

A Deterministic Model for Measles

John Cunningham

School of Mathematics and Computer Science, University
College of North Wales, Bangor, Gwynedd, LL57 2UW

Z. Naturforsch. **34 c**, 647 – 648 (1979); received March 28/
May 7, 1979

Epidemic Waves, Deterministic Model, Measles, Commu-
nity Size, Recurrent Epidemic

A deterministic model of recurrent epidemics is constructed using a non linear relationship between infection rate and number of contacts. Epidemic waves which are not damped are predicted and a relationship between community size and the period of recurrence is established. A possible explanation of measles outbreaks is suggested.

Introduction

The theory of periodic outbreaks of diseases such as measles dates from a paper by Soper [1]. This model is inadequate because the periodic epidemic waves are invariably damped down to an equilibrium level of infectives. Moreover the Soper model does not predict the observed relations between community size and periodic time: it is observed that epidemics tend to die out or have long periods of recurrence in small communities. To remedy these defects it is generally held that a stochastic approach is required (see Bartlett [2,3] and Black [4]). In this paper a deterministic mechanism is described whereby recurrent epidemics may be generated and by which smaller communities are expected to have less frequent epidemics.

The Model

Let S denote the effective number of susceptibles. In the case of measles this will be some subset of the immature section of the society. Very young children below school age and mature adults rarely suffer from measles. Let I denote the number of infective persons. Let C denote the number of persons who although recovered from the disease have not yet reached maturity (say 15 years old). Let R denote the number of mature persons (who may or may not have had the disease while immature) who are immune and whose number controls the supply of new

susceptibles through the birth rate. The basic equations of the model are

$$\begin{aligned}\dot{S} &= -\lambda (S I)^p - \tau_1 S + \alpha R \\ \dot{I} &= \lambda (S I)^p - \mu I \\ \dot{C} &= \mu I - \tau_2 C \\ \dot{R} &= \tau_1 S + \tau_2 C - \alpha R\end{aligned}$$

where λ , p , τ_1 , τ_2 , μ and α are positive parameters. Here the total population is constant in size and, for simplicity, has a conserved age structure *i. e.*

$$\dot{R} = 0, \quad S + I + C = Y_0$$

where Y_0 is a constant.

When $p = 1$ the model does not differ significantly from the Lotka-Soper model [5].

Equilibrium will occur when $\lambda (S I)^p = \tau_2 C = \mu I$. We shall seek oscillatory solutions by setting

$$S = S_0 + \varepsilon_1, \quad I = I_0 + \varepsilon_2, \quad C = C_0 + \varepsilon_3$$

and neglecting powers and products of

$$\frac{\varepsilon_1}{S_0}, \quad \frac{\varepsilon_2}{I_0}, \quad \frac{\varepsilon_3}{C_0}$$

where S_0 , I_0 and C_0 are the equilibrium values of S , I and C .

Solutions of periodic time $2\pi/\omega$ are possible with

$$\omega = \frac{1}{2} \sqrt{P}$$

provided

$$P = 4\mu^2 p r - \{\mu p (1-r) - \mu + \tau_2\}^2 > 0$$

where $r = I_0/S_0$.

Solutions will contain a factor

$$\exp(Q/2)$$

where

$$Q = \mu p (1-r) - \mu - \tau_2$$

and will be damped if $Q < 0$, amplified if $Q > 0$ and purely oscillatory only if $Q = 0$.

When $p \leq 1$ Q is necessarily negative which is why the original Soper model was discarded. When $p > 1$, however, the solutions oscillate and grow in amplitude so that the approximation by means of which they were derived will eventually become invalid.

We suggest that the values of p and λ should be fixed in such a way that amplification occurs and extinction becomes the likely consequence of the unapproximated equations. The recurrence of the disease

Reprint requests to J. Cunningham.

0341-0382/79/0700-0647 \$ 01.00/0



Dieses Werk wurde im Jahr 2013 vom Verlag Zeitschrift für Naturforschung in Zusammenarbeit mit der Max-Planck-Gesellschaft zur Förderung der Wissenschaften e.V. digitalisiert und unter folgender Lizenz veröffentlicht: Creative Commons Namensnennung-Keine Bearbeitung 3.0 Deutschland Lizenz.

Zum 01.01.2015 ist eine Anpassung der Lizenzbedingungen (Entfall der Creative Commons Lizenzbedingung „Keine Bearbeitung“) beabsichtigt, um eine Nachnutzung auch im Rahmen zukünftiger wissenschaftlicher Nutzungsformen zu ermöglichen.

This work has been digitalized and published in 2013 by Verlag Zeitschrift für Naturforschung in cooperation with the Max Planck Society for the Advancement of Science under a Creative Commons Attribution-NoDerivs 3.0 Germany License.

On 01.01.2015 it is planned to change the License Conditions (the removal of the Creative Commons License condition "no derivative works"). This is to allow reuse in the area of future scientific usage.

and hence the period will depend on the mechanism of re-introduction and a possible model is now described.

We propose that visitations from outside the community by human hosts of the disease are more likely the larger its size and we shall assume that the mean number of infectives visiting the community is proportional to its size N . The modified model is then, on eliminating R

$$\begin{aligned} \dot{S} &= -\lambda S^p (I + \epsilon N)^p + \tau_2 C \\ \dot{I} &= \lambda S^p (I + \epsilon N)^p - \mu I \\ \dot{C} &= \mu I - \tau_2 C \end{aligned}$$

where ϵN is the number of visiting infectives. If $S \rightarrow 0$ and causes the disease to die out the susceptible population will grow and the reintroduction term will restart the epidemic oscillation. The smaller ϵN the longer the periodic time which results. We may have a situation in which $I \rightarrow 0$ without $S \rightarrow 0$ and then the fadeout of disease may persist.

Feasibility Calculations

We shall assume that the model relates to a typical sub-population of 1,000 persons with $Y_0=200$. The calculations have been made using a simple finite difference scheme in which the time step is 0.1 years. We have assumed that an infective has a lifetime of about one third of a time step (about 12 days) and accordingly have set the cure parameter $\mu=0.75$. The transfer parameter has been fixed at $\tau_2=0.01$ (100 time steps from age 5 to 15 years). The factor ϵN has been fixed at 0.05 (Table I) which represents a mean visitation rate of infectives of 10 per 0.1 years per 200,000 total population. Table II shows the effect of reducing ϵN to 0.005 when

Table I. Reintroduction model with $\epsilon N=0.05$.

λ	p	Approx. period in years	Duration in years
0.01	1.3	4.3	0.8
0.02	1.3	2.9	0.6
0.03	1.3	2.3	0.6
0.01	1.5	3.3	0.5
0.02	1.5	2.4	0.5
0.03	1.5	2.0	0.5

Table II. Effect of total population size.

ϵN	Approx. period in years	Duration in years
0.05	2.4	0.5
0.005	4.4	0.6

$\lambda=0.02$ and $p=1.5$ i. e. to 1 infected person per 0.1 year per 200,000.

For purposes of gauging when an epidemic occurs an arbitrary level of 1 infective per 0.1 years per 1,000 has been set.

It is felt that the introduction of the parameter p provides a new feature in the deterministic modelling of epidemics and offers an explanation of recurrent outbreaks whose periodicity depends on population size. More sophisticated models can easily be envisaged e. g. an infection term

$$\sum_{i=1}^n \lambda_i (S I)^{p_i}$$

could be treated but there is little indication from our calculations that additional parameters will be necessary.

[1] H. E. Soper, J. Roy. Stat. Soc. **92**, 34 (1929).
 [2] M. S. Bartlett, J. Roy. Stat. Soc. **A120**, 48 (1959).
 [3] M. S. Bartlett, J. Roy. Stat. Soc. **A123**, 37 (1960).

[4] F. L. Black, J. Theor. Biol. **11**, 207 (1966).
 [5] A. J. Lotka, Nature **3**, 633 (1923).