Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Mathematical Biosciences 351 (2022) 108885

Contents lists available at ScienceDirect

Mathematical Biosciences

journal homepage: www.elsevier.com/locate/mbs

Original Research Article

Minimising the use of costly control measures in an epidemic elimination strategy: A simple mathematical model

Michael J. Plank

School of Mathematics and Statistics, University of Canterbury, Christchurch 8140, New Zealand

ARTICLE INFO

Keywords:
Branching process
Epidemiological modelling
Infectious disease modelling
Public health

ABSTRACT

Countries such as New Zealand, Australia and Taiwan responded to the Covid-19 pandemic with an elimination strategy. This involves a combination of strict border controls with a rapid and effective response to eliminate border-related re-introductions. An important question for decision makers is, when there is a new re-introduction, what is the right threshold at which to implement strict control measures designed to reduce the effective reproduction number below 1. Since it is likely that there will be multiple re-introductions, responding at too low a threshold may mean repeatedly implementing controls unnecessarily for outbreaks that would self-eliminate even without control measures. On the other hand, waiting for too high a threshold to be reached creates a risk that controls will be needed for a longer period of time, or may completely fail to contain the outbreak. Here, we use a highly idealised branching process model of small border-related outbreaks to address this question. We identify important factors that affect the choice of threshold in order to minimise the expect time period for which control measures are in force. We find that the optimal threshold for introducing controls decreases with the effective reproduction number, and increases with overdispersion of the offspring distribution and with the effectiveness of control measures. Our results are not intended as a quantitative decision-making algorithm. However, they may help decision makers understand when a wait-and-see approach is likely to be preferable over an immediate response.

1. Introduction

In response to the Covid-19 pandemic, several countries adopted an elimination strategy for varying periods of time, including Australia, New Zealand, China and Taiwan [1,2]. An elimination strategy requires a combination of strict border controls to minimise imported cases, strong surveillance and an early and effective response to stamp out community transmission [3]. Both Australia and New Zealand experienced numerous border-related re-introductions of SARS-CoV-2 [4,5]. Up until mid 2021, when both countries began to transition away from the elimination strategy, all of these re-introductions were subsequently eliminated. In some instances, this required strict lockdowns, for example in Victoria in July 2020, Auckland in August 2020, and Western Australia in February 2021 [4,6]. However, in the majority of instances, cases were picked up early with a clear link to the border, and outbreaks contained with intensive testing and contact tracing without the need for restrictions [5,7].

Mathematical modelling has played a key role in support of elimination strategies. For example, modelling has been used for situational awareness in conditions of low or zero prevalence [8], to assess risks associated with various border testing and quarantine policies [9–11], to estimate the probability of elimination under specific control measures [6,12], and to inform vaccination and reopening plans [13–15].

Mathematical models lie on a spectrum of complexity, from simple models that are highly idealised, through to complex models that aim to capture underlying processes at a more fine-grained level [16]. Models with differing levels of complexity are useful in different situations. Complex models typically require suitable high-quality data to estimate parameter values and calibrate model output [e.g. 17–19]. Such models can produce quantitative predictions in a specific situation, but may not be readily transferable to other situations or different data sources. Simple models make minimal assumptions about the specifics of the situation and can generate new qualitative insights which are broadly if approximately applicable [e.g. 20]. Idealised models have been used during the Covid-19 pandemic to generate policy-relevant insights [e.g. 21–23].

Branching processes have a long history in infectious disease modelling as a stochastic model that is mathematically tractable [24–26]. Models based around a branching process for transmission dynamics have been used to inform situational awareness and for policy and operational advice during the Covid-19 pandemic in New Zealand [12,27]. Branching processes capture stochastic effects which are important in the early stages of an outbreak when the total number of infections is relatively small. In particular, there is a probability that an outbreak starting from a single seed case will naturally self-eliminate without control. This probability is a decreasing function

E-mail address: michael.plank@canterbury.ac.nz.

https://doi.org/10.1016/j.mbs.2022.108885
Received 4 April 2022; Received in revised form 20 July 2022; Accepted 21 July 2022
Available online 27 July 2022

of the basic reproduction number R_0 but, for a given R_0, is an increasing function of the variance of the distribution of the number of secondary infections per primary infection (referred to as the offspring distribution) [26,28,29]. Pathogens whose transmission is characterised by superspreading, where a minority of cases are responsible for the majority of transmission, have an offspring distribution with high variance. This is associated with a higher probability of self-elimination than pathogens whose offspring distribution has the same mean but lower variance [28].

There is a large body of literature applying principles of optimal control to epidemiological models [30]. In most cases, these focus on finding the optimal time-varying reduction in contacts [e.g. 31,32] or allocation of vaccines [33,34] that minimises the final epidemic size or the cost of intervention. In these problems, the system typically ends in a herd immunity state.

In this paper, we use a highly idealised branching process model to address the question: what is the optimal trigger condition to introduce strong but costly control measures to eliminate a new outbreak? An elimination strategy typically requires a rapid and effective response to stamp out chains of transmission before they become too large [3]. However, there is a trade off which decision makers face in determining the appropriate threshold for introducing stringent control measures. Choosing too low a threshold risks overreacting with numerous lockdowns, leading to unnecessary cost and disruption, and potentially endangering public buy-in to the strategy. Importantly, controls may be imposed unnecessarily for outbreaks that would have naturally self-eliminated without intervention. On the other hand, choosing too high a threshold means control measures may be needed for longer, potentially over a wider geographical area, or may fail to eliminate the outbreak altogether. We do not aim to produce a decision-making algorithm and our results should not be interpreted as quantitatively accurate optima. Rather, we aim to identify qualitative epidemiological features of the transmission dynamics that influence decisions around timing of control measures as part of an elimination strategy. From a control theory perspective, the problem we study assumes that the intervention takes the form of a bang–bang control [32], i.e. control measures are either off or maximal at a given point in time. The problem is different from most epidemic control theory studies as it concerns the optimal way to maintain an elimination state (assuming that is determined to be the policy objective), rather than the optimal way to reach a herd immunity state [31].

2. Idealised discrete branching process model

To obtain a mathematically tractable model in which to analyse the optimal trigger for implementing control measures, we consider a Galton–Watson branching process. This provides is a highly idealised model of infectious disease transmission, with cases assumed to occur in discrete, non-overlapping generations. We use the generation number of the branching process as a proxy for time. The number of new cases in generation n is denoted Z_n and the branching process is defined in the standard way by $Z_0 = 1$ and

$$Z_{n+1} = \sum_{i=1}^{Z_n} X_i, \tag{1}$$

where X_i are independent, identically distributed random variables. The distribution of X_i is referred to as the *offspring distribution* in the absence of control measures. The basic reproduction number R_0 is equal to $E(X_i)$. We assume that, without control measures, $E(X_i) > 1$ so the branching process is supercritical. This means that the probability of self-elimination is strictly less than 1, i.e. some realisations of the process may go extinct without controls but there will be some that do not.

In Section 3 below, we will focus on the case where the offspring distribution is negative binomial, a canonical family of distributions that can be heavy-tailed (i.e. captures the possibility of superspreading-driven transmission dynamics). The negative binomial distribution is characterised by two parameters – the mean R_0 and the dispersion parameter κ – and includes other mathematically relevant distributions, such as the Geometric distribution ($\kappa = 1$) and Poisson distribution ($\kappa \to \infty$), as limiting cases. The variance is $R_0(1 + R_0/\kappa)$ so smaller values of κ are associated with larger variance, i.e. more heavy-tailed offspring distribution, higher probability of zero secondary infections ($P(X_i = 0)$), and higher probability of self-elimination. Other distributions could readily be used within the model framework, however the two-parameter negative binomial family captures a wide range of epidemiological transmission characteristics and can provide a good fit to empirical data from a range of pathogens [28,35]. The parameter κ is a standard epidemiological measure of overdispersion in the transmission dynamics [36] and its value has been estimated empirically from outbreak data for a range of pathogens including SARS-CoV-2 [28,37–39].

We use the simple branching process model to address the question of what is the optimal outbreak size at which to impose control measures aimed at extinguishing the outbreak. To do this we make several simplifying assumptions:

1. Control measures are introduced if and when the number of new cases Z_n is greater than or equal to a pre-defined trigger k.
2. Control measures take effect immediately, i.e. there is no lag between infection, detection and response.
3. Control measures reduce the reproduction number to a known value R_c that is less than 1.
4. Once implemented, control measures remain in place until the branching process goes extinct (i.e. $Z_n = 0$) and are then immediately lifted.
5. The objective is to choose the trigger θ in order to minimise the expected number of generations of the branching process between the introduction of controls and elimination of the outbreak. This is assumed to be a proxy for the time spent under control measures.

This describes a model of an outbreak that starts from a single introduced seed case and which, by definition, always ends in elimination, whether naturally or after introduction of control measures. In reality, one would expect multiple introductions to occur sporadically over time [20]. If the outbreaks caused by these introductions are assumed to be independent and non-overlapping in time, then minimising the expected time spent under controls for a single introduction is equivalent to minimising the expected total time spent under controls over an extended period of time under an elimination strategy. The assumption of independent and distinct outbreaks is reasonable if outbreaks are infrequent and typically small enough they do not produce any significant population immunity, as occurred in New Zealand and Australia in 2020–21 for example [4,5]. If introductions became so frequent that outbreaks from different introductions were occurring simultaneously, the model would no longer apply.

The assumptions listed above are highly idealised, but nevertheless recapitulate a real problem faced by decision-makers. Implementing control measures when the number of cases is too small means that control will be applied unnecessarily to some outbreaks that would have self-eliminated without intervention. Waiting until the number of cases is too large before implementing control measures means that the outbreaks take longer to be eliminated. It is therefore useful to know the theoretically optimal number of cases to trigger control measures to minimise the expected amount of time spent under them. We can calculate this optimum by analysing two distributions: the distribution of the outbreak size if and when control measure are implemented; and the distribution of the number of generations under control measures required to extinguish an outbreak of a given size.

For a given trigger k, we define the two-phase branching process by $Z_0 = 1$, $R_0 = 0$ and

$$Z_{n+1} = \begin{cases} \sum_{i=1}^{Z_n} X_i, & \text{if } Z_m < k \text{ for } m = 1, \dots, n, \\ \sum_{i=1}^{Z_n} X_i^{(c)}, & \text{otherwise,} \end{cases} \tag{2}$$

where X_i and $X_i^{(c)}$ are independent, identically distributed random variables for the offspring distribution with and without control respectively. The optimisation problem could be approached by direct Monte Carlo simulations of the process defined above. However, additional efficiency and insight can be gained by analysing the growth phase and the controlled phase of the process separately.

We define the random variable S to be the outbreak size when the control trigger is first met:

$$S = \begin{cases} 0, & \text{if } Z_n < k \text{ for } n = 1, 2, \dots \\ Z_N, & \text{otherwise,} \end{cases} \tag{3}$$

where the random variable N is the first generation in which the control trigger is met, i.e. $N = \min\{n : Z_n \geq k\}$. We define

$$P_{s,k} = P(S = s \mid k), \qquad s = 1, 2, \dots, \tag{4}$$

i.e. the probability mass function for S when the control trigger is k. Clearly $P_{s,k} = 0$ for $s = 1, \dots, k-1$ and $\sum_{s=1}^{\infty} P_{s,k}$ is the probability the branching process will meet the trigger k before self-eliminating.

In the second phase of the outbreak, control measures are implemented when $Z_n = s$. Define the random variable C to be the number of generations taken for the branching processes to be eliminated. This is equivalent to extinction of s independent Galton–Watson branching processes $\hat{Z}^{(j)}$, each with $\hat{Z}_0^{(j)} = 1$ ($j = 1, \dots, s$) and offspring distribution $X^{(c)}$ with mean $R_c < 1$. Therefore C may equivalently be written

$$C = \min\{n : \hat{Z}_n^{(j)} = 0 \text{ for } j = 1, \dots, s\} \tag{5}$$

The assumption $R_c < 1$ means the s branching processes are subcritical, which guarantees that C is finite with probability 1. We denote the probability mass function for C conditional on the initial outbreak size s as

$$Q_{n,s} = P(C = n \mid S = s). \tag{6}$$

According to the fundamental theorem of branching processes, the probability that a branching processes starting with $\hat{Z}_0 = 1$ has reached extinction by generation n is $q_n = F^{(n)}(0)$, where F is the probability generating function of the offspring distribution under control. By independence of the s branching processes, the probability that the outbreak is eliminated in generation n is

$$Q_{n,s} = q_n^s - q_{n-1}^s. \tag{7}$$

Conditioning over the outbreak size s when control measures are first introduced, we can now write the probability that exactly n generations are spent under control measures for a given trigger k as

$$\theta_{n,k} = P(C = n \mid k) = \sum_{s=0}^{\infty} P(C = n \mid S = s) P(S = s \mid k) \tag{8}$$

This may be expressed compactly in matrix form as

$$\theta = QP \tag{9}$$

This provides an efficient way to calculate θ for a range of offspring distributions X and $X^{(c)}$ since P depends only on the offspring distribution X in the uncontrolled phase, and Q depends only the offspring distribution $X^{(c)}$ in the controlled phase. The objective function for the optimisation problem described above is

$$E(C) = \sum_{n=1}^{\infty} n\theta_{n,k} \tag{10}$$

3. Numerical results

In this section we use a hybrid numerical–analytical method that makes use of the conditional probability distributions in the matrices P and Q derived in Section 2. This is more efficient than full stochastic simulation of the outbreak during the uncontrolled and controlled phases because P depends only the offspring distribution in the uncontrolled phase and Q depends only on the offspring distribution in the controlled phase. We calculate the matrix P defined by Eq. (4) via $N = 5 \times 10^5$ Monte Carlo simulations of the uncontrolled phase of the branching process to find the distribution of outbreak sizes s if and when a given trigger k is first met. Q can be calculated efficiently via Eq. (7) by using the probability generating function $F(\xi)$ for the offspring distribution in the controlled phase. This was approximated via $F(\xi) = \sum_{j=1}^{j_{\max}} P(X_i^{(c)} = j)\xi^j$ where a value $j_{\max} = 100$ was used in numerical computations, sufficient to ensure $P(X_i^{(c)} > j_{\max}) < 10^{-6}$ for all parameter combinations investigated. Once the matrices P and Q have been constructed, the objective function $E(C)$ can be calculated via Eqs. (9) and (10). Matlab code for implementing the numerical calculations is available as Electronic Supplementary Material.

We assume the offspring distribution is a negative binomial distribution $\text{NegBin}(\mu, \kappa)$ with mean μ and dispersion parameter κ. The parameter κ defines the amount of heterogeneity in the offspring distribution: the variance of the distribution is $\mu(1+\mu/\kappa)$ so smaller values of κ representing increasingly heavy-tailed distributions. This is an established epidemiological model for pathogens with superspreading transmission dynamics [28,35]: when κ is small, a minority of cases are responsible for the majority of transmission and a relatively high proportion of cases, $P(X_i = 0)$, do not transmit the pathogen at all. The mean of the distribution μ is $R_0 > 1$ in the uncontrolled phase and $R_c < 1$ in the controlled phase.

Fig. 1 shows the expected time $E(C)$ spent under control measures, and the probability $\sum_{s=1}^{\infty} P_{s,k}$ that control measures are introduced at all, as a function of the control trigger, for $R_0 = 1.6$, $\kappa = 0.25$ and a range of values of R_c. This illustrates the trade offs involved in choosing a control trigger: too small a trigger leads to unnecessary introduction of control measures for outbreaks that would self-eliminate anyway; too large a trigger means it takes longer for control measures to drive the outbreak to elimination.

Fig. 2 shows the optimal trigger for various combinations of the reproduction number in the uncontrolled and controlled phases, and for four values of the dispersion parameter $\kappa = 0.1$, 0.25, 0.5 and 1.0. These values span a range of empirical estimates of κ for real pathogens. For example, estimates for κ for SARS-CoV-2 range from 0.1 to 0.7 [37–39]. Lloyd-Smith et al. [28] analysed empirical offspring distributions for several pathogens, producing estimates for κ ranging from 0.16 for SARS-CoV-1 to 5.1 for Ebola.

The results in Figs. 1 and 2 assume that κ takes the same value in the uncontrolled and the controlled phases of the outbreak. However, it is possible that control measures, such as gathering restrictions or school and business closures, could disproportionately reduce the likelihood of large superspreading events. This would reduce the variance of the offspring distribution (i.e. increase the value of κ). To investigate this possibility, we ran a second set of results where κ was fixed at $\kappa = 1$ in the controlled phase of the outbreak, representing a relatively homogeneous offspring distribution (see Supplementary Figure S1). These results for the optimum control trigger were almost identical to those in Fig. 2, showing that heterogeneity in the offspring distribution matters less in the controlled phase than in the uncontrolled phase. This is because the branching process in the controlled phase is subcritical, meaning that elimination is guaranteed to occur eventually, and the initial number of cases is larger than in the uncontrolled phase, which reduces the impact of stochasticity on the time taken to eliminate.

The results in Fig. 2 illustrate three general principles. (1) The optimal trigger is a decreasing function of the basic reproduction number in the uncontrolled phase R_0. This is because higher values

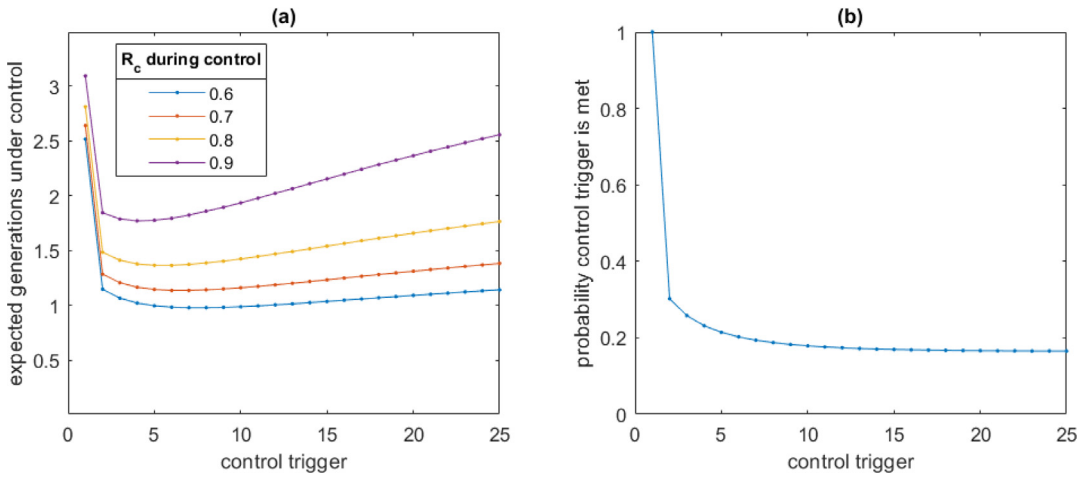

Fig. 1. (a) Expected number of generations of the branching process spent under control measures for different values of the reproduction number in the controlled phase R_c, and (b) probability that the control trigger is met (i.e. that control measures are introduced before the outbreak is eliminated), as a function of the chosen control trigger. Reproduction number in the uncontrolled phase $R_0 = 1.6$ and dispersion parameter $\kappa = 0.25$.

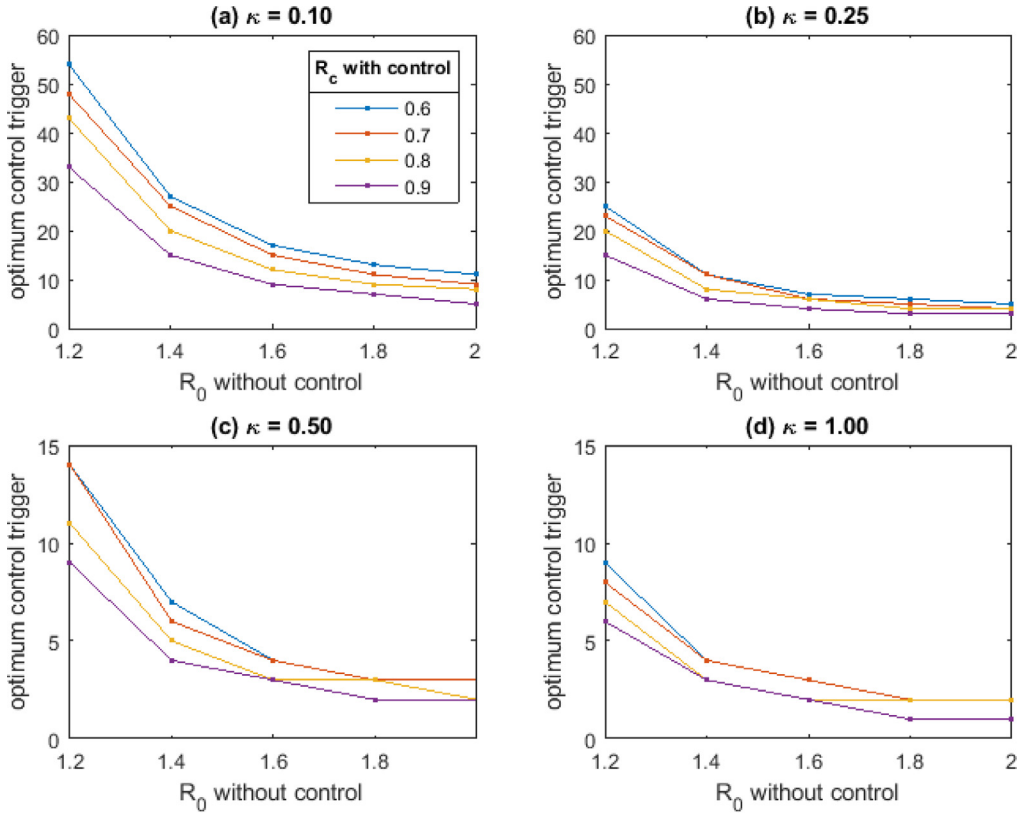

Fig. 2. Optimal trigger k for implementing control measures for combinations of the reproduction number in the uncontrolled and controlled phases and for three values of the offspring distribution dispersion parameter κ. Smaller values of κ correspond to more variance in the offspring distribution. Note different y-axis scales in (c,d) compared to (a,b).

of R_0 correspond to faster-growing outbreaks with lower probability of self-elimination without control. Therefore there is less incentive to take a wait-and-see approach and it is better to implement control when the outbreak is still relatively small. (2) The optimal trigger is a decreasing function of the reproduction number in the controlled phase R_c. This is because the smaller R_c is, the more quickly control measures will extinguish an outbreak of a given size, so outbreaks can be allowed to grow larger before control measures are necessary. (3) The optimal trigger is higher when the offspring distribution is more overdispersed (smaller κ). This is because heterogeneity in individual transmission increases the probability of the outbreak self-eliminating without control measures [28]. When there is a large degree of superspreading, some outbreaks will initially grow quite large but still eventually self-eliminate. In contrast, when the offspring distribution is relatively homogeneous, outbreak growth is more predictable, and once an outbreak has grown beyond even a relatively small size, it becomes highly unlikely to self-eliminate without control measures.

4. Discussion

We have shown results from an optimisation problem that is a highly idealised representation of the decision of when to introduce

stringent control measures in order to minimise the average time they are required to eliminate a small infectious disease outbreak. The advantage of this approach is that, although it makes simplifying assumptions to reduce the model to a caricature of a real decision-making problem, it requires minimal assumptions about the specifics of the pathogen or the population in which it is spreading.

Jurisdictions following an elimination strategy in the early stages of the Covid-19 pandemic are the clearest example of the model's applicability. For example in China, New Zealand and Australia, small outbreaks triggered strict control measures sufficient to reduce the reproduction number under 1 until transmission was eliminated. However, the qualitative model findings are also applicable to future novel pathogens that may prompt an elimination strategy. The key message from this is that a wait-and-see approach is more likely to be beneficial for pathogens with a significant superspreading component but relatively low R_0, and in situations where control measures are known to be highly effective in rapidly reducing transmission once introduced. On the other hand, an earlier response is more likely to be favourable when R_0 is larger, there is less heterogeneity in the offspring distribution, or the effectiveness of the proposed control measures is weaker or more uncertain.

The insights from this study are primarily qualitative and are not a quantitative guide on which to base decision making as part of an elimination strategy. This will always need to take account of specific details, such as the likelihood of undetected cases and whether the cases can be linked to known source, for example via forward and backward contact tracing and whole genome sequencing [4,40,41]. These factors will determine the likelihood of containing the outbreak with case-targeted measures such as contact tracing and isolation. The characteristics of the population affected, and epidemiological properties of the pathogen, such as the generation time and extent of pre-symptomatic or asymptomatic transmission will also affect this assessment.

The model makes highly idealised assumptions about the epidemic transmission dynamics and the effect of control measures on them. These assumptions will not be met in reality, but it is instructive to consider how specific assumptions are likely to affect model results. The model assumes that all cases are detected very quickly after being infected. In reality, some cases will be missed and there will be a lag from infection to detection. This means that the uncontrolled phase of the outbreak is likely to overshoot the control trigger by a greater amount than the model allows for. These effects could be modelled by defining an observation process Y_n on the underlying branching process Z_n that incorporates a specified case ascertainment rate and a distributed delay from infection to detection. Depending on the extent of under-ascertainment and detection lag, this would make the optimal trigger smaller than under the base model with perfect information.

The model assumes that control measures operate in a binary on–off manner and, when in place, have a fixed effect for an indefinite period of time and this is always sufficient to reduce the reproduction number below 1 regardless of the size of the outbreak. This is a crude model of control and does not cover more targeted interventions such as case isolation and contact tracing. In addition, it may be that stringent measures, such as stay-at-home orders or gathering restrictions, become less effective over time as people become fatigued. This could in principle be modelled via a more general objective function, for example $E(C^\alpha)$ with $\alpha > 1$, which penalises long periods of control measures more strongly. We did not attempt to prove that bang–bang control is optimal, and there may be

The model assumes that the value of the reproduction number after controls are introduced is known, whereas in reality this could be difficult to predict in advance. This would add significant uncertainty in the value of the optimal trigger. However, in reality, the decision problem is likely to be faced not just once, but multiple times for example as a result of travel-related re-introductions of the pathogen. This could allow estimates of the effect of controls, and therefore the optimal trigger point, to be refined over time.

The model assumes that control measures remain in place until the outbreak is eliminated and then immediately lifted. In fact, it may be preferable to lift control measures before elimination, for the same reason that it may be optimal to delay their introduction at the start the outbreak: there is a non-zero probability that small outbreaks will self-eliminate in the absence of controls. The optimal trigger for relaxation of controls could be analysed using the same model framework. Furthermore, controls may be more nuanced than a simple on–off switch. We do not claim that such a strategy (known as bang–bang control in the control theory literature) is necessarily optimal and other more gradated approaches are possible. For example, one strategy could be to introduce relatively light controls at a low trigger point, with a higher threshold use to trigger a more stringent intervention.

The objective function used in this model is extremely simple, considering only the duration of fixed control measures. Other considerations are likely to factor into the definition of an appropriate objective function, for example it may be desirable to avoid overly-frequent changes in control settings, or to reduce the total number of cases particularly if these are likely to cause a significant health burden.

We have used a simplified mathematical model to investigate epidemiological factors affecting the optimal use of costly control measures as part of an elimination strategy. We have not considered the circumstances in which such a strategy is or is not feasible or desirable, which is beyond the scope of this study. Australia's and New Zealand's relative geographic isolation allowed them to impose strict border controls in March 2020 before there had been too many introductions, and to maintain such controls for the next 18–24 months [1,2]. This was clearly beneficial for these countries as it meant domestic control measures could be almost entirely relaxed during periods without community transmission, and it allowed the vast majority of the population to be vaccinated before being exposed to the virus [42]. The feasibility of such a strategy will depend on the epidemiological characteristics of the pathogen and an individual country's circumstances, including its travel links and the prevalence of infection domestically and internationally. The results presented here help understand some of the factors affecting the use of control measures in jurisdictions that are following an elimination strategy.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

No new data is presented in this study. Code to run the numerical algorithms and reproduce the results is available as Electronic Supplementary Material.

Acknowledgements

The author is grateful to members of the New Zealand Covid-19 Modelling Government Steering Group for interesting discussions which in part motivated this work. The author thanks two anonymous reviewers for helpful comments on an earlier version of this manuscript.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Appendix A. Supplementary data

Supplementary material related to this article can be found online at https://doi.org/10.1016/j.mbs.2022.108885.

References

[1] J. Summers, H.-Y. Cheng, H.-H. Lin, L.T. Barnard, A. Kvalsvig, N. Wilson, M.G. Baker, Potential lessons from the Taiwan and New Zealand health responses to the COVID-19 pandemic, Lancet Reg. Health-West. Pac. 4 (2020) 100044.

[2] M.G. Baker, N. Wilson, A. Anglemyer, Successful elimination of Covid-19 transmission in New Zealand, N. Engl. J. Med. 383 (8) (2020) e56.

[3] R.N. Binny, M.G. Baker, S.C. Hendy, A. James, A. Lustig, M.J. Plank, K.M. Ridings, N. Steyn, Early intervention is the key to success in COVID-19 control, R. Soc. Open Sci. 8 (11) (2021) 210488.

[4] J. Douglas, J.L. Geoghegan, J. Hadfield, R. Bouckaert, M. Storey, X. Ren, J. de Ligt, N. French, D. Welch, Real-time genomics for tracking severe acute respiratory syndrome coronavirus 2 border incursions after virus elimination, New Zealand, Emerg. Infect. Diseases 27 (9) (2021) 2361.

[5] L. Grout, A. Katar, D. Ait Ouakrim, J.A. Summers, A. Kvalsvig, M.G. Baker, T. Blakely, N. Wilson, Failures of quarantine systems for preventing COVID-19 outbreaks in Australia and New Zealand, Med. J. Aust. 215 (7) (2021) 320–324.

[6] T. Blakely, J. Thompson, N. Carvalho, L. Bablani, N. Wilson, M. Stevenson, The probability of the 6-week lockdown in Victoria (commencing 9 July 2020) achieving elimination of community transmission of SARS-CoV-2, Med. J. Aust. 213 (8) (2020) 349–351.

[7] N. Eichler, C. Thornley, T. Swadi, T. Devine, C. McElnay, J. Sherwood, C. Brunton, F. Williamson, J. Freeman, S. Berger, et al., Transmission of severe acute respiratory syndrome coronavirus 2 during border quarantine and air travel, New Zealand (Aotearoa), Emerg. Infect. Diseases 27 (5) (2021) 1274.

[8] N. Golding, D.J. Price, G.E. Ryan, J. McVernon, J.M. McCaw, F.M. Shearer, Estimating the transmissibility of SARS-CoV-2 during periods of high, low and zero case incidence, MedRxiv (2021) http://dx.doi.org/10.1101/2021.11.28.21264509.

[9] C. Zachreson, F.M. Shearer, D.J. Price, M.J. Lydeamore, J. McVernon, J. McCaw, N. Geard, COVID-19 in low-tolerance border quarantine systems: impact of the Delta variant of SARS-CoV-2, Sci. Adv. 8 (14) (2022) eabm3624.

[10] N. Steyn, M.J. Plank, A. James, R.N. Binny, S.C. Hendy, A. Lustig, Managing the risk of a COVID-19 outbreak from border arrivals, J. R. Soc. Interface 18 (177) (2021) 20210063.

[11] M.J. Plank, R.N. Binny, S.C. Hendy, A. Lustig, K. Ridings, Vaccination and testing of the border workforce for COVID-19 and risk of community outbreaks: a modelling study, R. Soc. Open Sci. 8 (9) (2021) 210686.

[12] S. Hendy, N. Steyn, A. James, M.J. Plank, K. Hannah, R.N. Binny, A. Lustig, Mathematical modelling to inform New Zealand's COVID-19 response, J. R. Soc. N. Z. 51 (sup1) (2021) S86–S106.

[13] T. Nguyen, M. Adnan, B.P. Nguyen, J. de Ligt, J.L. Geoghegan, R. Dean, S. Jefferies, M.G. Baker, W.K. Seah, A.A. Sporle, et al., COVID-19 vaccine strategies for Aotearoa New Zealand: a mathematical modelling study, Lancet Reg. Health West. Pac. 15 (2021) 100256.

[14] Doherty Institute, Doherty Modelling Report for National Cabinet - Revised - 10 August 2021, 2021, www.doherty.edu.au/uploads/content_doc/DohertyModelling_NationalPlan_and_Addendum_20210810.pdf.

[15] N. Steyn, A. Lustig, S.C. Hendy, R.N. Binny, M.J. Plank, Effect of vaccination, border testing, and quarantine requirements on the risk of COVID-19 in New Zealand: A modelling study, Infect. Dis. Model. 7 (1) (2022) 184–198.

[16] E. Brooks-Pollock, L. Danon, T. Jombart, L. Pellis, Modelling that shaped the early COVID-19 pandemic response in the UK, Philos. Trans. R. Soc. B 376 (1829) (2021) 20210001.

[17] M.J. Keeling, L. Dyson, G. Guyver-Fletcher, A. Holmes, M.G. Semple, M.J. Tildesley, E.M. Hill, ISARIC4C Investigators, Fitting to the UK COVID-19 outbreak, short-term forecasts and estimating the reproductive number, Stat. Methods Med. Res. (2020) 09622802211070257.

[18] R. Sonabend, L.K. Whittles, N. Imai, P.N. Perez-Guzman, E.S. Knock, T. Rawson, K.A. Gaythorpe, B.A. Djaafara, W. Hinsley, R.G. FitzJohn, J.A. Lees, D.T. Kanapram, E.M. Volz, A.C. Ghani, N.M. Ferguson, M. Baguelin, A. Cori, Non-pharmaceutical interventions, vaccination, and the SARS-CoV-2 delta variant in England: a mathematical modelling study, Lancet 398 (10313) (2021) 1825–1835.

[19] R.C. Barnard, N.G. Davies, C.A. Pearson, M. Jit, W.J. Edmunds, Projected epidemiological consequences of the Omicron SARS-CoV-2 variant in England, December 2021 to April 2022, MedRxiv (2021) 2021.12.15.21267858.

[20] G.S. Tomba, J. Wallinga, A simple explanation for the low impact of border control as a countermeasure to the spread of an infectious disease, Math. Biosci. 214 (1–2) (2008) 70–72.

[21] M.T. Barlow, A branching process with contact tracing, 2020, arXiv preprint arXiv:2007.16182.

[22] J.R. Gog, T.D. Hollingsworth, Epidemic interventions: insights from classic results, Philos. Trans. R. Soc. B 376 (1829) (2021) 20200263.

[23] J.R. Gog, E.M. Hill, L. Danon, R.N. Thompson, Vaccine escape in a heterogeneous population: insights for SARS-CoV-2 from a simple model, R. Soc. Open Sci. 8 (7) (2021) 210530.

[24] N. Becker, Estimation for discrete time branching processes with application to epidemics, Biometrics (1977) 515–522.

[25] C. Jacob, Branching processes: their role in epidemiology, Int. J. Environ. Res. Public Health 7 (3) (2010) 1186–1204.

[26] J.C. Miller, A primer on the use of probability generating functions in infectious disease modeling, Infect. Dis. Model. 3 (2018) 192–248.

[27] N. Steyn, M.J. Plank, R.N. Binny, S.C. Hendy, A. Lustig, K. Ridings, A COVID-19 vaccination model for Aotearoa New Zealand, Sci. Rep. 12 (1) (2022) 1–11.

[28] J.O. Lloyd-Smith, S.J. Schreiber, P.E. Kopp, W.M. Getz, Superspreading and the effect of individual variation on disease emergence, Nature 438 (7066) (2005) 355–359.

[29] A. James, J.W. Pitchford, M.J. Plank, An event-based model of superspreading in epidemics, Proc. R. Soc. B 274 (1610) (2007) 741–747.

[30] O. Sharomi, T. Malik, Optimal control in epidemiology, Ann. Oper. Res. 251 (1) (2017) 55–71.

[31] M. Kantner, T. Koprucki, Beyond just "flattening the curve": Optimal control of epidemics with purely non-pharmaceutical interventions, J. Math. Ind. 10 (1) (2020) 1–23.

[32] E. Hansen, T. Day, Optimal control of epidemics with limited resources, J. Math. Biol. 62 (3) (2011) 423–451.

[33] H. Behncke, Optimal control of deterministic epidemics, Optim. Control Appl. Methods 21 (6) (2000) 269–285.

[34] H. Gaff, E. Schaefer, Optimal control applied to vaccination and treatment strategies for various epidemiological models, Math. Biosci. Eng. 6 (3) (2009) 469.

[35] S. Blumberg, J.O. Lloyd-Smith, Inference of R 0 and transmission heterogeneity from the size distribution of stuttering chains, PLoS Comput. Biol. 9 (5) (2013) e1002993.

[36] J. Wang, X. Chen, Z. Guo, S. Zhao, Z. Huang, Z. Zhuang, E.L.-y. Wong, B.C.-Y. Zee, M.K.C. Chong, M.H. Wang, et al., Superspreading and heterogeneity in transmission of SARS, MERS, and COVID-19: A systematic review, Comput. Struct. Biotechnol. J. 19 (2021) 5039–5046.

[37] A. Endo, S. Abbott, A.J. Kucharski, S. Funk, Centre for the Mathematical Modelling of Infectious Diseases COVID-19 Working Group, Estimating the overdispersion in COVID-19 transmission using outbreak sizes outside China, Wellcome Open Res. 5 (2020) 67.

[38] J. Riou, C.L. Althaus, Pattern of early human-to-human transmission of Wuhan 2019 novel coronavirus (2019-nCoV), December 2019 to January 2020, Eurosurveillance 25 (4) (2020) 2000058.

[39] A. James, M.J. Plank, S. Hendy, R.N. Binny, A. Lustig, N. Steyn, Model-free estimation of COVID-19 transmission dynamics from a complete outbreak, PLoS One 16 (3) (2021) e0238800.

[40] J. Douglas, F.K. Mendes, R. Bouckaert, D. Xie, C.L. Jiménez-Silva, C. Swanepoel, J. de Ligt, X. Ren, M. Storey, J. Hadfield, et al., Phylodynamics reveals the role of human travel and contact tracing in controlling the first wave of COVID-19 in four island nations, Virus Evol. 7 (2) (2021) veab052.

[41] A. James, M.J. Plank, S. Hendy, R. Binny, A. Lustig, N. Steyn, A. Nesdale, A. Verrall, Successful contact tracing systems for COVID-19 rely on effective quarantine and isolation, PLoS ONE 16 (6) (2021) e0252499.

[42] G. Vattiato, O. Maclaren, A. Lustig, R.N. Binny, S.C. Hendy, M.J. Plank, An assessment of the potential impact of the Omicron variant of SARS-CoV-2 in Aotearoa New Zealand, Infect. Dis. Model. 7 (2022) 94–105.