Modelling the Eyam Plague*

G. F. RAGGETT, FIMA
Department of Mathematics, Statistics and Operational Research,
Sheffield City Polytechnic

Two models, one deterministic and one stochastic, are presented which simulate an outbreak of plague within a closed population. The data for this model are taken from that of Eyam, Derbyshire, England, which experienced such an outbreak following the Great Plague of London. The numerical results of the deterministic model are found to agree remarkably well with data derived from the available Eyam data. Those from the stochastic model are less encouraging, but the shortfalls of the existing model are appreciated and modifications for future work are indicated.

1. Background

EYAM is a village lying approximately 12 miles South-west of Sheffield, England, and within the picturesque Peak District National Park. It was the scene of an horrific outbreak of plague in the late seventeenth century, which left the village so decimated that only 83 people survived out of an initial total of 350 villagers. Thus in relative terms, this may be viewed as more catastrophic than the Great Plague of London where, although thousands died, only about one-sixth of the population finally succumbed to the disease.

Many books have been written on the events of the Eyam plague. The original book by Wood[1] and the much more recent one of Daniel,[2] who currently resides in the village, both give good accounts. What follows here is a brief résumé of the salient details required for the modelling of the plague. The interested reader is referred to the two excellent texts above.

The source of the plague at Eyam is now widely accepted as being from that of the Great Plague of London which ravaged through that city from 1664 to 1666. Apparently a tailor in Eyam received from London some cloth which was infected with plague-carrying rat fleas, which can produce plague in humans by biting their victims. Wood supposed that the first victim contracted the infection as a result of a slight graze caused from skin contact with the infected cloth. However, the former explanation is now medically confirmed as being the root cause of the bubonic form of the plague. The first victim was George Viccars who was buried on September 7th, 1665. Thereafter, the plague started to infect other villagers to a limited extent, the pattern of burials over the first 9 months of the plague being as follows:

1665	September	6 deaths
	October	23 deaths
	November	7 deaths
	December	9 deaths
1666	January	5 deaths
	February	8 deaths
	March	6 deaths
	April	9 deaths
	May	4 deaths

and at this point the effects of the plague appeared to be diminishing. However, with the onset of summer the plague established itself with renewed vigour and the figures for the remaining 5 months of the plague were as follows:

1666	June	19 deaths
	July	56 deaths
	August	77 deaths
	September	24 deaths
	October	14 deaths

These figures are taken directly from Wood's book, which lists most of the deaths, to the day, as taken from the records of the Reverend William Mompesson, who was the young rector of the village at the time of the plague. Mompesson made the courageous decision to isolate his enclosed community from outside and this most certainly was the reason why the plague did not spread significantly outside the village*.

How mournfully he must have entered the name of the 200th plague victim in his death record:

"1666. Aug. 25. Bur: Katharin ye wife of Mr. William Mompefson." For more details of Mompesson's life see Beaumont.[3]

Although the original cause of the Eyam plague was certainly due to the bubonic strain†, it is possible that the outbreak did not continue completely in this form. This is borne out by Pollitzer,[4] who asserts that even though "free-living" infected rodent fleas may be temporarily important in the causation of bubonic plague in man, the continued total existence of this form of the disease depends in the long run upon the persistence of the infection in rodents. Since in this case the original rodent infection came from a source 150 miles away, then a further continued infection of local rodents would have been required. This seems questionable in view of the fact that the plague was largely contained within the boundaries of Eyam, bearing in mind that the rodent population could not be prevented from going outside. While many of the house rat population would not attempt to migrate, one is nevertheless brought to the belief that the resulting plague was initially bubonic but then perhaps partially pneumonic, *i.e.*, infected directly from person to person. This belief is furthered by Wood who asserts that contraction of the infection was often transmitted from the cough of a victim suffering from plague pneumonia. This situation always seems to have been a very likely possibility even in previously supposed purely bubonic

* This paper formed the basis for a talk given to the Sheffield branch of the IMA in December 1981. It is also to be presented to the British Region of the Biometric Society in March 1983.

* Daniel cites only two minor exterior occurrences of plague over the Eyam outbreak period.

† Many of the victims were infected with bubues, an inflammatory swelling of a lymphatic gland, especially in the groin or armpit, and a characteristic feature of bubonic plague.

outbreaks of the disease. There is evidence of this from the Great Plague of London, and of other plagues, where children used to sing the now famous nursery rhyme

"Ring-a ring o' roses
A pocket full of posies
A-tishoo, A-tishoo
We all fall down."

Finally it should be realised that the information is somewhat suspect. Although the above data are given for deaths, they are strictly burial dates. Information in Wood and Daniel leads one to believe that burials were almost immediate for health reasons, but the vastness of the outbreak in the latter months makes one wonder if each death corresponds exactly to each interment. Every family, up to July or perhaps the latter end of June, 1666, had been compelled to bury its own dead. When either the last one of the household died, or the remaining occupants were too ill, someone was required to bury the corpse. Marshall Howe was the self appointed grave digger, who paid himself for his services from the belongings of the dead. He buried his wife on August 27th and his son on August 30th, 1666.

Further, some of the exact dates for March and October, 1666 are unknown and some discrepancies are apparent between Wood's figures given above and the official Register of Deaths on permanent display at the Eyam Parish Church.

2. The deterministic model

The basic deterministic model for the spread of an infectious disease, within a fixed closed population of size N, may be written as the following coupled set of ordinary differential equations

$$\dot{S} = -aSI, \tag{1}$$

$$\dot{I} = aSI - bI, \tag{2}$$

$$\dot{R} = bI, \tag{3}$$

where $S(t)$, $I(t)$ and $R(t)$ are time dependent representations of the susceptible, infective and removed populations, respectively. The dots denote differentiation with respect to time t, while the positive coefficients a and b represent the infection rate and removal rate, respectively.

Further, one has the relationship

$$S(t)+I(t)+R(t) = N, \tag{4}$$

along with appropriate initial conditions at $t = 0$ as

$$S(0) = S_0, \quad I(0) = I_0, \quad R(0) = 0. \tag{5}$$

For further details of both the analytical and numerical treatments of the $S-I$ phase plane solution of (1) and (2), the interested reader is referred to Raggett.[5]

With regard to this investigation we wish to determine if accurate values of both a and b exist which well simulate the plague outbreak at Eyam.

3. Modelling the Eyam data

The mathematical model posed above is apparently seen to be a reasonable model to apply to the Eyam plague for the following reasons.

(*i*) The assumed transmission of the plague may be simulated as a rapidly spreading infectious disease.

(*ii*) The complete isolation of the village keeps N fixed as required.

In fact this is only an approximation as a few of the wealthy villagers fled the village initially. There was also an exodus of children including Mompesson's own two. A few births and natural deaths were also chronicled during the plague outbreak. However, the figures for the above are not known and were certainly minimal compared with those villagers who remained.

For the purposes of this study N may be estimated at the initial outbreak time as the sum of the final survivors and those who died during the plague. Good estimates of these figures are known.

(*iii*) Daniel cites only three cases of infected individuals recovering. One of these was Marshall Howe who presumably built up some kind of inbuilt immunisation, being involved in repeated burials. For the purpose of this simplified model we shall assume that infected individuals are not allowed to recover. Thus the time dependent removed population is exactly measurable from the dead list.

Examination of the phase-plane solution in Raggett[5] predicts only a single infection peak I_p given by

$$I_p = N - c + c\ln(S/S_0), \tag{6}$$

where $c = b/a$ is known as the relative removal rate. This infection peak occurs at a corresponding susceptible population S_p, given by

$$S_p = c. \tag{7}$$

This is contrary to the Eyam data which evidently contain at least two local infection peaks. Thus the Eyam data are apparently made up of at least two infectious disease type models.

The relatively mild infection at the outbreak of the plague compared with the devastating later effects, along with the apparent randomness of the data during the winter months, has convinced me that it is just not worthwhile attempting to model the data prior to May or June, 1666, by any sort of model. The modelling of the data from this period onwards with this proposed model has the double advantage of containing just a single infection peak, as required, while still encompassing the major effect of the epidemic.

It was decided to use a relatively large time interval so as to smooth the data somewhat by overcoming some significant daily variations in the death rate. I have already applied this deterministic model with success using a starting date of May 18th, 1666, and a constant time interval of 31 days (see Raggett[6]). For reasons which will become apparent (see section 6) this date may not be used as a starting point for the stochastic model. For comparison of the two models we will therefore start both models from June 18th, 31 days after the May 18th date.

Further, because of the resulting loss of data over the May 18th to June 18th period it has been decided to halve the previous time interval to one of $15\frac{1}{2}$ days duration. This time interval will be used to assess both the proposed models.

Taking readings from the dead list, the data for the deceased and removed (cumulative deceased) populations over the major outbreak period are as given in Table I. Note that since each time interval contains a half-day, the actual data have been averaged.

Table I

Deceased and Removed Populations over Major Outbreak Period

Period (1666)	Deceased	Removed $R(t)$ (measured at end of period)
1. June 19–July 3/4	11·5	11·5
2. July 4/5–July 19	26·5	38
3. July 20–Aug 3/4	40·5	78·5
4. Aug 4/5–Aug 19	41·5	120
5. Aug 20–Sept 3/4	25	145
6. Sept 4/5–Sept 19	11	156
7. Sept 20–Oct 4/5	11·5	167·5
8. Oct 5/6–Oct 20	10·5	178

From Table I we note that the initial time zero is taken on June 18th, 1666 (when $R(0) = 0$ and $N = 261$). This latter figure is arrived at by subtracting the 89 prior deaths from the initial population of 350 (note that 12 of the 19 deaths recorded in June occurred prior to June 19th).

The infective plague population may be approximated from recent estimates of both the incubation period (the interval between exposure to the infection and the appearance of the first symptoms of the disease) and the length of illness prior to death.

According to the Concise Medical Dictionary,[7] the incubation period of human plague is a maximum of 6 days. Pollitzer[4] gives the length of illness as $5\frac{1}{2}$ days.

I take a uniform period of 11 days for the total infection period. Thus at the end of each time interval, one measures the current infective population by examining the death register for the following 11 days.

Using the information now available for the removed and infective populations, the corresponding susceptible populations are directly estimated at the end of each period using equation (4). This gives the following information for $S(t)$ and $I(t)$ at the terminal period dates (Table II). The unknown entries result from the incompleteness of the Eyam data for October 1666.

A good estimate of c may be obtained from the limiting equation (see Raggett[5])

$$N - S(\infty) + c \ln (S(\infty)/S(0)) = 0, \qquad (8)$$

where $S(\infty)$ is taken to be 83, the number of surviving villagers. Use of (8) with the above set of values gives $c \simeq 159$.

Furthermore, substitution of this c value into equations (6) and (7) gives $I_p \simeq 27$ with $S_p = c \simeq 159$. Thus, equation (4) gives the removed population at peak infection, $R_p \simeq 75$. Examination of the available data gives the removed populations on August 2nd and 3rd as 70 and 77, respectively. Thus we conclude that this was the peak infection time. Taking $I_p = 27$ at both these dates, gives a prediction of infection periods of 11 days in each case; this gives a further justification for taking this period as 11 days.

Using the above value of c, and noting that $c = b/a$, effectively reduces the problem to one of a single degree of freedom in the choice of either a or b.

Table II

Susceptible and Infective Populations at Terminal Period Dates

$S(0) = 254$, $I(0) = 7$, $R(0) = 0$, $N = 261$

Date (1666)	$S(t)$	$I(t)$
July 3/4	235	14·5
July 19	201	22
Aug 3/4	153·5	29
Aug 19	121	20
Sept 3/4	108	8
Sept 19	97	8
Oct 4/5	Unknown	Unknown
Oct 20	83	0

4. Numerical procedure and results of the deterministic model

To provide a comparison between the deterministic model and the derived Eyam data as given in Table II, I have numerically solved equations (1), (2) and (3) subject to $S(0) = 254$, $I(0) = 7$, $R(0) = 0$. The resulting nonlinear initial value problem was solved using Gill's modification of the fourth order Runge–Kutta method. The integrations were performed with a time step length of 10^{-2} from $t = 0$ to 4 and eight sets of solutions at steps of $t = 0{\cdot}5$ were noted; each set of solutions so obtained was compared with those at the (eight) end of period dates described in section 3. A uniform value of $c = 159$ was taken throughout, integrations being performed by varying b, then evaluating $a = b/159$.

The best criterion used to choose b was based on a root mean square error test between the fourteen available values in Table II and their corresponding values as predicted numerically*. The best two decimal place approximation to b obtained in this way was $b = 2{\cdot}78$, with a RMS error of approximately 3·36 on the cited $S(t)$ and $I(t)$ values.

One should remember here that the estimated removal rate, $b = 2{\cdot}78$, is based on a computed time step of one unit, corresponding to a real time interval of length 31 days. Since the original premise is based on an 11 day infection period, then one would expect a removal rate of unity, had one computed time unit corresponded to a real time interval of 11 days. Using linear arguments this would estimate a removal rate of about 2·82 based on a 31 day period. This indicates a very good agreement with the computed value obtained.

Table III

Numerical Estimates of Susceptible and Infective Populations as Predicted by the Deterministic Model

Date (1666)	$S_N(t)$	$I_N(t)$
July 3/4	232	15
July 19	195	24
Aug 3/4	155	27
Aug 19	124	23
Sept 3/4	105	15
Sept 19	94	9
Oct 4/5	89	5
Oct 20	86	3

Table III gives the numerical estimates $S_N(t)$ and $I_N(t)$ of $S(t)$ and $I(t)$, respectively, each rounded to the nearest integer (corresponding to those given in Table II), with the above mentioned b value.

* This was different from the data set chosen in reference 6 in which I used a set of fifteen values including five removed estimates. Since the population is assumed fixed, this effectively meant a comparison of ten independent values as opposed to the fourteen values cited above.

Apart from the poor infective prediction for September 3/4* the comparison between the results of the deterministic model (as presented in Table III) and those of the Eyam data with infection period predicted as 11 days (as presented in Table II) are reasonably good. Certainly, one is led to believe that the deterministic model does well simulate the outbreak of such a disease.

Fig. 1 illustrates the $S-I$ phase-plane solution of the deterministic model with $S(0) = 254$, $I(0) = 7$. This trajectory has been produced on a Techtronics 4014 device using an interactive computer graphics package which I previously developed.[8]

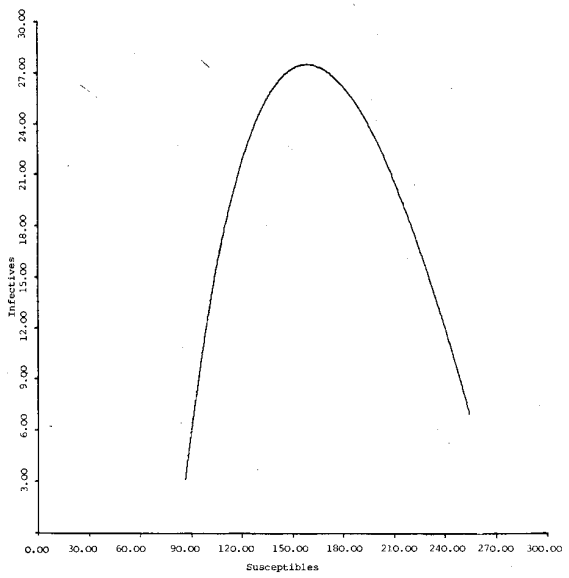

Fig. 1. $S-I$ phase plane solution of (1) and (2). The particular trajectory chosen is for $S(0) = 254$, $I(0) = 7$, simulating the major effect at Eyam

Finally, in an attempt to refine the deterministic model, I have modified the coefficients a and b to be functions of time which vary as the temperature varies. In this way one is attempting to model both the pneumonic (infection between individuals) and bubonic (continued possible infection from fleas whose density is increased with increasing temperature) aspects of the disease. Details of this approach are given in reference 6, but are not presented here as they do not provide further improvement of results. Perhaps this is hardly surprising in view of the excellent agreement already obtained.

5. The stochastic model

I again divide the total population into the three subpopulations, $S(t)$, $I(t)$ and $R(t)$, as previously defined.

Due to the effective dependence of $R(t)$ on $S(t)$ and $I(t)$ (with N assumed fixed) one may regard the problem as the determination of $S(t)$ and $I(t)$ only. This approach will also lend itself to direct comparison with the deterministic model using the same data set as given in Table II. Given particular susceptible and infective populations at a time t, then using first order probabilities, one has the following possible outcomes over $[t, t+\delta t]$.

Case (i)

Each susceptible has a chance of becoming infective, due to contact, over $[t, t+\delta t]$, proportional to the infective population. Let this be denoted by $aI(t)\,\delta t$. Thus the chance of the whole susceptible population producing a single infective over $[t, t+\delta t]$ is $aS(t)\,I(t)\,\delta t$.

Case (ii)

Each infective has a constant chance of becoming removed over $[t, t+\delta t]$. Let this be denoted by $b\delta t$. Thus the chance of the whole infective population producing a single removal over $[t, t+\delta t]$ is $bI(t)\,\delta t$.

Case (iii)

No change in the susceptible and infective populations over $[t, t+\delta t]$, with a total chance of

$$1 - aS(t)\,I(t)\,\delta t - bI(t)\,\delta t.$$

We now denote by $p_{S,I}(t)$ the probability that there exist $S(t)$ susceptibles and $I(t)$ infectives at time t, *i.e.*,

$$P_{S,I}(t) = P\{S(t) \text{ susceptibles; } I(t) \text{ infectives}\}. \qquad (9)$$

The spread of the resulting disease may then be simulated by obtaining an expression for $P_{S,I}(t+\delta t)$ in terms of appropriate probabilities evaluated at time t. These latter expressions are assumed to emanate from expressions of the type given in cases (*i*), (*ii*) and (*iii*) above. However, they will not be exactly as given above due to transitions of susceptible and infective populations.

For example, case (*i*) physically corresponds to a reduction of one susceptible giving a corresponding increase of one infective. Thus if there are to be S susceptibles and I infectives at time $t+\delta t$, then there were $S+1$ susceptibles and $I-1$ infectives at time t. This may be represented symbolically as

$$(S+1, I-1) \to (S, I).$$

Similarly, case (*ii*) corresponds to a single reduction of one infective, giving rise to

$$(S, I+1) \to (S, I).$$

Case (*iii*) preserves the subpopulations giving simply

$$(S, I) \to (S, I).$$

Using the above information one may immediately write $P_{S,I}(t+\delta t)$, in terms of probabilities at time t, as the probabilistic difference equation

$$\begin{aligned} P_{S,I}(t+\delta t) = {} & a(S+1)(I-1)\,\delta t\, p_{S+1,I-1}(t) \\ & + b(I+1)\,\delta t\, p_{S,I+1}(t) \\ & + [1-(aS+b)\,I\delta t]\, P_{S,I}(t). \end{aligned} \qquad (10)$$

Alternatively, subtraction of $p_{S,I}(t)$ from both sides of (10), division by δt throughout, and then allowing $\delta t \to 0$ gives rise to the corresponding differential difference equation

$$\begin{aligned} \frac{dp_{S,I}}{dt} = {} & a(S+1)(I-1)\,p_{S+1,I-1}(t) \\ & + b(I+1)\,p_{S,I+1}(t) \\ & - (aS+b)\,I p_{S,I}(t). \end{aligned} \qquad (11)$$

If one has a good estimate of the relative removal rate $c = b/a$, it is convenient to transform (11) by the change

* The infective Eyam data are somewhat "freak" here as no deaths were recorded in the period between September 10th and 16th, 1666.

of time variable $\tau = at$ giving

$$\frac{dp_{S,I}}{d\tau} = (S+1)(I-1)\,p_{S+1,I-1}(\tau) + c(I+1)\,p_{S,I+1}(\tau) - (S+c)\,I p_{S,I}(\tau). \quad (12)$$

This equation is given in Bailey[9] who derives it in a somewhat more complicated manner.

If at time zero there are known to be $S(0)$ susceptibles and $I(0)$ infectives then one has

$$P_{S(0),I(0)}(0) = 1 \quad (13)$$

with all the other probabilities assigned as zero. This gives a starting point for the solution of (12). One should also note the bounded relations (see Bailey[9]).

$$0 \leqslant S(t) + I(t) \leqslant S(0) + I(0);$$
$$0 \leqslant S(t) \leqslant S(0);$$
$$0 \leqslant I(t) \leqslant S(0) + I(0) \quad (14)$$

and we therefore assume that any $p_{S,I}(t)$ whose suffices fall outside the permitted range is zero.

6. Computational difficulties and results of the stochastic model

For computational convenience, the derivative term in (12) has been replaced by the simple forward difference expression

$$\frac{dp_{S,I}(\tau)}{d\tau} = \frac{P_{S,I}(\tau+\delta\tau) - p_{S,I}(\tau)}{\delta\tau} \quad (15)$$

which leads to a numerical solution of the difference equation

$$p_{S,I}(\tau+\delta\tau) = (S+1)(I-1)\,\delta\tau\, p_{S+1,I-1}(\tau) + c(I+1)\,\delta\tau\, p_{S,I+1}(\tau) + [1-(S+c)\,I\delta\tau]\, p_{S,I}(\tau), \quad (16)$$

where S and I are evaluated at the prior time τ. Equation (16) is a correspondence in the τ variable, to the original difference equation (10) in the t variable.

When solving equation (16) one must be aware that the solutions are probabilities and therefore must lie in the interval $[0, 1]$. In practice one overcomes this difficulty by choosing $\delta\tau$ relatively small so that the last term in (16) always remains positive. This satisfies the lower condition. Further, due to the small choice of $\delta\tau$, there results, for fixed $\tau > 0$, a relatively greater number of non-zero small probabilities which then satisfy the upper condition.

In section 3, it was indicated that the stochastic model may not be applied with a May 18th starting date as previously used for the deterministic model in reference 6. The reason is as follows. The available data from this date onwards show that $S(t)$ decreases monotonically while $I(t)$ initially increases to a single infection peak and then decreases to zero. Thus from the starting point indicated, one is initially looking for a decrease in $S(t)$ with a corresponding increase in $I(t)$. Following the notation of section 5 one is then searching for a major contribution emanating from transitions of the form

$$(S+1, I-1) \rightarrow (S, I)$$

which are directly produced from the first term on the right hand side of equation (16). Unfortunately this term contains a factor of $(I-1)$ which becomes identically zero using the initial $I(t) = 1$ value at May 18th. If one attempts to compute from this date, the infective population remains probabilistically most likely to remain at unity; thus the model effectively breaks down for this case.

In view of this, it is sensible to start the solution of (16) at a later time, when $I(t)$ is sufficiently far in excess of unity. The June 18th date is thus also chosen as the starting date for this model simulation.

Comparisons of the solutions obtained are then made with the Eyam data for $S(t)$ and $I(t)$ given in Table II.

Computations have been performed on equation (16) starting from $\tau = 0$, where $P_{254,7}(0) = 1$, and with all other initial probabilities assumed zero. A step length of $\delta\tau = 10^{-4}$ was chosen and the resulting probabilities were obtained at later scaled times. In order to compare the results of this model with the Eyam data, one retains at each step the probabilistically most likely estimate of susceptible and infective numbers. These estimates are compared with the data in Table II to determine any possible correlation, with a view to choosing optimally the fixed scaled τ step length between successive values which best approximate the two sets of results in the root mean square error sense. Proceeding in this way gives a minimum RMS error of approximately 5·49 with a τ step length of 0·0104 (to four decimal place accuracy), corresponding to a real time step of $15\frac{1}{2}$ days.

Table IV gives the probabilistically most likely estimates, $S_p(t)$ and $I_p(t)$, of $S(t)$ and $I(t)$, respectively, obtained in the above manner.

Table IV

Probabilistic Estimates of Susceptible and Infective Populations

Date (1666)	$S_p(t)$	$I_p(t)$
July 3/4	234	11
July 19	194	20
Aug 3/4	155	20
Aug 19	128	15
Sept 3/4	112	9
Sept 19	105	4
Oct 4/5	100	1
Oct 20	93	1

If one measures t in 31 day months then $a = \tau/t \simeq 0{\cdot}0104 \times 2 = 0{\cdot}0208$; thus one estimates $b = ac \simeq 0{\cdot}0208 \times 159 \simeq 3{\cdot}31$. This compares with an estimate of $b = 2{\cdot}78$ using the deterministic model. The larger b value for this stochastic model is reflected by a relatively larger removal rate which tends to limit the infective population. However, the comparison between Tables II and IV indicates that the stochastic model at least simulates the nature of the outbreak.

Further details of the stochastic model are given in Raggett.[10]

7. Further possible work

While both models presented here are rather simplistic, the results obtained give some real grounds for hope that they could be suitably modified and extended to simulate other present day plague outbreaks.

A major existing shortfall of both models is the effective assumption of a zero incubation (or latent) period, *i.e.*, once an individual is transferred from the susceptible to the infected class, that individual is assumed to be immediately capable of infecting other susceptibles. This is clearly unrealistic but could be overcome by the inclusion of a time delay factor.

If one assumes purely bubonic infection one should also attempt to model the rodent infective population and its interrelationship with the human population. However, such a compartment type model is likely to be inadequate due to the difficulty of obtaining realistic rodent data.

One should also perhaps consider the spatial aspects of plague infection. Certainly, for Eyam, the records clearly indicate that deaths occurred fairly rapidly among individual family units. The assumptions for the models presented here are based on perfect mixing which is clearly not accurate. Once again, however, one would have the difficulty of data availability in order to apply such models.

For present day outbreaks one would also need to include other factors such as vaccination control and variable population sizes. I am currently considering such models.

Readers requiring further background knowledge of mathematical epidemiology should refer to the papers by Bailey[11] and Anderson.[12] Both authors recognise the importance of modelling epidemic processes.

I am most indebted to Mr. K. Rennolls of the Forest Research Station, Farnham, Surrey, for discussions related to the setting up of the stochastic model described here.

References

1. Wood, W., "History of Eyam," Bell and Daldy, London, first published in 1865.
2. Daniel, C., "The Story of the Eyam Plague," published by the author, 1977.
3. Beaumont, R. M., "The Reverend William Mompesson 1638–1708, Hero of Eyam," Woods, Southwell, Notts., 1980.
4. Pollitzer, R., "Plague," World Health Organisation Monograph Series 22, 1954.
5. Raggett, G. F., *Applied Mathematical Modelling*, 1980, **4**, 479–482.
6. Raggett, G. F., "A Mathematical Model of the Eyam Plague," Proceedings of 1st IASTED International Conference on Applied Modelling and Simulation, Lyon, France, Mesnard, G., *Editor*, 1981.
7. "Concise Medical Dictionary," Oxford Medical Publications, 1980.
8. Raggett, G. F., *Comp. Graphics*, 1979, **4**, 149–154.
9. Bailey, N. T. J., "The Elements of Stochastic Processes with Applications to the Natural Sciences," Wiley, 1964.
10. Raggett, G. F., *Bulletin Appl. Stats.*, 1982, **9**, No. 2, 212–225.
11. Bailey, N. T. J., "The Estimation of Parameters from Epidemic Models," *in* Bartlett, M. S., and Hiorns, R. W., *Editors*, "The Mathematical Theory of the Dynamics of Biological Populations I," Academic Press, London, 1973, pp. 253–267.
12. Anderson, R. M., "Infectious Disease Agents and Cyclic Fluctuations in Host Abundance," *in* Hiorns, R. W., and Cooke, D., *Editors*, "The Mathematical Theory of the Dynamics of Biological Populations II," Academic Press, London, 1981, pp. 47–80.

THE INSTITUTE OF MATHEMATICS AND ITS APPLICATIONS

IRISH BRANCH

Dr. S.K. Houston, FIMA

Mr. W. J. Cromie, AFIMA

Chairman: Dr. S. K. Houston, FIMA (School of Mathematics, Ulster Polytechnic)
Secretary: Mr. W. J. Cromie, AFIMA (School of Mathematics, Ulster Polytechnic)

Branch activities

Throughout the year about eight meetings of the Branch are held covering a wide range of mathematical topics. The majority of the meetings are held in or near Belfast but some meetings are also held in Coleraine, Londonderry and Dublin. One meeting is arranged as a joint venture with the Royal Statistical Society. Occasional visits are also arranged and the most recent one was to the Planetarium and Observatory in Armagh. The Annual General Meeting of the Branch is usually held in May.