpha(t - s)]$, the effect of an accident decreases exponentially with time. If the integral $\int_0^t \phi[x(v), v]dv$ exists in the Riemann sense, we can write

$$P_0(t_1, t_2|x(t)) = \exp\left[\int_{t_1}^{t_2}\phi(x(v), v)dv\right] \,. \tag{12}$$

If $k(t, s)$ has suitable integrability properties, then from the above assumptions one can obtain the probability of exactly n accidents in an interval (t_1, t_2).

3.7. *Multiple exposure.* Tucker [31] has tackled the difficult problem, indicated in figure 1, of exposure to several kinds of risks. Consider for individual I a fixed decomposition of the positive time axis into intervals in each of which only one type of risk is met. Let each type of risk have a weight associated with it, and let the effective time of exposure be the weighted sum of the various exposure times. This is the idea used by Warren, et al., in [32]. Tucker goes further, however, by formulating the problem as follows. Let $f(t) = w_i$, $i = 1, 2, \cdots, r$, if t belongs to a time interval having a risk of type i. Then $T(t)$, defined as

$$T(t) = \int_0^t f(U)dU \tag{13}$$

is called the effective time of exposure to all risks up to calendar time t. Put $T(t_1) = T_1$ and $T(t_2) = T_2$, $t_1 < t_2$. Consider T_1 and T_2 as points on the scale of effective exposure time. In particular, let $l_j(T_1)$ be the effective time of exposure to a risk situation of type j prior to T_1,

$$l_j(T_1) = \int_{R_j} f(U)dU \ , \tag{14}$$

where R_j includes all intervals of j-type risk prior to time t_1. Obviously,

$$T_1 = \sum_{j=1}^{r} l_j(T_1) \ . \tag{15}$$

Let $\mathbf{l}(T_1)$ denote the vector $(l_1(T_1), \cdots, l_r(T_1))$. Also let $m_j(T_1)$ denote the number of accidents associated with risk j prior to T_1, with $\mathbf{m}(T_1) = (m_1(T_1), \cdots, m_r(T_1))$.

Define $P(n \mid i, T_1, T_2, \mathbf{l}, \mathbf{m})$ as the conditional probability that during the effective time of exposure (T_1, T_2), the individual I will incur exactly n accidents, given that during (T_1, T_2) I is constantly exposed to a risk situation of type i and given that prior to T_1, his exposure and accident experience are characterized by vectors $\mathbf{l}(T_1)$ and $\mathbf{m}(T_1)$ respectively.

The key postulate in this case is: at least at $t_2 = t_1$ the probabilities $P(n \mid i, T_1, T_2, \mathbf{l}, \mathbf{m})$ are differentiable with respect to t_2, and, specifically,

$$\left.\frac{\partial P(n \mid i, T_1, T_2, \mathbf{l}, \mathbf{m})}{\partial t_2}\right|_{t_2 = t_1} = \begin{cases} -\lambda\left[\dfrac{1 + \boldsymbol{\mu}\mathbf{m}(T_1)}{1 + \boldsymbol{\nu}\mathbf{l}(T_1)}\right] w_i \text{ for } n = 0 \\ \lambda\left[\dfrac{1 + \boldsymbol{\mu}\mathbf{m}(T_1)}{1 + \boldsymbol{\nu}\mathbf{l}(T_1)}\right] w_i \text{ for } n = 1 \\ 0 \text{ for } n \geqq 2 \ . \end{cases} \tag{16}$$

The risk function $\phi(m, t)$ is now replaced by $\phi(\mathbf{m}, \mathbf{l})$ with the vectors $\boldsymbol{\mu}$ and $\boldsymbol{\nu}$ playing the roles of μ and ν.

The probability generating function approach of Bates and Neyman can be repeated here with similar results.

3.8. *Fatal accidents.* One of the points made by Arbous [1], in his excellent dis-

cussion of accident research, is the need for considering fatal accidents. It is just the people involved in such accidents who may provide information crucial to questions of proneness and contagion. The sections above on the Pólya and Greenwood-Yule-Newbold models purposely deleted provisions for fatal accidents for the sake of simplicity. Now a brief discussion of this problem will be given.

Let Z be a random variable defined as the number of complete periods of time survived by the individual I. The random variables X_i used above are now defined as the number of accidents incurred after moment t_{i-1} and up to and including t_i, which the individual I will survive. We modify the postulates so as to permit the possibility of dying from an accident and we stipulate that to each accident there corresponds a fixed probability θ of surviving it. Then define $Q_{m,n}(T_1, T_2)$ as the conditional probability that during the time interval (T_1, T_2) individual I will incur exactly $n + 1$ accidents, the last one being fatal, given that he incurred m accidents prior to time T_1. Using the fact that

$$Q_{m,0}(T_1, T_2) = (1 - \theta)[1 - P_{m,0}(T_1, T_2)] \tag{17}$$

we can then proceed to find the joint probability generating function of the variables $Z, X_1, \cdots, X_{S+1}$. This has been done by Bates and Neyman [9] for both the Pólya and the Greenwood-Yule-Newbold models. Tucker [31] also utilized this device in his work on multiple exposure. Most other work, however, has skipped over this important part of accident analysis.

3.9. *Joint distribution of the times of occurrence of accidents.* Consideration of problems associated with the time intervals between accidents has been given by Maguire, Pearson, and Wynn [29]. At this time, however, we turn attention to some more recent work. Bates [10] in a paper, yet unpublished, has attacked the problem of determining the joint density function of the times, $\tau_1, \tau_2, \cdots, \tau_n$, of occurrence of accidents. Furthermore, she has developed a test of the hypothesis of no contagion in the set of admissible hypotheses which assume no time-effect and linear contagion. Among other cases, Bates obtains the joint probability density functions when the risk function $\phi(m, t)$ is of the forms

$$\phi(m, t) = \lambda(1 + \mu m) = \lambda + \psi m \tag{18}$$

and

$$\phi(m, t) = \lambda . \tag{19}$$

In the former case the density is

$$p_{\tau_1,\cdots,\tau_n}(t_1, \cdots, t_n|\psi) = n! \left(\frac{\psi}{e^\psi - 1}\right)^n \exp\left(\psi \sum_{k=1}^{n} t_k\right), \tag{20}$$

while in the latter it is

$$p_{\tau_1,\cdots,\tau_n}(t_1, \cdots, t_n|\psi = 0) = n! . \tag{21}$$

These are both independent of λ; thus no restrictive assumptions concerning the distribution $p_\Lambda(\lambda)$ of Λ among a population of pilots need be made. The joint density of the times of accidents is seen to be a uniform one under the hypotheses that $\psi = 0$.

3.10. *Tests of hypotheses.* The availability of the distributions in the last section

makes possible the development of a test for the hypothesis of no contagion and no time-effect, $\phi(m, t) = \lambda$, against linear contagion and no time-effect, $\phi(m, t) = \lambda + m\psi$. Bates constructs such a test by utilizing the fact that, when $\phi = \lambda + m\psi$, then $\sum_{k=1}^{n} t_k$ is a sufficient statistic for ψ.

Suppose there is a total of S accidents and the values t_j of the τ's are observed. Under the hypothesis of no contagion, the statistic

$$\bar{t} = \frac{1}{S} \sum_{j=1}^{S} t_j \tag{22}$$

is distrbuted very nearly normally with a mean 1/2 and standard deviation $1/\sqrt{12S}$. Bates shows that the test $\bar{t} > t_0 (\bar{t} < t_0)$ provides a uniformly most powerful test of hypothesis $\psi = 0$ against the class of alternates $\psi > 0$ ($\psi > 0$), where t_0 is chosen for an appropriate level of significance.

Le Cam investigates tests of contagion under his model of tapering contagion. He derives a locally best test for the presence of contagion, testing for $\rho = 0$ in his expression

$$\phi(x(t), t) = a(t) + \rho_0 \int_0^t k(t, s)\, dx\,(s)\,. \tag{23}$$

Bates' test has been extended by Tucker to the case of multiple exposure.

4. Stochastic theory of epidemics

4.1. *Comments on the deterministic theory.* Attention should be called to Serfling's excellent review [30] of the development of deterministic epidemic theory. In it, the work of the pioneers in the mathematical study of the spread of disease is presented. Some of the most extensive and elegant work was that of Kermack and McKendrick in their general theory and particularly in their analyses [25] of the experimental mouse epidemics previously mentioned. It is not the purpose of this paper to discuss the work done in this deterministic field. Instead, attention is directed to recent literature devoted to the development of probabilistic methods for describing epidemics.

4.2. *A few problems concerning the spread of disease.* We now continue the discussion begun in section 2.1 above. At one of the recruit training bases of the United States Air Force, a practically continuous stream of newly enlisted men flows in and out of the base. As they arrive, the men are grouped into "flights" of 50 or 60 men. Each man lives with his flight for the entire period of training (about 12 weeks). A flight occupies one floor of a barracks, and its members sleep, eat, and train together. Detailed information is available on the health of these men, and consequently the disease experience of a great many flights is known. The problem is to utilize this information by building stochastic models to describe the situation, by estimating parameters, and by testing certain hypotheses connected with types of barracks and other matters. Present are many of the classical difficulties associated with varying incubation periods, temporary immunity, variable proneness, seasonal changes, etc. In addition, and not having obvious counterparts in deterministic theory, are probabilistic features generated by the existence of the small,

semi-independent groups of 50 or 60 men. Similar problems occur in family living, of course, but data on as large a scale are not available.

In another paper in this symposium [14] some specific new methods of attacking the above problems are presented by A. T. Bharucha-Reid. Consequently, the remainder of this paper will be concerned only with some of the efforts made in the recent past to study contagion and theoretical epidemics.

4.3. *Continuous time approach.* Many articles appearing in the past six years refer to the notes of lectures on stochastic processes given in 1946 by Bartlett [6]. These, together with his 1949 Symposium paper [7], form the nucleus of some of the recent work in epidemic theory. As in the accident studies, the key to many models of epidemics is a risk function similar to $\phi(m, t)$. Differing from the accident approach, however, is the predominance of the concept of between individual contagion. The individual is no longer the 'population' studied. Now a group of individuals must be studied together.

One of the concomitants of epidemic contagion models is the nonlinearity of the associated differential equations. This occurs with many of those models in which the risk of a new case occurring in a small population depends on the number of infectious persons. Nonlinearity also occurs in many models featuring two or more populations between which interaction exists.

(a) *The logistic case.* As an example, we first consider one of the simplest of between individual contagious situations, the well-known logistic case. We assume that at $t = 0$ there are N susceptible people and one infectious person in the population. No immigration or deaths occur. Once infected, a person remains infectious. If there are Y_t infectious people at time t, then the individual risk function is given by $\phi(Y_t, t) = Y_t\mu$. The risk to the population, however, is $(N + Y_t - 1)Y_t\mu$. Denote the expectation of Y_t as m_t. Feller has pointed out in [15] that in this logistic case the stochastic mean, m_t, does not agree with the corresponding deterministic solution, m_t^*, which is the logistic function,

$$m_t^* = \frac{N + 1}{N + e^{(N+1)\mu t}}. \tag{24}$$

However, an explicit expression for m_t itself has until recently eluded workers in this field. Bailey, in a clever attack on this problem [3], considers the evaluation of the probabilistic mean m_t. This is a rather formidable task, partly because the usual differential equation in m_t involves the second moment and the probability generating function is of second order. In his work Bailey turns to the Laplace transform in addition to the probability generating function. While failing to solve the problem completely, he succeeds in deriving expressions which simplify it greatly.

In a highly satisfying piece of work, Haskey [22] builds upon Bailey's results and conquers a mountain of algebra to obtain a fairly simple expression for m_t, the expected number of infectious persons,

$$m_t = N + 1 - \sum_{r=1}^{N/2} \frac{N}{(N - r)!(r - 1)!} \cdot \left\{(N - 2r + 1)^2 t + 2 - (N - 2r + 1) \sum_{p=r}^{N-r} \frac{1}{p}\right\} e^{-\alpha t} \tag{25}$$

for N even, while for N odd, m_t equals the above expression summed over $r = 1, 2, \cdots, (N + 1)/2$ less a term, $4N! \exp(-at)/[(N + 1)!]^2$, where $a = r(N-r+1)$. This constitutes a rather neat solution to a problem which has, by its algebraic unpleasantness, bothered a great many people.

Something which is obvious but which the author has not seen in the literature is the interpretation of a simple growth process as a contagious process. Suppose the individual risk $\phi(m, t)$ is given as

$$\phi(Y_t, t) = \frac{Y_t \mu}{N - Y_t + 1} \tag{26}$$

where again Y_t is the number of infectious persons. We see that, as in the logistic case, the risk increases as the number of infectious individuals increases. Considering the whole population, however, the risk of a new infection is $Y_t\mu$ as in a birth process. The assumption that the total population is of size $N + 1$ leads to conclusions different from those of the usual birth process, however. The expression for the mean is

$$m_t = e^{\mu t}[1 - (1 - e^{-\mu t})^{N+1}] . \tag{27}$$

(*b*) *Immunity.* Let X_t and Y_t be the number of susceptible and infectious individuals respectively. Suppose an infectious individual has a risk λ of becoming noninfectious and immune, while a susceptible has the risk $Y_t\mu$ of becoming infected. Bailey [4] studied this model; he did not get far in deriving the probability generating function but he did go on to consider the total size of the epidemic. Letting time tend to infinity, he obtained a recursion method for finding the probability that X_∞ equals $N - w$ and $Y_\infty = 0$, given that at time 0, $X_0 = N$ and $Y_0 = a$.

(*c*) *Immunity plus immigration of susceptibles.* Add to the assumptions in (*b*) above a provision that the risk of a new susceptible entering the population is a constant ν. Bartlett, in [7], briefly states this problem, obtains a second order partial differential equation for the probability generating function and then leaves it after a discussion of various limiting properties.

Whittle [34] considers the moment generating function for X and Y and studies the relation of this stochastic model with its deterministic counterpart. He utilizes operational methods similar to those of Bartlett [7] in obtaining time expansions to approximate various expectations.

(*d*) *Incubation period.* Bartlett [7] adds to the model above by introducing a new 'state' of being in an incubation period, with risk λ_1 of then becoming infectious. He does little but state these assumptions and mention that Kendall's [23] device of introducing a number of such sub-states can be used to vary the distribution of lengths of the incubation periods.

(*e*) *Infection from within and without.* Gaffey [18] makes one of the first attempts at deriving a test for between individual contagion. He studies a variation of the logistic case in which a susceptible has risk λ of becoming infected from outside his family and risk $Y_t\mu$ of infection from the infectious individuals within his family. Gaffey follows the method used by Bates in obtaining the joint distribution of times $\tau_1, \cdots, \tau_n$ of occurrence of infection and developing tests of the hypothesis $\mu = 0$ against alternative hypotheses that $\mu > 0$, $\mu < 0$, or $\mu \neq 0$. As in Bates' work, the test is based on the mean of the τ's.

4.4. *Discrete time, the chain binomial.* Suppose the incubation period is fairly constant, the period of infectiousness is quite short, and this period is followed by complete immunity. Greenwood [20] introduced a method which he called 'chain binomials' to apply to such situations and used it as a tool in studying the incidence of measles in families. Lidwell and Sommerville [27] improved this method somewhat. Let p be the chance a particular susceptible becomes infected by a particular infectious person during the latter's brief infectious period. For example, if there is one primary (index) case and three susceptibles, then the probability of two new cases occurring is the probability of one secondary case which in turn leads to one tertiary case plus the probability of two secondary cases,

$$P\{2 \text{ new cases}\} = (3pq^2)(2pq)q + (3p^2q)q^2 = 6p^2q^4 + 3p^2q^3 \qquad q = 1 - p . \tag{28}$$

When theoretical distributions so derived were fitted to observations, Greenwood found that the fits were not always satisfactory and suggested that the risk p may vary considerably from family to family. Bailey [5] recently studied this problem and assumed that p was distributed among the population of families as a beta distribution,

$$dF = \frac{1}{B(x, y)} p^{x-1}q^{y-1}\, dp . \tag{29}$$

This leads to the probability of, say, 2 new cases out of 3 susceptibles, given one primary case, as

$$\frac{1}{B(xy)} \int_0^1 (6p^2q^4 + 3p^2q^3)p^{x-1}q^{y-1}\, dp = 6\,\frac{B(x+2, y+4)}{B(x, y)} + 3\,\frac{B(x+2, y+3)}{B(x, y)} . \tag{30}$$

When Bailey fitted theoretical distributions of this type to observations, excellent fits were obtained in all cases tried.

4.5. *Promising approaches in the probabilistic study of epidemics.* It seems appropriate in closing this paper to mention some of the work which seems to the author to be most promising for future research in epidemic theory. This is naturally a subjective list and not nearly an exhaustive one. Briefly, then, these approaches are:

(a) Whittle's [34] approximations, which relate the older and better developed deterministic results to stochastic cases.

(b) Utilization of approaches similar to that of Bellman and Harris [11] in their study of branching processes. Bharucha-Reid [13] has made a start here. There is need for models having variable incubation periods and variable periods of infectiousness.

(c) Expansion of David Kendall's [24] artificial realization of a birth and death process to epidemic situations.

(d) Interpretation of birth (growth) processes as a particular type of contagious process, then using the simple expressions which result to study epidemics.

(e) Extension and exploitation of Gaffey's [18] test for contagion.

(f) Application of LeCam's [26] generalized model of tapering contagion to the spread of the disease.

(g) Use of sampling procedures via electronic computers. In particular, Bartlett [8] mentions the use of Monte Carlo methods in epidemic theory.

(h) The work of Landau, Rapoport, and others from the Committee on Mathematical Biophysics of the University of Chicago. See [12] for comments and references.

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