A Stochastic Model for Measles Epidemics in a Multi-Region Setting
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A stochastic model for measles epidemics in a multi-region setting

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ABSTRACT. In this paper, a regionally based stochastic model for the diffusion of measles epidemics is developed. The model is tested with an application to ten contiguous sub-areas centred on Bristol.

IN this paper we explore some ways in which a stochastic version of the classic Hamer–Soper deterministic model for measles epidemics (Soper, 1929) may be extended to analyse patterns of measles outbreaks in a regional setting. The models developed are applied to a set of ten contiguous sub-areas centred on the city of Bristol. Following a description of the nature of the data and the regional setting, the Hamer–Soper model and a stochastic equivalent are outlined for a single areal unit and some of the properties of the stochastic model are illustrated using an artificial data set. The multi-region model is then described and fitted to notifications of measles cases in the ten sub-area system and the results of the analysis are outlined. A modification of the model to give a better fit to the incidence of measles in Bristol itself is also discussed.

THE DATA AND THE REGION

The measles data used in this paper have been abstracted from the Registrar General's *Weekly Return*. As described in Cliff, Haggett, Ord, Bassett and Davies (1975, section 6.2), this source gives the number of measles cases, as notified to the (then) Medical Officer of Health, in each local authority area for each week ending on a Friday. The local authority reporting units are known as General Register Office areas (G.R.O.s). Detailed clinical work suggests that notifications may underestimate the actual incidence of measles by about $1\frac{1}{2}$–2 times because of variations in diagnosis and reporting practice (Stocks, 1949). However, these records are the only source of data, and insufficient is known about regional variations in under-reporting to allow correction factors to be applied. The data employed cover a 222-week period beginning in week 40 of 1966 and running through to week 52 of 1970, and were collected for the set of G.R.O.s shown in Figure 1. The choice of G.R.O.s was dictated partly by the fact that one of the authors has some knowledge of the area, and partly because the ten G.R.O.s form a fairly self-contained city region. With the exception of Bath, the non-Bristol G.R.O.s are very much dormitory areas for Bristol, and inter-area travel patterns are dominated by flows to and from Bristol. Cliff *et al.* (1975, p. 84) have already noted that the majority of measles cases are children in the 1–5-year age group, and have suggested that, at the local level, distance decay factors operate strongly in guiding the spatial pattern of outbreaks; that is, the spatial pattern may be expected to reflect patterns of mixing and contact among the persons at risk. A compact study area with fairly clear travel lines therefore seemed desirable.

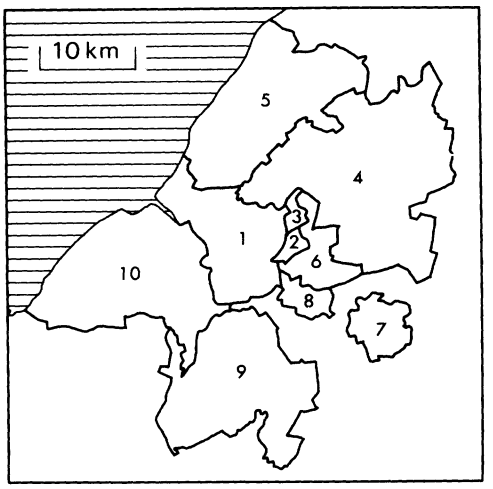

FIGURE 1. The General Register Office areas comprising the study region. 1 = Bristol C.B.; 2 = Kingswood U.D.; 3 = Mangotsfield U.D.; 4 = Sodbury R.D.; 5 = Thornbury R.D.; 6 = Warmley R.D.; 7 = Bath C.B.; 8 = Keynsham R.D.; 9 = Clutton R.D.; 10 = Long Ashton R.D.

The time series of notifications for each of the ten G.R.O.s are shown in Figure 2. These series reflect the temporal pattern of outbreaks for the South-West as a whole in this period; namely a major outbreak at the beginning and the end of the record period in most G.R.O.s in the South-West, with a minor outbreak in some areas roughly half-way through the time series (see Cliff *et al.*, 1975, p. 85). Bartlett (1957) and Cliff *et al.* (1975) have noted that (1) measles epidemics are temporally cyclical and form undamped waves and (2) stochastic 'fade out' occurs between epidemic peaks in areas with a population of less than *circa* 250 000, while measles is endemic in areas with larger populations. Bristol has a population approaching half a million, Bath about 82 000, and the remainder all less than 50 000. The time series shown in Figure 2 therefore confirm (1) and (2).

Measles is a highly infectious disease, with approximately 99 per cent of susceptibles contracting the disease after first contact with an infective. The time elapsing between receiving infectious material and the development of infectiousness (the *latent period*) is anywhere from 5–10 days, while the *infectious period* lasts for about another 7 days thereafter (see Bailey, 1957, chapter 6). The models to be described in the following sections incorporate this sort of information.

SOME DETERMINISTIC AND STOCHASTIC MODELS OF MEASLES EPIDEMICS

A single-region model

At any time t, we assume that the total population in the region can be divided into three classes: namely, the population at risk or *susceptible population* of size $S(t)$, the *infected population* of size $I(t)$, and the *removed population* of size $R(t)$.[1] The removed population is taken to be composed of people who have had the disease, but who can no longer pass on the disease to others because of recovery, isolation on the appearance of overt symptoms of the disease, or death. Four types of transition are allowed:

(1) A susceptible being infected by contact with an infective.

(2) An infective being removed. We assume that infection confers life long immunity, after recovery, to further attack, which is reasonable in the case of measles.

(3) A susceptible 'birth'. This can either come about through a child growing up into the critical age range (i.e. reaching about six months of age), or through a susceptible entering the population by migration into the region from outside.

(4) An infective entering the I population by migration into the region from outside.

For simplicity, we assume that there is no migration out of the region.

Suppose that transition i occurs at the rate $r_i (i = 1, 2, 3, 4)$; that is, in a small time interval, $(t, t+\delta t)$, the probability of transition i occurring is $r_i \delta t + o(\delta t)$, where $o(\delta t)$ means a term of smaller order (i.e. considerably smaller then) than δt. All events are assumed to be independent

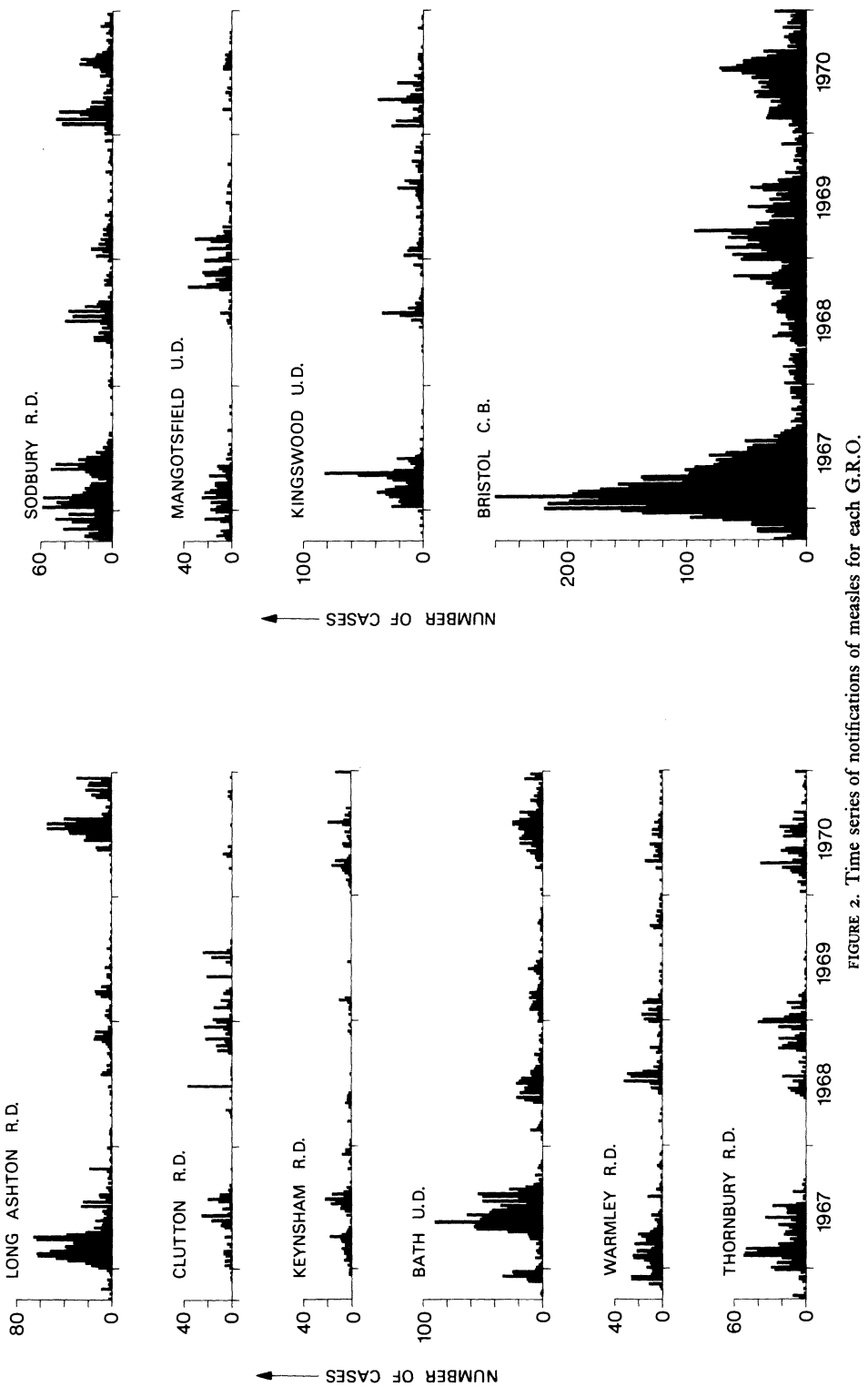

FIGURE 2. Time series of notifications of measles for each G.R.O.

TABLE I

Transition types and rates

Type of transition	*Rate*
$S \to S-1, I \to I+1 \; R \to R$	λIS
$S \to S \quad I \to I-1 \; R \to R+1$	μI
$S \to S+1 \; I \to I \quad R \to R$	ν
$S \to S \quad I \to I+1 \; R \to R$	ε

and to depend only on the present state of the population. The probability density of the time between *any* pair of successive transitions is

$$r\, e^{-rt}, \tag{1}$$

where

$$r = \sum_{i=1}^{4} r_i, \tag{2}$$

and the probability that the next transition is of type i is

$$r_i/r, \; i = 1, 2, 3, 4. \tag{3}$$

We assume, in the transition types 1–4, that the infection rate is proportional to the product, SI, that the removal rate is proportional to I, and that the birth and immigration rates are constant. We can thus prepare the table of transitions shown in Table I.

This model was first put forward in its deterministic form by Hamer (1906) and was studied extensively by Soper (1929). The main drawback of the deterministic model is that it leads to damping of successive epidemic waves which is not observed in practice. Soper believed erroneously that this damping could be eliminated by allowing for the fact that individuals exposed to measles undergo a latent period before becoming infectious, instead of assuming, as in the deterministic model described, that they transmit infectious material for the entire time between initial exposure and removal. However, even with this modification and, in addition, modifications to allow for seasonal variations in the infection rate and for spatial factors, damping still persists although it is reduced.

We therefore consider the more realistic stochastic formulation of the model. Even this relatively simple model is surprisingly intractable analytically, and except in special cases such as $\nu = \varepsilon = 0$, it is best studied using Monte Carlo techniques. It is, however, possible to see intuitively how the model operates. An infective is isolated after an average period of $1/\mu$ days, and while he is infectious, he causes new infections at the rate of λS per day. If we ignore the changes in S during this period, one infective infects an average number of $\lambda S/\mu$ ($= d$, say) susceptibles before he is removed. From the theory of the simple birth and death process, we would expect that, when $d \leqslant 1$, a small epidemic would die out. However, when $d > 1$, a small epidemic will spark off a major outbreak, although, of course, as the epidemic spreads, S will fall and $\lambda S/\mu$ can become less than 1. Thus the general pattern will be that the susceptible population will build up (transition type 3) to around the critical population size $S = \mu/\lambda$, when an epidemic will spread until the susceptible population falls sufficiently for the epidemic phase to pass [cf. Kendall's (1957) Pandemic Threshold Theorem]. The cycle will then repeat itself. In large communities where measles is endemic, the period between epidemic peaks is of approximate length $\mu/(\lambda\nu)$, the mean time for the birth of μ/λ susceptibles. In smaller communities, where there is fade out, the period is longer because once the critical susceptible population size is

TABLE II

Parameter values used in the three simulations of the stochastic version of the Hamer–Soper model for a hypothetical region

Simulation	μ	λ	ν	ε	*Incubation period*
1	0·14	0·00014	1·4	0·014	7 days
2	0·07	0·00007	1·4	0·014	14 days
3	0·035	0·000035	1·4	0·014	28 days

reached, there is a delay until the disease is introduced again into the region from outside (see Bartlett, 1956).

To illustrate these features, three simulations of the model were run for an imaginary region. In the simulations, the critical ratio, μ/λ, was fixed at 1000, and the birth rate, ν, was chosen to make $\mu/(\lambda\nu)$ 100 weeks. Three values of μ were tried corresponding to *incubation periods* (i.e. the length of time from receipt of infection to the appearance of overt symptoms) of 7, 14 and 28 days. Fourteen days is about the average length of the incubation period in the case of measles (Bailey, 1957, p. 118). Seven days would represent an absolute lower bound, while 28 days was tried to give some idea of the response that might be expected if this sort of model is used for other diseases with much longer incubation periods. The immigration rate was chosen to be small (one immigrant per 70 days) so that it would not influence the course of the epidemics but merely serve to reintroduce the infection after a period of fade out. The actual parameter values used are given in Table II. One day is taken as the time throughout. The initial population sizes were taken as $S = 1000$ and $I = 10$. At each stage, the four transition rates given in Table I were evaluated, and an exponential random variable with parameter r was generated, to find the time of the next event [equation (1)]. A uniform random variable was then used to decide which type of transition had occurred, since their relative probabilities are known from equation (3). A flow chart for the computer program appears as Figure 3.

The three simulations were each run for a period of 10 years, and the results of simulations 1, 2 and 3 are shown in Figures 4A–4C respectively. The vertical scales give the number of notifications per week, and the horizontal scales are divided into years. Figure 4B shows many of the features of the observed series for the Bristol area (Fig. 2), in particular the periodic epidemics. A prolonged period of fade out after an epidemic allows the susceptible population to build up, leading to a severe epidemic when the disease is reintroduced. When the incubation period is long (Fig. 4C), a periodicity can still be observed in the weekly numbers of notifications, but the peaks are greatly smoothed out, and there are no long periods of fade out. When the incubation period is short, the results are quite different (see Fig. 4A). The epidemics are still periodic, but each epidemic is compressed into approximately 6 months, with a prolonged period of fade out between recurrences. As observed earlier, fade out tends to increase the period between successive epidemics.

A criticism of the model used in the simulations is that it ignores the nature of the incubation period by assuming that an infective spreads the disease at a steady rate from the moment he is infected until he is removed. In fact, as we have noted, there is for measles a fairly well defined latent period of about 5–10 days before an infected person becomes 'contagious'. It was therefore decided to subdivide the infected population into a latent population of size L and a 'contagious' (transmitting) population of size C in the models described in the remainder of the paper. The possible transitions are given in Table III. The effect of this change upon the single-region model may be judged by comparing Figure 5 with Figure 4B. To obtain Figure 5 we re-ran

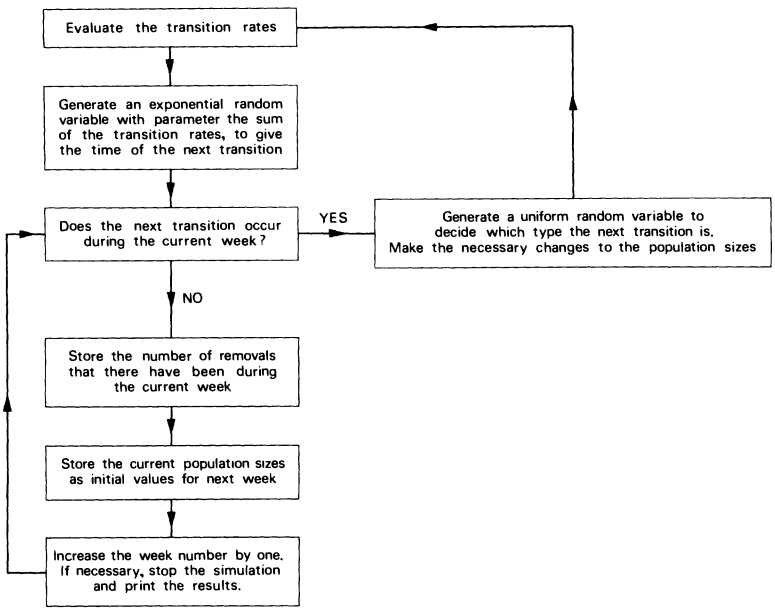

FIGURE 3. Flow chart of computer program used to simulate patterns of measles notifications

TABLE III

Revised table of transition types and rates

Type of transition	*Rate*
$S\to S-1, L\to L+1, C\to C, R\to R$	λCS
$S\to S, L\to L-1, C\to C+1, R\to R$	βL
$S\to S, L\to L, C\to C-1, R\to R+1$	μC
$S\to S+1, L\to L, C\to C, R\to R$	ν
$S\to S L\to L, C\to C+1, R\to R$	ε

simulation 2 with the parameter values given in Table II, except that we divided the 14-day incubation period into a 7-day latent period followed by a 7-day contagious period, as suggested for measles in Bailey (1957, p. 118). The infection rate was doubled to compensate for the halving of the length of the contagious period. The initial sizes of the S, L and C populations were fixed at 1000, 10 and 5 respectively. The effect of this change is to produce much clearer epidemic and inter-epidemic phases.

The multi-regional model

Suppose we have n regions, each with its own susceptible, latent, contagious and removed populations; that is, we have

$$\{(S_i, L_i, C_i, R_i), i = 1, 2, \ldots, n\}. \tag{4}$$

Evidently, the transition rates given in Table III must carry similar subscripting and be specified for each region. Of these parameters, β and μ depend only on the nature of the disease and so can be taken as regionally invariant. We chose values for β and μ so as to give the latent and con-

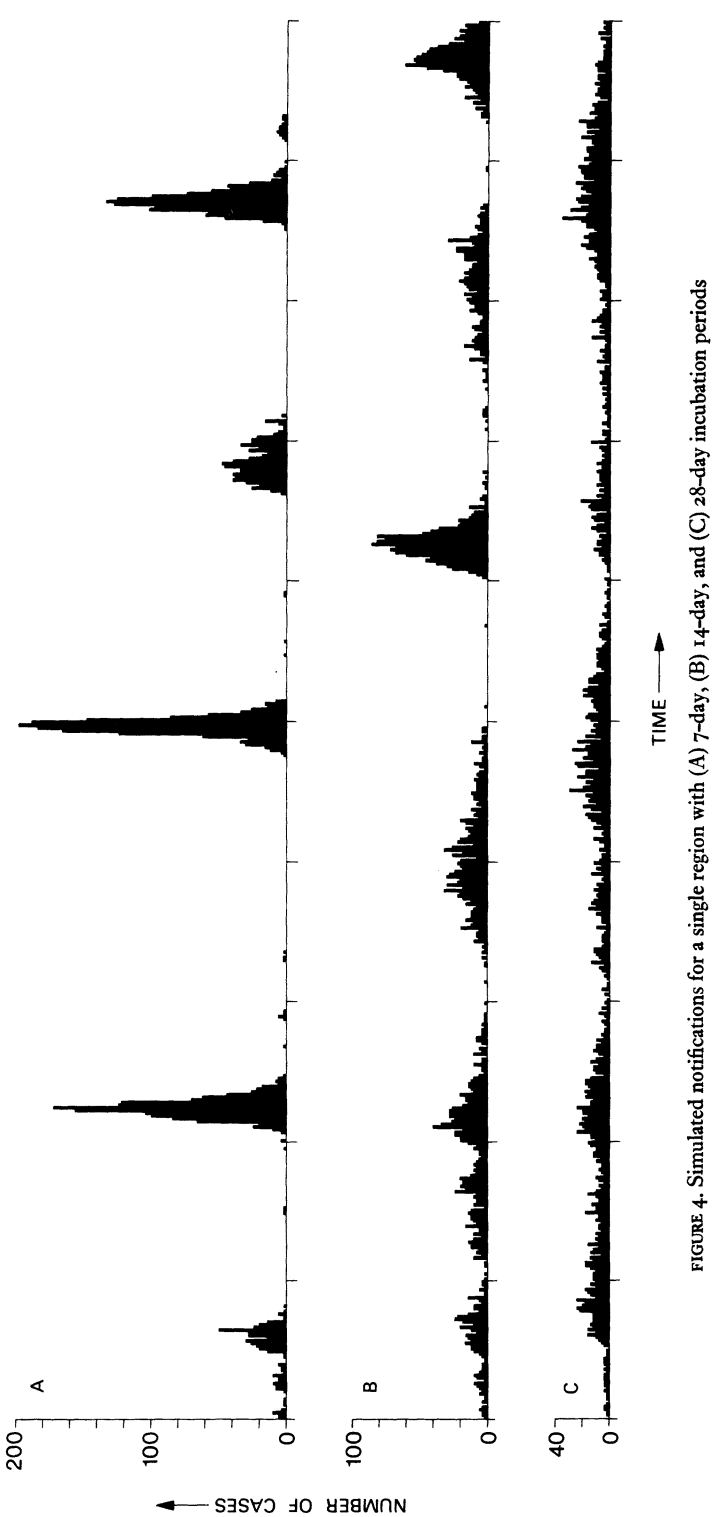

FIGURE 4. Simulated notifications for a single region with (A) 7-day, (B) 14-day, and (C) 28-day incubation periods

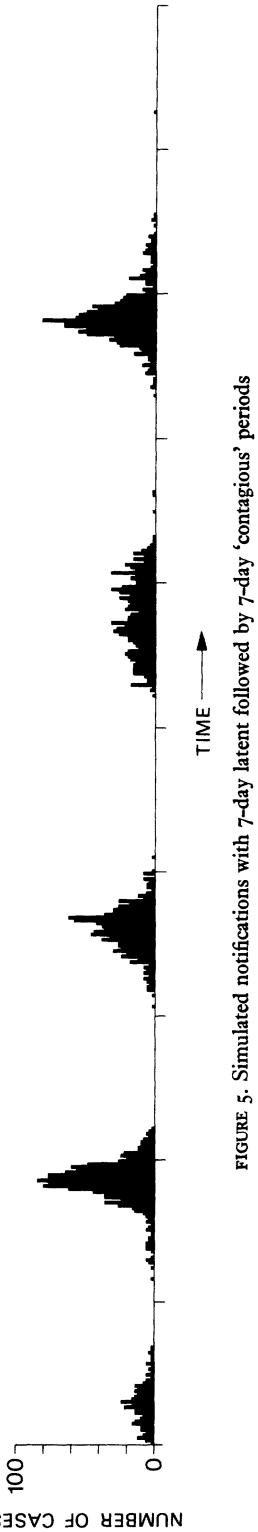

FIGURE 5. Simulated notifications with 7-day latent followed by 7-day 'contagious' periods

tagious periods average lengths of 5 and 7 days respectively, which, as noted above, are in the range of the true values for measles.

Inter-regional interaction is entered into the model in the following way. We define the probability rate of infection for region i, λ_i, as

$$\lambda_i = \sum_{j=1}^{n} \lambda_{ij} S_i C_j, \qquad i = 1, 2, \ldots, n. \tag{5}$$

This sum includes the term, $\lambda_{ii} S_i C_i$, as in the single-region model, and covers the ith *intra*-region interaction. The remaining terms handle interaction between region i and other regions j. Again, for simplicity, we assume that there is no migration between regions, although there is no difficulty in adding migration terms to the model. At the aggregate level, distance decay ideas imply that the probability of contact between a C person and an S person will fall off with increasing distance between them. A plausible way of modelling the $\{\lambda_{ij}\}$ is to use the gravity formulation,

$$\lambda_{ij} \propto 1/d_{ij}^2, \qquad (i \neq j), \tag{6}$$

where d_{ij} is the distance between the (demographic) centroids of regions i and j.

The diagonal terms $\{\lambda_{ii}\}$, the within-region probability interaction rates, represent the proportion of the total population of region i with which a susceptible in region i makes contact. λ_{ii} can be taken to be inversely proportional to the total population of region i by the rough argument that any given susceptible might be expected to have approximately the same size of acquaintance and kinship circle (that is, contact group at home, school, etc.), and to have the same level of risk of infection as a result, whatever the population size of the region. We would not expect, for example, a child in Bristol to have close contact with six times as many people as a child in Bath, simply because Bristol is six times the size of Bath. We accept that the argument is very tenuous. However, it is difficult to think of a better model without much more field work, which is not the purpose of this paper. Rather, we are trying to establish the broad outlines of a workable regional stochastic model of the process.

Parameter values chosen for the Bristol area G.R.O.s

The ten G.R.O.s listed in Table IV were considered. β and μ were fixed as described in the previous section. The parameters $\{\varepsilon_i\}$ were taken, as in the simlpe region model, to be very small (ten contagious immigrants a year into Bristol, and one a year into each of the other G.R.O.s) so that they would not affect the course of the epidemics but merely serve to reintroduce the disease after a period of fade out. The birth rates $\{\nu_i\}$ in the various G.R.O.s were chosen so as to make the average daily number of notifications in the simulations agree with the observed daily averages. In the model, the only way in which a susceptible can leave the system is by becoming infected and then being removed. Thus each susceptible will ultimately be infected,[2] and the average daily number of notifications in the model is given by the sum of the birth and immigration rates. These estimates will, however, be subject to error, because the size of the susceptible population would not necessarily have been the same at the start as at the end of the study period. In order to eliminate these errors, a much longer series of notifications would need to be examined.

The critical threshold ratio, μ/λ_{ii}, which the susceptible population must exceed for an epidemic to take off (see section on single-region model) then had to be estimated for each region. We consider Bristol first. In Bristol, approximately 5000 measles cases were notified during the course of the first epidemic. The value of ν_{BRISTOL}, chosen as described above, means that the model can generate in Bristol, over the same time span, about 2000 susceptible 'births'.

TABLE IV

Under-15 population values for the 10 study area G.R.O.s

Administrative areas and identity numbers	*Under-15 population (1971 census)*	*Total number of reported cases 1966–70*	*Daily average number of reported cases*
1 Bristol	93 740	8291	5·34
2 Kingswood	8 395	1125	0·72
3 Mangotsfield	5 500	743	0·48
4 Sodbury	18 580	2072	1·33
5 Thornbury	11 945	1422	0·92
6 Warmley	6 710	863	0·56
7 Bath	17 290	1925	1·24
8 Keynsham	4 720	452	0·29
9 Clutton	5 015	539	0·35
10 Long Ashton	10 755	1709	1·10

TABLE V

Inter-centroid distances in kilometres for Bristol area G.R.O.s

Area I.D. no./Area I.D. no.	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*
1	0	7·0	7·7	17·3	17·9	8·5	20·0	8·3	13·9	8·5
2		0	2·6	11·4	16·5	1·8	16·0	5·3	14·7	14·6
3			0	9·8	13·9	3·8	17·9	8·8	18·2	16·0
4				0	12·0	11·2	21·4	16·0	25·6	25·6
5					0	15·8	31·4	21·4	31·4	25·0
6						0	13·9	4·5	13·8	16·2
7							0	11·0	13·0	24·6
8								0	10·1	13·9
9									0	13·9
10										0

Source: Cliff and Ord (1969, p. 47). The G.R.O. identity numbers correspond with Table IV

These figures together imply a *net* reduction of about 3000 in the total susceptible population by the end of the first epidemic in the city, as compared with the size of the susceptible population in Bristol at the start of the epidemic. Bearing in mind that μ is already fixed (above), it was determined by trial and error that setting $\mu/\lambda_{ii} \sim 4500$ would consistently produce in the simulation results a net reduction of about 3000 in Bristol's susceptible population during the course of one major epidemic. We use λ_{ii} to denote the within-Bristol interaction rate. Since the $\{\lambda_{ii}\}$ are, by the arguments given in the previous section, to be taken as inversely proportional to the total populations of their associated regions, it is evident that, once λ_{ii} is fixed, the remaining λ_{ii}, $i \neq 1$, can be calculated from the relation,

$$\lambda_{ii} = \lambda_{11} \text{ (total population of 1/total population of } i), \tag{7}$$

where the populations are given in Table IV. The set of $\{\lambda_{ii}\}$ are thus fixed at the appropriate levels, relative to each other (and therefore to the total population size of each G.R.O.).

Apart from the constant of proportionality, the $\{\lambda_{ij}\}$, $i \neq j$, may be calculated from model (6) and the distances given in Table V. The proportionality constant is used to adjust the sizes of the $\{\lambda_{ij}\}$ thus calculated relative to the $\{\lambda_{ii}\}$. The scaling was again done by trial and error. The best approximation of the model results to the observed notification rates was obtained with

TABLE VI

Values of parameters (rates per day) used in the Bristol area measles simulation model. Note that the terms in Λ *should be multiplied by* 10^{-4}

β:	0·2									
μ:	0·14									
ν_1–ν_{10}:	5·335	0·724	0·478	1·333	0·915	0·555	1·239	0·291	0·347	1·100
ε_1–ε_{10}:	0·03	0·003	0·003	0·003	0·003	0·003	0·003	0·003	0·003	0·003
$\Lambda =$	0·32	0·052	0·043	0·009	0·008	0·036	0·006	0·037	0·013	0·036
	0·052	4·0	0·391	0·020	0·009	0·826	0·010	0·092	0·012	0·012
	0·043	0·391	5·0	0·027	0·013	0·174	0·008	0·033	0·008	0·010
	0·009	0·020	0·027	1·5	0·018	0·020	0·006	0·010	0·004	0·004
	0·008	0·009	0·013	0·018	2·5	0·010	0·003	0·006	0·003	0·004
	0·036	0·826	0·174	0·020	0·010	4·0	0·013	0·128	0·014	0·010
	0·006	0·010	0·008	0·006	0·003	0·013	2·0	0·021	0·015	0·004
	0·037	0·092	0·033	0·010	0·006	0·128	0·021	6·0	0·025	0·013
	0·013	0·092	0·008	0·004	0·003	0·014	0·015	0·025	6·0	0·013
	0·036	0·012	0·010	0·004	0·004	0·010	0·004	0·013	0·013	2·5

an average within- to between-region interaction ratio of about 12:1 in the case of Bristol and 70:1 in the case of the other G.R.O.s. Given the city region structure of the selected study area, we would expect, as these figures reflect, a much higher interaction rate between Bristol and the other G.R.O.s, than between the other G.R.O.s themselves.

RESULTS FOR THE BRISTOL REGION SIMULATIONS

The ten sub-area system

The results produced by two runs of the model appear in Figures 6 and 7. The order of the series in both these figures corresponds exactly with that of Figure 2. A FORTRAN listing of the program is available on request from G.D.M. The numerical values of all the parameters used in the model, determined as described above, are given in Table VI. The differences between the two figures are solely the result of using different pseudo-random numbers in the simulation procedure, and so give some idea of the sampling variability that can arise in this kind of model. The runs were started with the susceptible population in each G.R.O. slightly in excess of that G.R.O.'s critical μ/λ_{ii} ratio. Just ten latent and five contagious individuals were 'planted' in Bristol; no carriers were planted in any of the other G.R.O.s. Comparison of Figures 6 and 7 with Figure 2 shows the close correspondence of the simulated results to reality; in particular the noticeable phase differences between G.R.O.s are successfully modelled. Given the small pocket of infection used to seed the model, those results bring home the extent to which a large scale spread of measles can occur in a regional system, once the susceptible population is large enough, if only a very small pocket of infection appears in the area. This picture is characteristic of measles epidemics in reality. Finally, we note that in the case of the simulated results for Bristol, there is some evidence of damping of the epidemic waves with time (see following section).

To investigate the importance of the regional interaction parameter matrix, Λ, in determining the spread pattern of the disease among the G.R.O.s, we tried several different sets of values for the terms in this matrix. All the other parameters in the model were held at the values already determined. Figure 8 shows the results obtained with the $\{\lambda_{ii}\}$ taken to be the same in each region, and all the $\{\lambda_{ij}\}$ at half of this value. With such very high $\{\lambda_{ij}\}$ in proportion to the $\{\lambda_{ii}\}$, compared with the proportions used to generate Figures 6 and 7, we obtain a

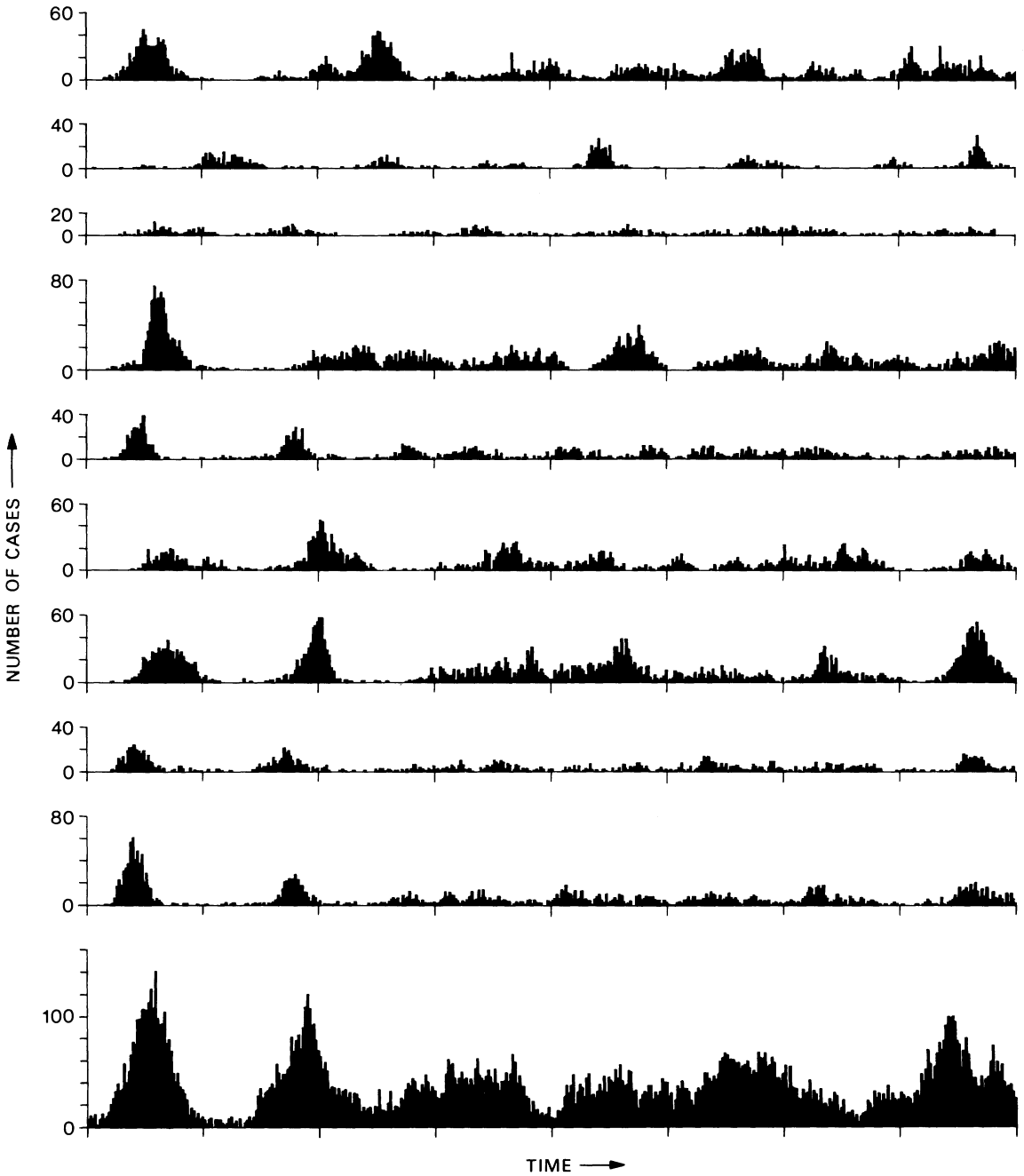

FIGURE 6. Simulated notifications for the ten area system using a given random number stream. The order of the series from top to bottom is Long Ashton, Clutton, Keynsham, Bath, Warmley, Thornbury, Sodbury, Mangotsfield, Kingswood and Bristol, as in Figure 2

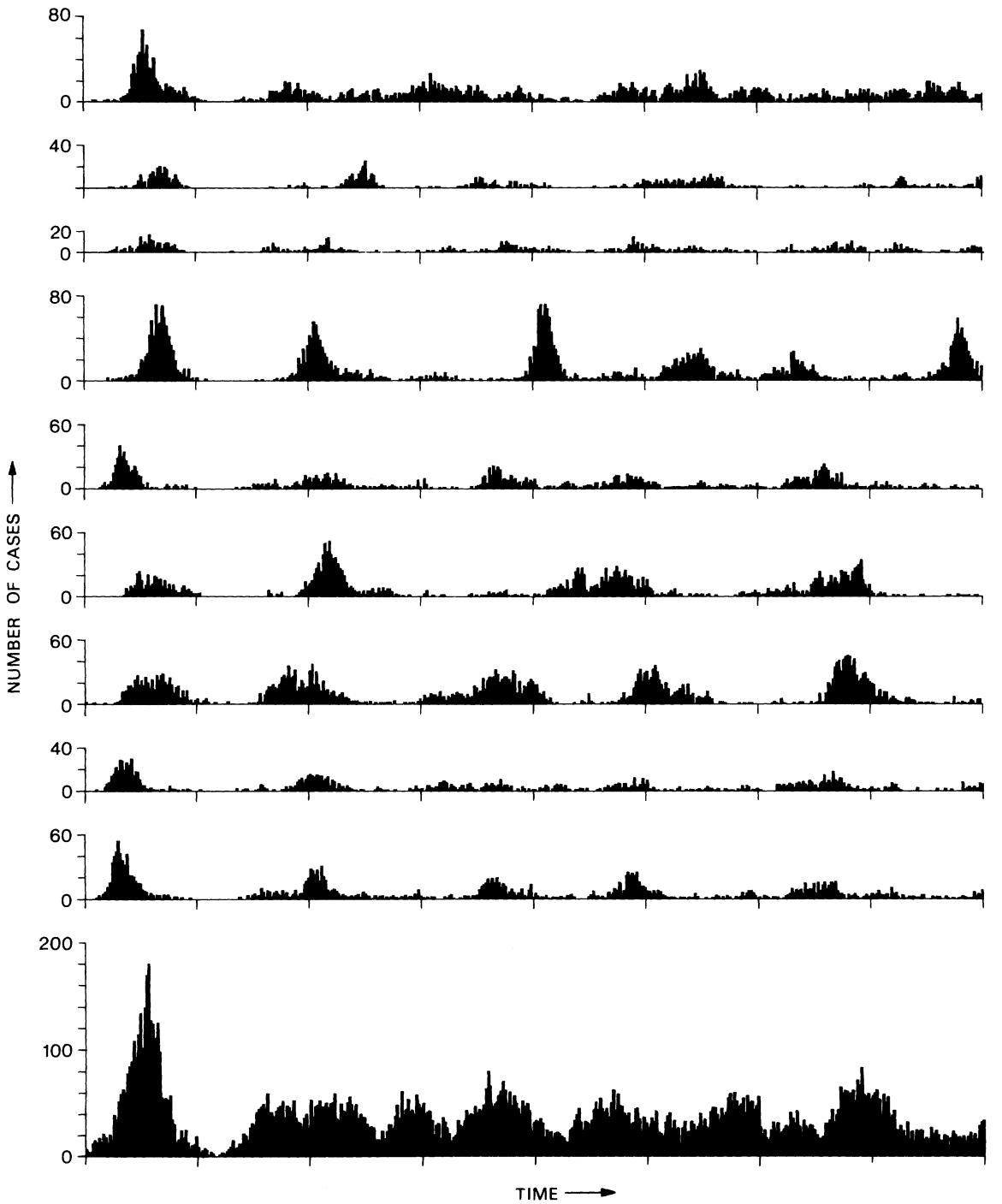

FIGURE 7. Simulated notifications for the ten area system based on a different random number stream to Figure 6

smoothing out of regional differences—all the series are very similar, and there are few noticeable phase differences among the regions in the epidemic starting times.

The problem of Bristol C.B.

The large size of Bristol, with its correspondingly large susceptible population, means that the stochastic model can be closely approximated by the deterministic model in that G.R.O. This fact is reflected in the temporal damping of the simulated epidemic waves in Bristol which we

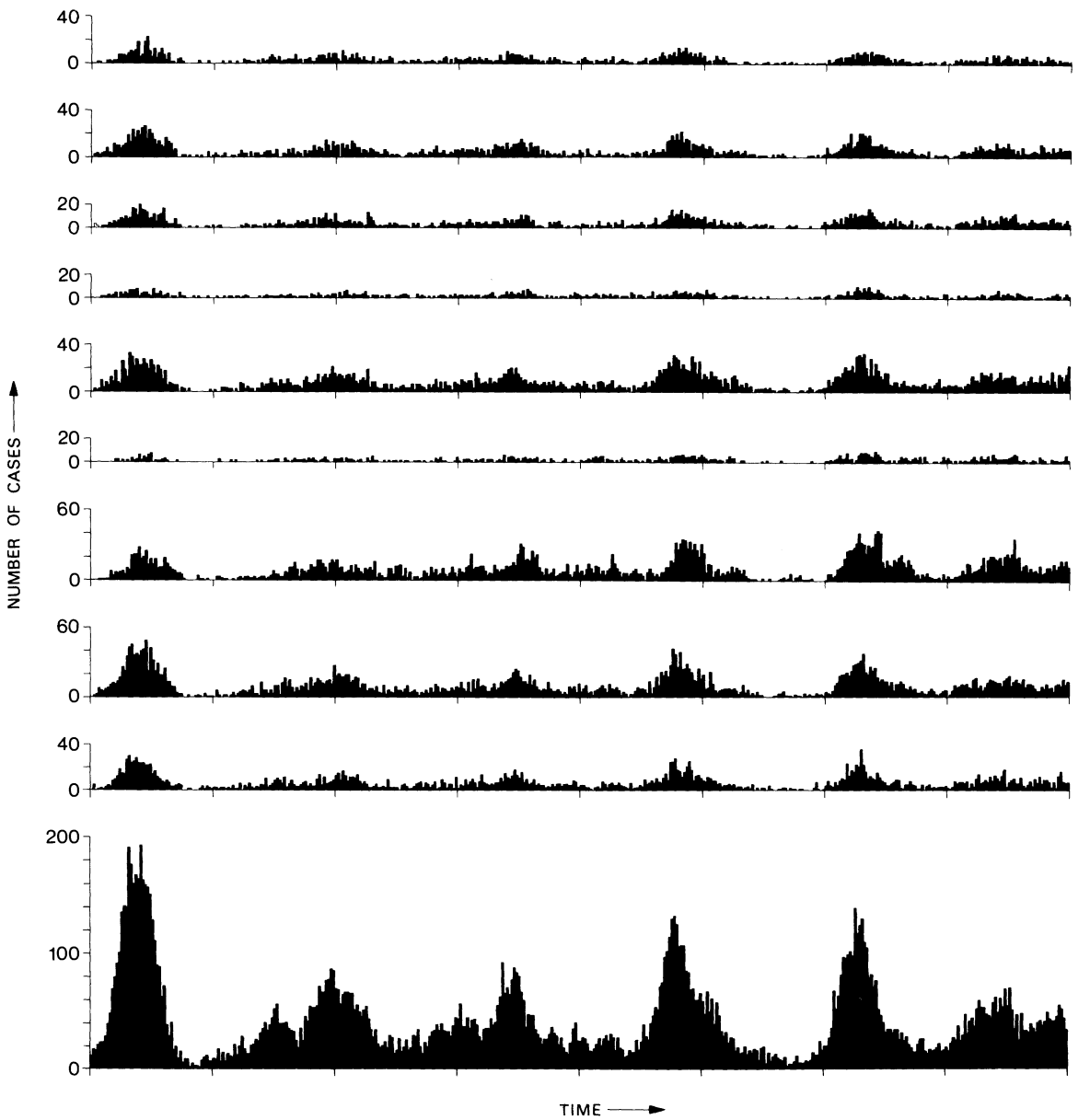

FIGURE 8. Simulated notifications for the ten area system with greater levels of inter-area interaction permitted than in Figures 6 and 7

commented on in connection with Figures 6 and 7. The damping is caused by the unrealistic assumption that the entire population of Bristol mixes homogeneously. To see if any improvement could be made by dropping this assumption, we took a hypothetical city of the same size as Bristol, and supposed that the city could be divided into ten sub-areas. The population of each sub-area was assumed to mix homogeneously, but the sub-areas were assumed to interact. Thus the spatial scale over which homogeneous mixing was taken to occur is greatly reduced.

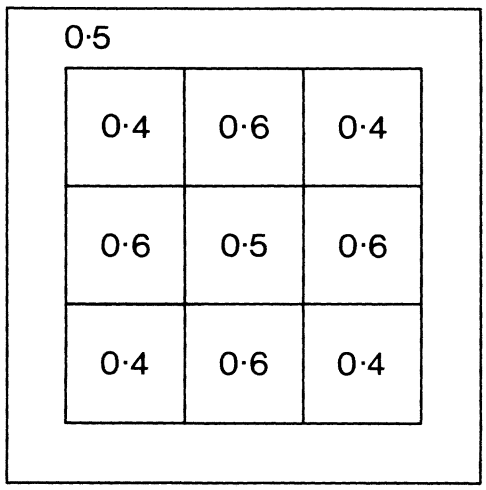

FIGURE 9. Areal subdivisions for the hypothetical city

The areal subdivisions of the hypothetical city are shown in Figure 9. The figures in each sub-area give the sub-areal birth rates, $\{\nu_i\}$. It was assumed that only contiguous sub-areas would interact, and that this interaction rate would be one-third of the within sub-area interaction rates, which were taken to be the same for each community. The latent and contagious periods were fixed at 7 days each, and the immigration rates were one person per year into each community.

Figure 10 shows the results of two separate simulations for this example. Both simulations were run with the same parameter values, but differed with respect to the streams of pseudo-random numbers used. In order to give some comparability between the Bristol C.B. results of the previous section and those for the hypothetical city, both simulations were run with initial population sizes of 450 susceptibles in each sub-area, and ten latent and five contagious individuals in the central community. These figures are (*pro rata* in the case of the susceptible population, and exactly for the L and C populations), the same as for Bristol treated as a whole. Results for the hypothetical city as a unit were obtained by adding together the results for the individual sub-areas.

It is evident from Figure 10, in contrast to the Bristol series of Figures 6 and 7, that there is no tendency for successive epidemic waves to be damped. We thus have the important piece of information that models of the sort considered in this paper should only be applied to regional systems whose component sub-areas are of small or moderate size (say $\leqslant$ 100 000 population), because of the assumption of homogeneous mixing of the S and C populations within communities. If the spatial scale is too coarse, distortion of results will occur. In addition, we note that the simulated notifications of Figure 10 show a greater degree of correspondence with the observed Bristol series of Figure 2 than do the simulated series of Figures 6 and 7.

FIGURE 10. Simulated notifications for the hypothetical city

CONCLUSION

In this paper we have outlined, in a single region context, the Hamer–Soper deterministic model for measles epidemics. Because of the problem of damping of recurrent epidemic waves associated with the model, a stochastic version which overcomes this drawback was outlined and illustrated. The model was extended to a set of interacting sub-areas and was fitted to ten G.R.O.s in the Bristol region. The assumption of homogeneous mixing *within* G.R.O.s suggests that the model should only be applied to small and moderate sized G.R.O.s. Large units, such as Bristol, are best handled by subdividing the unit and then adding together the results for each subdivision to obtain the results for the larger unit. Although the model was able to replicate fairly well the epidemic waves in the Bristol area G.R.O.s, the reader may feel that our methods of fixing the various parameter values of the model were very rudimentary. While it is clear that more precise methods are needed, it is important to remember that, with this type of model, it is usually the *relative* magnitudes and sizes of the parameter values in relation to one another that is important, rather than their absolute values. This is brought home by referring once again to Figures 6, 7 and 8, where changes in the relative level of within- to between-area interaction produced very different outcomes. There is also some evidence for seasonal variations in the virulence of the measles virus (Bailey, 1957, pp. 138–9) which would argue for the inclusion of a periodic element in the model. We note that knowledge of the level of the susceptible populations in G.R.O.s would also make the use of these models more precise. However, while it is evident that many more complicating factors could be introduced into the model, such as seasonally varying migration rates and models for the development of the susceptible population with time, there is a danger of finishing up with a final model which is not parsimonious in the parameters. The formulation proposed here already models reality tolerably well, and so one would want to see real gains in the fit from any additional terms introduced.

In concluding the paper, we note one interesting fact which emerges from the references; namely, the apparently very dated epidemiological literature upon which the work described here is based. In fact, this reflects the lack of progress which has been made since about 1960 in the further development of analytically tractable spatial epidemic models. Thus Bailey (1957, p. 135) has noted 'the complexity of this type of work is formidable, especially when an attempt is made to allow for geographical spread as well'. While it is relatively easy to write down models which are impossible to test empirically, we would stress the very great deal of research which still needs to be done if we are successfully to model space-time processes.

NOTES

1. Where the sizes of the S, I and R populations can be referred to unambiguously without the (t) notation, it will be dropped for simplicity. In addition, it should be noted that throughout this paper, we use the term 'total population' always to refer to the total *under-15* population of the study area; 97 per cent of all measles cases occur in this age group. This concentration of cases in the under-15 age group also implies that analysis at the spatial scale of school catchment areas, as opposed to the local authority scale used here, might be worth pursuing. However, as noted, at the present time, data are only available at the local authority level

2. This is not an unreasonable assumption. Thus, Butler (in the discussion on Bartlett's (1957) paper) has indicated that some 97 per cent of the population contract measles by the age of 15

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