

It has long been the Society's tradition that, when papers are read and discussed, the early discussants make a vote of thanks to the authors, and I happily do so today. I would extend those thanks, not only to the present authors, but to all the modelling groups, here and abroad, who have tried to make sense of the epidemic as it evolved, and to assess the likely effect of possible ways of mitigating it. Two years ago, we knew nothing about this new virus, and there is still much to learn, but the progress that has been made, alongside the development and manufacture of new vaccines, has been a remarkable achievement.

Tribute too should be paid to the British public, who have accepted radical changes to their way of life in order to cooperate with measures to reduce the spread and virulence of the epidemic. My comments are about the way the public has been informed, and must not be construed as criticism of the detailed technical work behind the scenes.

At the outset, it was explained that, as Kermack and McKendrick had pointed out in 1927, an epidemic has a critical threshold, and it will not take hold unless the initial rate of infections exceeds the rate of recovery or death of infected individuals. The 'R number' is the ratio of these two rates, and if greater than 1 gives a rough idea of the factor by which mixing must be reduced to bring the epidemic under control.

The UK government drew on the work of the various groups modelling the epidemic to announce a national value of the R number, and this has been updated each week. Unusually for official statistics, it takes the form of an interval estimate, currently 1 to 1.2. The length of the interval has varied, and it has often overlapped the critical value 1.

This imprecision is not ordinary statistical variation, because several of the modelling groups announce values of R to 2 decimal places. It seems to be caused by the fact that the estimates from different groups vary widely. This is not surprising, because different models make different epidemiological assumptions, and their definitions of R reflect these. It is not that some are right and some wrong; the late George Box used to say that all models are wrong but some are useful. The usefulness of the different models lies not in their promulgation of R but in the detailed insight they offer into possible interventions to control the epidemic.

The public was not however told that the published R number was a compromise between the artefacts of different models. On the contrary, we were told over and over again that R is the average number of people to whom an infected person transmits the disease. We were therefore invited to envisage the epidemic as a branching process, supercritical when the mean family size R exceeds 1. This lends itself to graphic imagery, showing the successive generations of R^2, R^3, \dots infections.

Since R is dimensionless, this simple picture gives no insight into some of the most important quantities, like rates of hospitalisation or death. It is therefore necessary to introduce time, by making the branching model age-dependent. The theory of age-dependent branching processes was well studied fifty years ago (Mode, 1971), and anticipates some more recent progress in epidemic theory. Thus we specify the way in which the average number R of infections are distributed along the lifetime of the infection. This is a measure of total mass

R , and we then compute its Laplace transform $\varphi(\theta)$. This is a function of a positive real variable θ , with $\varphi(0) = R$, and when $R > 1$ there is a unique value of r with $\varphi(r) = 1$. This value of r is then the rate of exponential increase in the number of infectives.

The equation $\varphi(r) = 1$ occurs several times in the papers for this meeting, first as equation (3) in Parag, Thompson & Donnelly. It is seen as relating R and r , but it only does so if the infectivity distribution is fixed. In practice, interventions do not just change R but may radically alter the distribution. This is particularly germane to the current epidemic, because infections may be transmitted before symptoms appear.

Thus R is not a safe measure of the seriousness of the epidemic. It is possible for R to be decreasing when r is increasing, and vice versa. I therefore think that Parag, Thompson & Donnelly are too generous to the R number, even in the simplest situation. The balance shifts much further against it when it is observed that the current epidemic is far from homogeneous. Throughout the past 18 months, the epidemic has behaved differently in different parts of the UK (let alone the wider world), in different age groups, different social classes and ethnic groups. Care homes and prisons have shown diverse effects. To try to summarise this variation in a single number is absurd.

The simplest way of seeing this is conceptually to disaggregate the population into different 'types' and to invoke the theory of multi-type age-dependent branching processes. The R number must be replaced by an R matrix, whose rows and columns are indexed by the different types, the (i,j) element being the mean number of type j susceptibles infected by an infective of type i . The criticality parameter is now the spectral radius (= largest eigenvalue) of the R matrix, and the epidemic grows exponentially if this exceeds 1. The Laplace transform $\varphi(\theta)$ is replaced by a matrix $\Phi(\theta)$, and the overall growth rate r is the value of θ for which its spectral radius equals 1.

This is where the concept of 'local R ' falls into place. The average number of infections by a type i infective is the i th row sum of the R matrix. Knowing all these row sums does not determine the spectral radius, although it is the case that this lies between the largest and smallest of the row sums.

In this analysis it makes no sense to talk of an overall national R number, but it does make sense to talk of a national growth rate. For decision makers, the primary concern is with total deaths or total hospital beds needed, and these increase exponentially at rate r . It may be difficult to measure the instantaneous value of r , but its compounding effect over days and weeks can be estimated.

To summarise, national R is meaningless, local R does not even determine criticality. On the other hand, to talk of cases increasing by a percentage week on week, or doubling (or halving) in so many days, is understandable and verifiable. It is too late in the current epidemic to banish the R number from public discourse, but I hope it can be pensioned off before the next one.

References

W.O. Kermack & A.G. McKendrick (1927), A contribution to the mathematical theory of epidemics, *Proc. Roy. Soc. A* 115, 700-721.

C.J. Mode (1971), *Multitype Branching Processes*, Elsevier, New York.