A Difference Equation Model of Infectious Disease

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Abstract: *In the context of so much uncertainty with coronavirus variants and official mandate based on seemingly exaggerated predictions of gloom from epidemiologists, it is appropriate to consider a revised model of relative simplicity, because there can be dangers in developing models which endeavour to account for too many variables. Predictions and projections from any such models have to be in the context of relevant contingencies. The model presented here is based on relatively simple second order difference equations. The context here is as important as the content in that in many Western counties where the narrative currently seems more important than the truth, and the results of empirical science are valued more as a shield for politicians than a sword for protection of citizens.*

Keywords: *Difference equations, Cayley-Hamilton theorem, Characteristic equation, Vector, Ill-conditioning, Susceptibles, Resistants. Recursive sequences.*

Introduction

The apparently haphazard containment strategies adopted by various countries on the outbreaks of the COVID-19 variants [22] suggest the need to look again at mathematical models of transmission chains. We refer here particularly to difference equation models rather than apparently sophisticated differential equation models, especially in the absence of continuous calibration of data [20]. The latter can result in the danger of making unwarranted assumptions about behaviour between measurement time-points which can then result in errors from ill- conditioning. Exaggerated prognoses as forecasts of doom from such modelling can in turn hasten the advent of national financial over-reactions and individual emotional disturbances, such as examples of Covid child syndrome [16]. It would also seem from documents being collected by the European Medicines Agency that the panic-induced haste caused failures in industry-standard quality management practices during preclinical toxicology studies which did not meet good laboratory practice levels [24].

Since 13 May 2022, cases of monkey pox have been reported to WHO from 12 member states that are not endemic for monkey pox virus, across three WHO regions. Epidemiological investigations are ongoing, but reported cases thus far have no established travel links to endemic areas. It is almost a case of the three-monkey saying: "see no evil, hear no evil, speak no evil" [4].

Monkey pox was first discovered in 1958 when two outbreaks of a pox-like disease occurred in colonies of monkeys kept for research, hence the name 'monkey pox' [9]. The first human case of 'monkey pox' was recorded in 1970 in the Democratic Republic of Congo during a period of intensified effort to eliminate smallpox. While monkey pox seems to be considerably less infectious than respiratory illnesses like COVID-19, and the outbreak is not yet a cause for panic, the rapid spread of the virus appears to signal a shift in its behaviour in European countries with no natural history of monkey research colonies [1]. Moreover, there are few data regarding viral kinetics or the duration of viral shedding and no licensed treatments [18]. Two oral drugs, *brincidofovir* and *tecovirimat*, have been approved for treatment of smallpox and have demonstrated some efficacy against monkeypox in animals. As can be seen from the key references on the topic, there was a flurry of research and development a few years ago, with a renewed interest this year [13].

In the context of so much uncertainty, and with the exaggerated predictions of gloom from seemingly sophisticated modelling, it is time to consider a revised model which is characterised by relative simplicity [6]. This latter has been extended and simplified from a previous version [7]. Furthermore, there can be dangers in developing models which endeavour to account for too many variables [5], where the risk is "Garbage in – Garbage out". In any case, the solutions have to be in the context of relevant contingencies [21]. The "butterfly effect" of an apparently insignificant cause warrants more investigation of models in general [2], possibly by distinguishing the efficient of experimental sciences from the formal causality of mathematical sciences [15]. In the last analysis, a model is no better than the assumptions which go into it.

Materials and methods

Following Makhmudov [14] and in the light of the Covid-19 pandemic, three epidemiological stages in the process of spreading infectious diseases can be postulated:

(i) an initial (incubation) stage of r periods (periods 0, 1, 2, . . ., r − l) during which those who are ill with the disease do not affect others,

(ii) a mature (infectious) stage of periods (periods r, r + 1, . . ., $r + t - 1$) when each person infects s healthy people, and

(iii) a removal stage of m periods (periods $r + I$, . . ., $r + I + m - 1$) when those who have been infected are no longer infectious.

An example might be the common cold which, on average, takes about two days to develop so $r = 2$, a person is then infectious for about three days ($t = 3$), and the symptoms persist for about seven days ($r + t + m = 7$, hence $m = 2$). In general, s is variable, but we shall treat it as a constant in the absence of other information. In terms of a modification to Fibonacci's rabbit problem, these correspond respectively to:

(i) the infancy stage,

(ii) the reproductive stage, and

(iii) the post-reproductive stage.

For the original Fibonacci model, $r = 2$, $s = 1$, and $t = m + \infty$. Following Dubeau [6], let u_n be the total number of disease carriers at the n^{th} period, and v_n^i be the number of i-period old disease carriers at this n^{th} period. More precisely, v_n^i represents the disease carriers in the

a) initial stage for $i = 0, \ldots, r - 1$,

b) mature stage for $i = r, \ldots, r + t - 1$,

c) removal reproductive stage for $i = r + t, \ldots, r + t + m - 1$,

and for $i = r + t + m, \ldots, v_n^i$ represents the disease carriers who have been infected in the past but have already recovered. It is convenient to define u_n and v_n^i for all $n \in Z = \{\ldots, -3, -2, -1, 0, 1, 2, 3, \ldots\}$ and $i \in N = \{0, 1, 2, 3, \ldots\}$.

We consider the following initial conditions on v_n^i:

$$v_n^i = \begin{cases} 0, & \begin{cases} n < 0 \text{ and } i = 0, 1, 2, \ldots, \\ n = 0 \text{ and } i = 1, 2, 3, \ldots, \end{cases} \\ 1 \ (or\ v_0^0), & n = 0 \text{ and } i = 0. \end{cases}$$

Consequently, for any $n \in Z$, $v_n^i = v_{n-1}^{i-1}$ for $i > 0$, and

$$v_n^0 = \begin{cases} 1, & n = 0, \\ s\{v_n^r + \cdots + v_n^{r+t-1}\}, & n \neq 0. \end{cases}$$

From this, we obtain

$$v_n^0 = v_{n-1}^0 + s\{v_{n-r}^0 - v_{n-r-t}^0\},\ n > 1. \tag{1}$$

Then from the definition we have, for any $n \in Z$,

$$u_n = \sum_{i=0}^{r+t+m-1} v_n^i.$$

It follows that

$$u_n = \begin{cases} 0, & n < 0, \\ u_0 + s\sum_{k=r}^{r+t-1} u_{n-k}, & n = 0, \ldots, r + t + m - 1, \\ s\sum_{k=r}^{r+t-1} u_{n-k}, & n \geq r + t + m, \end{cases} \tag{2}$$

or

$$u_n = u_{n-1} - \delta_{n,r+t+m} u_0 + s\{u_{n-r} - u_{n-t}\} \tag{3}$$

for $n \geq 1$, where $\delta_{i,j} = 0$ if $i \neq j$, or 1 if $i = j$.

Let $\{u_n\}_{n=0}^{+\infty}$ and $\{\tilde{u}_n\}_{n=0}^{+\infty}$ be the sequences generated with m and $m + 1$ for the same values of r, t, and s.

From (3) we have, for $n \geq 1$, that

$u_n = u_{n-1} - \delta_{n,r+t+m}u_0 + s\{u_{n-r} - u_{n-r-t}\}$,

$\tilde{u}_n = \tilde{u}_{n-1} - \delta_{n,r+t+m}u_0 + s\{\tilde{u}_{n-r} - \tilde{u}_{n-r-t}\}$.

Next let $\Delta_n u = u_n - \tilde{u}_n$, so that from (3)

$\Delta_n u = \Delta_{n-1}u + (\delta_{n,\ r+t+m} - \delta_{n,\ r+t+m+1})u_0 + s(\Delta_{n-r}u - \Delta_{n-r-t}u)$.

It follows then from (1) that

$\Delta_{r+t+m+n}u = v_n^0$ for $n \geq 0$.

Following Klarner [11], let us consider the sequence of $(r + t + n)$-vectors $\{v_n\}_{n=0}^{+\infty}$:

$v_n = [v_n^0,\ v_n^1,\ \ldots,\ v_n^{r+t+m-1}] \quad (n = 0, 1, 2, \ldots)$.

They are related by the equations

$$v_{n+t} = v_n F = \cdots = v_0 F^{n+1}, \tag{4}$$

where $v_0 = [1,\ 0,\ \ldots,\ 0]$ and $F = (f_{ij})$ is a square matrix of order $r + t + m$ with entries f_{ij} $(i = 0,\ \ldots,\ r + t + m - 1;\ \ j = 0,\ \ldots,\ r + t + m - 1)$, such that

$$v_{n+1}^j = \sum_{i=0}^{r+t+m-1} v_n^j f_{ij}.$$

Within our context,

$$f_{i0} = \begin{cases} s, & i = r, \ldots, r + t - 1, \\ 0, & elsewhere, \end{cases}$$

and for $j = 1, \ldots, r + t + m - 1$,

$$f_{ij} = \begin{cases} 1, & i = j - 1, \\ 0, & elsewhere. \end{cases}$$

The characteristic polynomial of F is

$det(xI - F) = x^{r+t+m} - s(x^{t+m} + \cdots + x^{1+m}) = c_F(x)$.

From the Cayley-Hamilton theorem, the matrix F satisfies its characteristic equation, and we have $c_F(F) = 0$. Hence, $F^n c_F(F) = 0$ for any $n \geq 0$. It follows that

$F^n - s\left(F^{n-r} + \cdots + F^{n-(r+t-1)}\right) = 0$ for $n \geq r + t + m$.

Finally, from (4), we have

$$v_n - s\left(v_{n-r} + \cdots + v_{n-(r+t-1)}\right) = 0$$

and, since $u_n = v_n\,\bar{1}$, where $\bar{1} = [1, \ldots, 1]^{\mathrm{T}}$,

$$u_n - s\left(u_{n-r} + \cdots + u_{n-(r+t-1)}\right) = 0, \text{ for } n \geq r + t + m.$$

Results and discussion

Limit of ratios u_{n+1}/u_n

We now consider the difference Eq. (2) of order $r + t - 1$:

$$u_n = s\sum_{k=r}^{r+t-1} u_{n-k}, n \geq r + t + m$$

The sequence $\{n\}_{n=r+t+m}^{+\infty}$ is completely defined if we assume that the values u_{m+1}, u_{m+2}, …, $u_{r+t+m-1}$ are known.

For our model (2) or (3), we observe that the finite sequence $\{u_n\}_{n=0}^{r+t+m-1}$ is a sequence of nondecreasing integers with $u_0 = 1$ (or any initial value $u_0 > 0$).

We consider two cases for the analysis of the ratios u_{n+1}/u_n the case $t = 1$ and the case $t > 1$.

The Case $t = 1$:
We have

$u_n = su_{n-r}$, $n \geq r + m + 1$.

It follows that

$$\frac{u_{n+r+1}}{u_{n+r}} = \frac{u_{n+1}}{u_n}, n \geq m + 1,$$

and the sequence of ratios u_{n+1}/u_n is a sequence of length r repeated infinitely many times. It is completely characterized by the finite sequence $\frac{u_{n+1}}{u_n}$ for $n = m + 1, \ldots, m + r$.

Using (2), for the initial value $u_0 = 1$, we have

$$u_n = \begin{cases} \sum_{i=0}^{k} s^i, & \begin{cases} n \leq r + m, \\ kr \leq n < (k+1)r \end{cases} \\ \sum_{i=0}^{\left\lfloor \frac{n}{r} \right\rfloor} s^i, & n = 0, \ldots, r + m \end{cases}.$$

Let $\rho_U = \left\lfloor \frac{r+m}{r} \right\rfloor$ and $\rho_L = \left\lfloor \frac{1+m}{r} \right\rfloor$,

then $\rho_U = \rho_L$ or $\rho_L + 1$. Hence, the sequence $\left\{\frac{u_{n+1}}{u_n}\right\}_{n=m+1}^{m+r}$ is such that

$$\frac{u_{n+1}}{u_n} = \begin{cases} 1, & r-2 \text{ times}, \\ \dfrac{s\sum_{i=0}^{\rho_L} s^i}{\sum_{i=0}^{\rho_U} s^i}, & 1 \text{ time}, \\ \dfrac{\sum_{i=0}^{\rho_U} s^i}{\sum_{i=0}^{\rho_L} s^i}, & 1 \text{ time}. \end{cases}$$

It can be shown that the set $\left\{\frac{u_{n+1}}{u_n} | n = m+1, \ldots, m+r\right\}$ converges to the set $\{1, s\}$ when m goes to $+\infty$.

The Case t > 1:
Let $k = r + t - 1$. The linear difference equation (3) is equivalent to the following linear difference equation of order K,

$$u_{n+K} = s\sum_{k=0}^{t-1} u_{n+k}, n \geq 0$$

if the sequence $\{u_n\}_{n=0}^{K-1}$ corresponds to the sequence $\{u_n\}_{n=m+1}^{m+K}$; that is the limit of u_{n+1}/u_n, is the same for both equations.

Let us also recall some definitions and results about linear difference equations of the form:

$$u_{n+K} - b_1 u_{n+K-1} - \cdots - b_{K-1} u_{n+1} - b_K u_n 0 \quad (n \geq 0) \tag{5}$$

a) The polynomial $\varphi(\lambda) = \lambda^K - b_1\lambda^{K-1} - \cdots - b_k$ is called the characteristic polynomial of (5).

b) The equation $\varphi(\lambda) = 0$ is the characteristic equation for (5).

c) The solutions $\lambda_1, \ldots, \lambda_\ell$ of the characteristic equation are the characteristic roots, so that the first result is a standard result about the general solution of (5) [11].

Theorem 1. Suppose (5) has characteristic roots $\lambda_1, .., \lambda_k$ with multiplicities $j_1, .., j_k$, respectively. Then (5) has n independent solutions $n^j\lambda_\ell^n$, $j = 0, \ldots, j_\ell - 1; \ell = 1, \ldots, k$. Moreover, any solution of (5) is of the form

$$u_n = \sum_{\ell=1}^{k}\sum_{j=0}^{j_\ell-1} \beta_{\ell,j} n^j \lambda_\ell^n, \qquad n \geq 0$$

where the $\beta_{\ell,j}$ are obtained from the values of u_n, for $n = 0, \ldots, K-1$.

Proof. See, for example, Kelley and Peterson [15].

The next two results depend on the form of (5).

Theorem 2. Assume the b_i are nonnegative in (5).

- **a)** If at least one b_i is strictly positive, then (5) has a unique simple characteristic root $\sigma > 0$ and all other characteristic roots of (5) have moduli not greater than σ.
- **b)** If the indices of the b_i that are strictly positive have the common greatest divisor 1, then (5) has a unique simple characteristic root $\sigma > 0$, and the moduli of all other characteristic roots of (3.5) are strictly less than σ.

Proof. See Ostrowski [17, pp. 91-92].

Theorem 3. If in (5), the b_i are nonnegative and $\{u_n\}_{n=0}^{+\infty}$ is a sequence satisfying (5) such that $u_0, u_1, \dots, u_{K-1}$ are strictly positive, then we have $u_n \geq \alpha\sigma^n$ $(n \geq 0)$ where $\alpha > 0$ is given by

$$\alpha = min\left\{\frac{u_n}{\sigma^n} \mid n = 0, \dots, K-1\right\}.$$

Proof. See Ostrowski [17, p. 93].

Since

$$b_i = \begin{cases} 0, & i = 1, \dots, r-1, \\ s, & i = r, \dots, r+t-1, \end{cases}$$

and the common greatest divisor of $r, \dots, r+t-1$ is 1 for $t > 1$, it follows from Theorem 2 that (5) has a unique simple characteristic root $\sigma > 0$ and the moduli of all other characteristic roots are less than σ.

Let $\lambda_1, .., \lambda_k$ and σ be the characteristic roots of (5), then, from Theorem 2, we get the form

$$u_n = \beta\sigma^n + \sum_{\ell=1}^{k}\sum_{j=0}^{j_\ell-1}\beta_{\ell,j}n^j\lambda_\ell^n.$$

Moreover, since $u_0 \geq 1$ and $\{u_n\}_{n=0}^{K-1}$ is a non-decreasing sequence, we obtain from Theorem 3, $u_n \geq \alpha\sigma^n$ for $\alpha = min\left\{\frac{u_n}{\sigma^n} \mid n = 0, \dots, K-1\right\}$.

It next follows that:

$$\alpha \leq \frac{u_n}{\sigma^n} = \beta + \sum_{\ell=1}^{k}\sum_{j=0}^{j_\ell-1}\beta_{\ell,j}n^j\left(\frac{\lambda_\ell}{\sigma}\right)^n,$$

and taking the limit on both sides we have $\lim_{n\to+\infty} u_n/\sigma^n = \beta \geq \alpha > 0$ as a consequence of the following lemma.

Lemma 1. If $|\rho| < 1$, then $\lim_{n\to+\infty} n^\alpha\rho^n = 0$ for any $\alpha = 0,1,2, \dots$.

Finally,

$$\frac{u_{n+1}}{u_n} = \sigma \frac{u_{n+1}/\sigma^{n+1}}{u_n/\sigma^n}$$

and we obtain $\lim_{n\to+\infty} u_{n+1}/\, u_n \, = \sigma$ where σ is the unique positive root of

$$\varphi(x) = x^{r+t-1} - s\sum_{i=0}^{t-1} x^i \,, t > 1.$$

More realistically, in any real population, the epidemiological status of members is as follows:

(i) susceptibles,

(ii) infected, and

(iii) resistants,

and there is not an unlimited supply of susceptibles.

Let

- *N b*e the total population,
- S_n be the number of susceptibles at the n^{th} period,
- U_n be the number of infected and carriers at the n^{th} period, and
- R_n be the number of resistants at the n^{th} period.

Then $N = S_n + U_n + R_n$ and the initial conditions are $S_0 = N - 1,\ U_0 = 1$, and $R_0 = 0$.

By using the previous notation, we have that

$Un = \sum_{i=0}^{r+t+m-1} v_n^i$,

$$Rn = \sum_{i=r+t+m}^{+\infty} v_n^i$$

$$= \sum_{i=r+t+m}^{+\infty} v_{n-i}^0$$

$$= \begin{cases} 0, & if\ n < r+t+m, \\ \sum_{i=r+t+m}^{n} v_{n-i}^0 \,, & if\ n \geq r+t+m, \end{cases}$$

$S_n = N - U_n - R_n$,

but, the number of susceptible subjects is limited, so that

$$v_n^0 = min\left\{ S_{n-1}, s\sum_{i=r}^{r+t-1} v_n^i \right\}$$

and

$S_n = S_{n-1} - v_n^0$,

$U_n = U_{n-1} + v_n^0 - v_n^{r+t+m}$,

$R_n = R_{n-1} + v_n^{r+t+m}$.

For R_n we now have that

$$R_n = R_{n-(r+t+m)} + \sum_{i=0}^{r+t+m-1} v_{n-i}^{r+t+m}$$

$$= R_{n-(r+t+m)} + \sum_{i=0}^{r+t+m-1} v_{n-(r+t+m)}^i$$

$$= R_{n-(r+t+m)} + U_{n-(r+t+m)} = N - S_{n-(r+t+m)}.$$

It follows that

$U_n = S_{n-(r+t+m)} - S_n$

and, if $S_n = 0$, then

$U_n = S_{n-(r+t+m)}$.

Without a limit on susceptibles,, $\{U_n\}$ becomes part of the sequence {1,1,2,3,5,7,11,15,23,33,49,71,105,153,225,329,483,707,1037,1519,2227,…}, so that U_{20} would then be 2227.

The sequences for $\{U_n\}$ and $\{R_n\}$ in the table can be readily extended for larger cohorts beyond the maximum turning points in the columns as the sequences are part of A306276 and A023435, respectively of [20]. They are parts of lacunary recurrence relations [21] which are respectively 4th and 5th order recursive sequences which can be separately related to the difference equations of this paper.

Table 1. Theoretical application of the model for $r = 2$, $t = 3$, $m = 2, s = 1$ and $N = 200$.

$N \setminus j$	0	1	2	3	4	5	6	U_n	R_n	S_n
0	1	0	0	0	0	0	0	**1**	**0**	**199**
1	0	1	0	0	0	0	0	**1**	**0**	**199**
2	1	0	1	0	0	0	0	**2**	**0**	**198**
3	1	1	0	1	0	0	0	**3**	**0**	**197**
4	2	1	1	0	1	0	0	**5**	**0**	**195**
5	2	2	1	1	0	1	0	**7**	**0**	**193**
6	4	2	2	1	1	0	1	**11**	**0**	**189**
7	5	4	2	2	1	1	0	**15**	**1**	**184**
8	8	5	4	2	2	1	1	**23**	**1**	**176**
9	11	8	5	4	2	2	1	**33**	**2**	**165**
10	17	11	8	5	4	2	2	**49**	**3**	**148**
11	24	17	11	8	5	4	2	**71**	**5**	**124**
12	36	24	17	11	8	5	4	**105**	**7**	**88**
13	52	36	24	17	11	8	5	**153**	**11**	**36**
14	36	52	36	24	17	11	8	**184**	**16**	**0**
15	0	36	52	36	24	17	11	**176**	**24**	**0**
16	0	0	36	52	36	24	17	**165**	**35**	**0**
17	0	0	0	36	52	36	24	**148**	**52**	**0**
18	0	0	0	0	3	52	36	**124**	**76**	**0**
19	0	0	0	0	0	36	52	**88**	**112**	**0**
20	0	0	0	0	0	0	36	**36**	**164**	**0**
21	0	0	0	0	0	0	0	**0**	**200**	**0**

Conclusion

The work in this paper outlines the steps involved to utilize a relatively simple model of the spread and management of an infectious disease. The model utilizes well-known properties of second order difference equations; this is a modification of previous work. The theoretical example indicates that it is realistic. In practice, the parameters of susceptible, infected and resistant subjects should normally be readily available [8]. In practice, the total numbers within any cohort vary during the course of an epidemic, but there are statistical methods to modify the cells of the table [19].

That it could not be applied to more extensive real data is mainly due to the almost haphazard and random collection of medical data for Covid-19 by public health authorities during the last three years. Much of the existing data has been contaminated by changing collection criteria even within single state jurisdictions, so there is no readily accessible and reliable common pro-forma across jurisdictions at the time of preparation of this paper. This paper is merely trying to sensitize epidemiologists to simplify their approaches. “The COVID-19 pandemic has once again reminded us the need for a responsive, intelligent and robust surveillance system that will rapidly provide an early warning and a timely response against unforeseen challenges. Learning and responding rapidly to emerging novel pathogens will not only benefit China but also globally” [3].

Most countries introduced draconian measures, with untested assumptions which are sometimes based on very little experimental evidence, to justify the safeguarding of their citizens in ways that can have long-term emotional, physical and financial consequences because of exaggerated and alarming prognoses from inappropriate mathematical modelling. The mathematical context of the times are the background painted by Larcombe [12] and the scientific context is on display in the Springer Nature Australian Academy of Science investigation of research integrity in third-five Australian universities [22].

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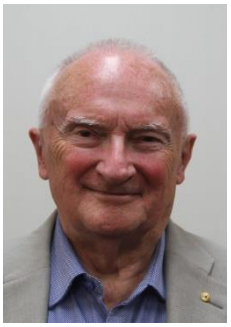

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