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EDITORIAL RESEARCH AND COMMENT

Modelling community spread of Covid-19 without complex mathematics

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Abstract
Aim: Current modelling of Coronavirus (Covid-19) spread relies on complex mathematical equations. I wished to develop a simple spreadsheet model that will allow the public to understand the dynamics of Covid-19 infection without the need to comprehend the underlying equations.
Methods: A simple spreadsheet model taking up 5 columns of cumulative calculations was developed for a population of 1 m. Each row represented a 6-day cycle. The known infection rate R_0 is applied to currently infectious persons but is adjusted downwards as the proportion of the population that remains susceptible declines. Social restrictions designed to slow the spread of the virus were represented in reduced R_0 values. Deaths were estimated at the reported global mortality rate.
Results: In the baseline model without restrictions there was a delay of 55 days before the rate of increase increased rapidly in an exponential manner to a peak at 85 days and cessation of further infections after 100 days. All but 21,000 (2.1%) became infected. There was a peak of 32,000 infections in any one 6-day cycle. A substantial progressive reduction in peak infections were found as R was reduced, but the duration of period of infections increased. At very low values of R the lengthening of the curves became substantial, indicating chronic low-level viral activity.
Conclusions: The model successfully reproduced qualitatively the main results of sophisticated modeling. It supported the notion that viral control can be achieved by taking early draconian measures to reduce the spread and prevent overwhelming pressure on the health system. It also clarified that reducing the peak effect comes at a price of prolonging the epidemic, potentially producing a chronic steady state at low infection rates.
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Introduction

Sophisticated mathematical modeling of epidemic or pandemic spread of infections is based on differential equations containing exponential functions. The sophistication of these models is essential but they have a disadvantage of being difficult or impossible to be understood by readers unfamiliar with advanced mathematics. The model predictions therefore have to be taken by the majority on trust. Complexity also means that the assumptions contained in the model may not be obvious and thus difficult to challenge. Such modeling has been applied[1] to Covid-19 and demonstrates persuasively that the most effective method of tackling its spread and minimize health service demands and deaths is application of unprecedented social measures that prevent person to person contact as soon as possible, when the number of known cases in a community is low – even in single figures.[2,3] The problem in that simple message lies in understandable political inertia to apply unpleasant policies or in recognizing soon enough that an emergency has arisen.

My feeling was that if a non-mathematical approach

could be found, the public understanding of Covid-19 and how it infects a community would be increased, and compliance with restrictive public health policies might be improved. Modeling by spreadsheet is one possibility. I have developed a spreadsheet model to estimate infection rates and mortality with Covid-19 as a function of time using only a few simple assumptions.

Methods

A spreadsheet model (Excel, Microsoft Corporation) of Covid-19 spread was constructed with time in days placed in column A, for a population of 1 million. Cells in the column represents successive 6-day cycles, assumed to equal the average time of spread before the patients become symptomatic. The number of new infections per incident case (R) was assumed to apply only during the asymptomatic phase, after which the active cases in the community remain potentially infectious but do not infect others because of self-isolation or admission to hospital. R_0, the value of R at the initial phased of an outbreak, was 2.6.[4] A mortality rate of 0.04 (4%), obtained from the global rate reported on 1 April 2020[5] (reported value was 4.9%) is applied to the number of infected cases in each cycle. Patients who die are assumed to do so within two 6-day periods and loss of viral load in survivors is assumed to occur after three 6-day periods. These patients are assumed to become non-infectious and to have full immunity to repeat infection. The overall structure of the spreadsheet is shown in Table 1.

Column	A	B	C	D	E	F
Column title	Cumulative infections	Infectious	New infections in each cycle	Susceptible	Dead	R adjustment factor
Calc-ulation	Previous cycle value + Col. C	Col. A − previous Col. A	Col. B × R × Col. F	Original population less Col. A	Col. A × mortality rate	Col. D ÷ baseline population

Table 1. Details of the arithmetical operations applied in each spreadsheet column over time. Col. = Column

The effect of social isolation can be modeled by measuring the effect of reducing R_0. However, the correlation between R and the extent of restrictions is uncertain except at extremes (R_0 = 2.6 for unimpeded infection, or R = 0, at zero social interactions).

The number of patients who remain susceptible to infection at any point is the total population less the number of patients who have ever been infected, which includes those that have died. As this number decreases, the opportunity to infect non-immune (susceptible) patients declines. At each time point R is adjusted by a factor equal to the remaining susceptible cases as a proportion of the baseline population. Thus R falls with time, as must therefore the number of new infections per infectious individual. When R falls to zero there are no new cases and the number of deaths will plateau. This conclusion disregards possible reinfection from asymptomatic carriers in the population, but this is of uncertain significance.[6] An implicit assumption which is clearly false is that the population is homogeneously distributed around infectious cases so that all at risk have an equal chance of being infected.

Results

The base model (Fig 1) without social restrictions shows that the cumulative number of infections in a previously non-immune population increases exponentially to a peak, after an initial period during which the incidence of disease and its growth are low. New infections then fall rapidly to zero because R declines as a result of the susceptible population adjustment. There is a delay of 50 days before the rapid rate of increase starts, a peak at 80 days and cessation of further infections after 100 days. All but 21,000 (2.1%) of the initial population become infected. There is a peak of 32,000 infections (3.2%) in any one cycle. The infection clears simply because the number of susceptible patients (red in Fig 1) becomes too low for further infection, as survivors develop immunity. Death occurs in 3.9% of the population when a mortality rate of 4% is used in the calculations.

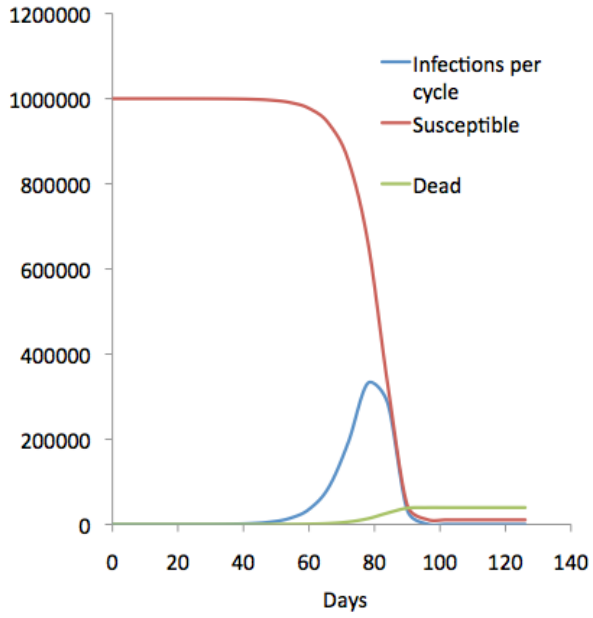

Fig. 1. Model predictions for a non-immune population that becomes infected with Covid-19 and does not take action to contain the epidemic.

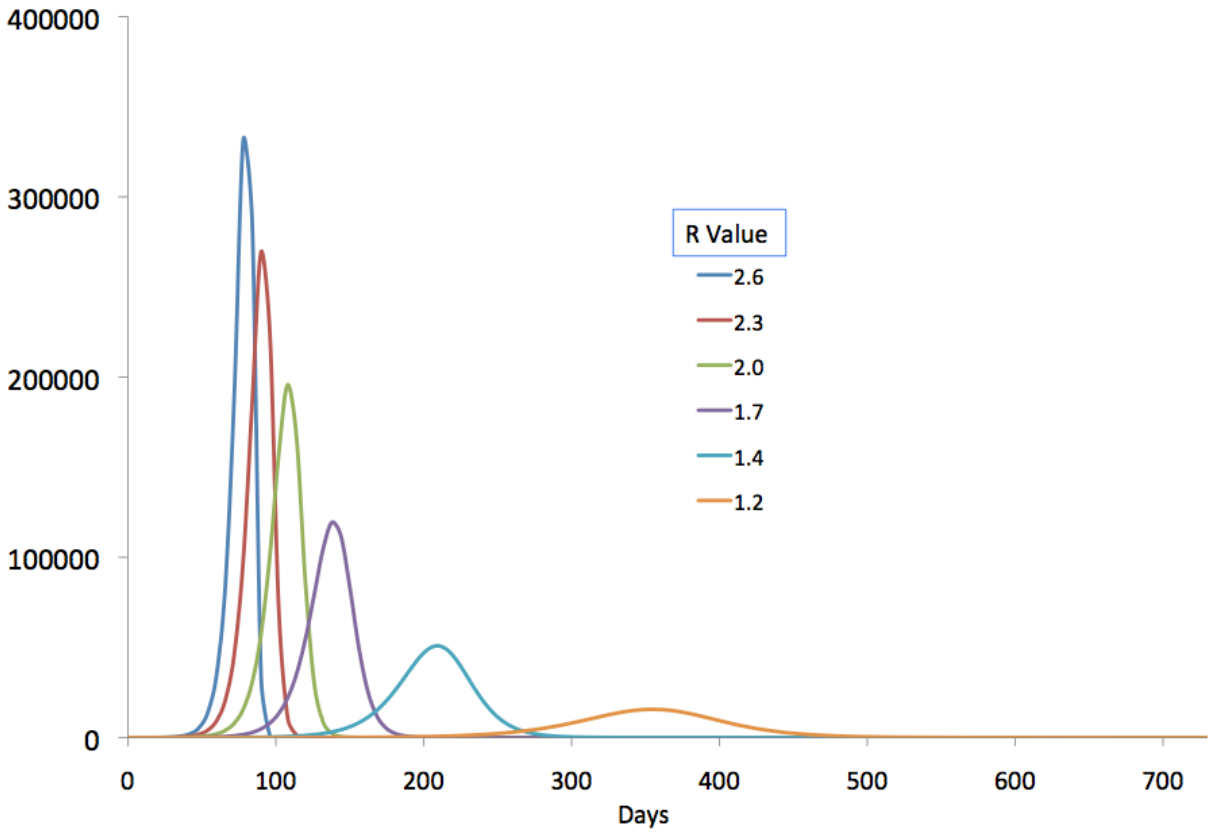

Fig. 2. New cycle (6 days) infection rates according to R (new infections per individual already infected) applied from the onset of Covid-19 outbreak in a population that is non-immune at the outset. The reduction in R is a proxy for social and economic measures taken by an authority to lessen the numbers of infections and deaths. Note that as the peaks reduce the duration of the outbreak increases and at values close to unity a pseudo-steady state is reached at low turnover. At R values < 1 (not shown), the infection at population level cannot be sustained.

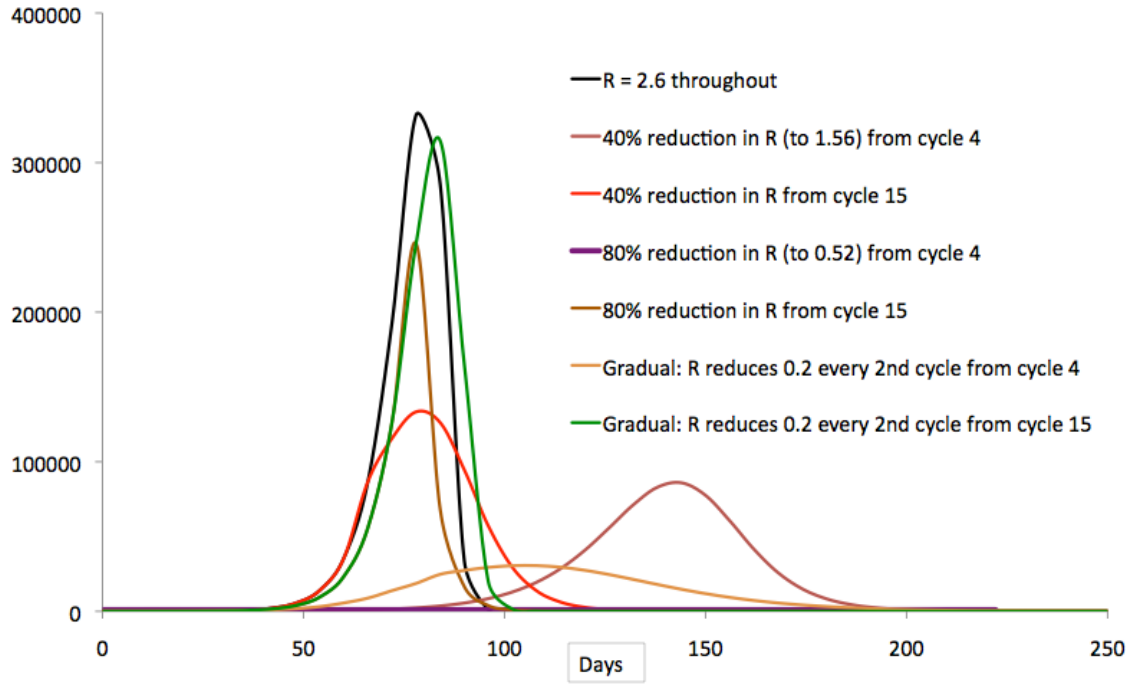

Fig. 3. Patterns of incidence of new Covid-19 infections over time according to different government measures that alter the assumed values of R proxy, as described in the legend. The effect of an 80% reduction in R is barely visible with the ordinate scale used. 40 and 80% reductions in R are equivalent to values of 1.56 and 0.52. R in these calculations is arbitrary and the degree of social measures required to achieve these values is not known.

If R_0 is decreased there is a progressive reduction in the peak numbers infected and the time to peak effect lengthens. Total deaths decline. However, as R_0 declines the total duration of the outbreak is progressively longer (Fig 2). This is because the susceptible population (Fig. 1) and hence the R adjustment factor takes longer to decrease to a value which has significant mitigating effect. Ultimately, R_0 reaches a value at which infections are much reduced but the lengthening of the curve becomes substantial. In other words there is a value of R (in this model at R ranging from 1.0 to 1.4) at which a pseudo-steady state results. In this state, which exists until the entire population is immune, the virus is resident in the population, and infections and deaths continue but at a low rate.

Fig 3 compares the effects of lowering R during the contagion using social means, by applying modest or severe social restrictions that result in 40% or 80% reduction in R continuously, starting early (cycle 4) or late (cycle 15), or by progressive 7.7% decrements (of the initial R; 0.2 absolute) in R applied every second cycle starting at the same time points. The latter method more closely models the stepwise restrictions imposed in practice. The results confirm that the most effective policy is to apply extreme social policies early and maintain them until the contagion is broken, because the duration of social disruption is minimised. Note that the change in favour of early marked social restriction is barely visible in Fig 3 at the ordinate scale deployed in the figure. However, the model also produces a paradoxical result suggesting that application of a modestly reduced R from cycle 4 actually prolongs the contagion, though at reduced rates compared with no intervention or a late application of the same intervention. This is explained by the absence of significant reductions in R due to the adjustment factor, because the susceptible population remains high until later in the outbreak.

Discussion

This spreadsheet is simple and requires understanding only of what is meant by R, what it means to adjust R over time according to changes in the susceptible population, how to calculate deaths as a constant proportion of the infected population, and how to link cells in a spreadsheet. It should be understandable by an average year 7 student and most adults. The spreadsheet values, in which each estimate depends on the value in the previous infection cycle, mean that the growth phase must be exponential. I note that another recent modelling of Australian infection rates by researchers at the University of Sydney[7] showing corresponding curves to the present model is at a pre-publication stage.

The predictions appear reasonable and indeed mirror well those based on sophisticated modeling. However, they may be quantitatively suspect and cannot be used to model situations such as new cases appearing from external sources such as cruise ships. The model is also limited by ignorance over the effect of social restrictions and R. The variation in R in Figs 2 and 3 is therefore empirical and the predictions must be interpreted cautiously. R_0 is as reported in the literature,[2] but may vary between different populations or dissimilar environments within a region or city. Indeed, substantially higher R_0 values (up to 3.6) have been reported.[2] The adjustment to R is also empirical, though it is reasonable to assume that R must fall in line with the number of susceptible persons in a population. The adjustment ensures that the rate of new infections collapses when adjusted R falls below the level at which the outbreak can be sustained. The mortality rate approximates the reported global value in the known infected population. The value[5] on 1 April 2020 was 4.9% but this may be an overestimate if there is a sizeable sub-population that is infected but asymptomatic or otherwise not identified, especially as the sensitivity of RNA testing is low.[8] The model assumes the population is homogeneous with respect to age, geographical spread and mixing with infectious cases.

In spite of these limitations, the model output suggests apparently valid insights into coronavirus spread and how it should be managed:

1. Substantial falls in the peak infection rate and hence demand on health services are observed with only modest reductions in R_0. Thus countries which quickly introduce effective social restrictions may escape precipitous and extreme demand on hospital beds. The total number of infections and deaths (mathematically, the area under the curves) also declines.
2. As R_0 falls and the peak infection rate decreases as above, the duration of the outbreak increases (Fig 2). Eventually an R_0 value (in this model in the range 1.0 to 1.2) is reached at which the curve "flattening" becomes a pseudo-steady state of low numbers of infections, potentially lasting years. R is already low and cannot decline further, and the

population adjustment factor also declines at only a low rate. Effectively, one enters a state of chronic pandemic and the adjusted R is effectively constant. There is a choice between early effective control using draconian social restrictions with major economic consequences, or entering a state of chronic infection with inadequate measures but without severe economic dislocation. Unfortunately the optimum value of R and the level of social restriction required to achieve that value are uncertain. This model does not report the relationship between R and any given degree of social restrction, nor can it be relied on to predict clinical outcomes in a chronic phase. Resort to the more complex models are required for that purpose.

3. Though "exponential" is spoken colloquially to refer to only the phase of rapid increase in infections and deaths, the curve is actually exponential from the first case. Initially the caseload and infection rates are low and this provides an opportunity to plan with little lost if there is a slight delay in applying adequate social measures to contain the virus. Nevertheless the advice to act early and forcefully is valid. Fig 3 indicates that weak interventions even if applied early may produce clinical effects that are the opposite of those intended.

These insights raise major political questions that affect community health. The severe social restrictions imposed in several countries including Australia and New Zealand have resulted in profound social and economic dislocation. In order to avoid negative secondary health and social impacts and permanent economic damage it is essential for the restrictions to be reversed as soon as possible. However, the circumstances under which this will happen have not been defined, or at least they have not been made public. Awaiting complete viral clearance from the community is hardly an option if the emergency enters a prolonged or chronic phase lasting years. In fact, it appears likely that populations or countries could enter a steady-state in which the virus is resident in the population and continues to infect susceptible citizens. This arises because, when even the most severe restrictions include exemptions for essential business activity and essential manpower needs, R can never be zero. Cases of Covid-19 infections amongst doctors and nurses raise the unpalatable prospect that the infection may be spread and perpetuated partly by the very people called upon to treat it.

The natural and expected political imperative is to prevent unnecessary infections and deaths. However, this must be balanced against the adverse chronic consequences of economic shutdown, which will produce its own health hazards, especially in the area of geriatric medicine and mental health. Thus one reaches the conclusion that reversal of restrictions in the interests of resuming normal life must be undertaken *before* the pandemic has been fully controlled.

The above arguments exclude the possible introduction of an effective vaccine and mass application of immunological point-of-care testing, but both may be many months away. The possibility of reaching a point of Covid-19 chronicity has been recognized by the Premier of Western Australia, who has been reported[9] as saying *"If WA maintained just nine new cases each day, in the long term, the curve would likely become too flat and the length of time the state would need to spend under tough restrictions would be untenable."* This is correct. In the same report, the WA Chief Health Officer did not rule out *"...the possibility some of those restrictions would need to be relaxed in coming months if WA maintained very low rates of new cases."* These comments are welcome recognition of the tension between this illness and its economic consequences and point to an urgent need to define the circumstances under which the former gives way to the latter.

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Note: The author notes the headline in the ABC News article (Ref 9). Though that headline may turn out to be true it is not a statement credited to any WA Government spokesman in the article.

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