Manaaki Whenua
Landcare Research

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Short webinars for environmental policy-makers and practitioners

Mathematics, Modelling and Simulation – Supporting the COVID-19 response in New Zealand

The following questions were asked during our live webinar with Rachelle Binny and Audrey Lustig but due to time restrictions, we were unable to answer these in the session.

One of the general issues with EIR models is that they tend to underestimate the peak infection rate overestimate the epidemic persistence after peak has passed? Has this model dealt with it?

Indeed, while deterministic compartment models (e.g. deterministic SEIR models) can provide accurate estimates for long-term dynamics of established epidemics, they are less suitable for exploring transient dynamics because they do not accurately capture the distribution of times that an individual spends in each compartment. As you point out, this can lead to e.g. under-estimation of peak infection rate. To explore short-term scenarios of elimination or containment for the NZ outbreak, which was still in its early stages and had very low case numbers, a stochastic branching process model was more appropriate. This model simulates the number of infections (both clinical and sub-clinical) over time and because it is individual-based (i.e. simulates individuals getting sick and recovering through contacts with other individuals in the population) it should reproduce transient epidemic dynamics more accurately in these short-term scenarios. The branching process can also simulate changes over time in the effective reproductive number (under different Alert Levels for example).

What are the most effective interventions from modelling overseas and in New Zealand? What decreased the R value the most?

Our research looked at the effect of interventions, approximately equivalent to NZ's Alert Levels 1-4, on reproduction number R. In general, countries that had implemented strict lockdown measures equivalent to NZ's Alert Level 4 (including border closures) and that acted early in their outbreak were the most successful at reducing R. While we have not looked at the effect of specific interventions in detail, the international literature suggests that physical distancing can reduce R up to 70% (equivalent of a level 4), good hygiene practice could reduce R up to 20%, strict isolation of cases and contacts can reduce R up to 90%, and excellent contact tracing can reduce R up to 80%. You can explore the

effect of these different interventions on R using our R Calculator on the Take Control simulator app: http://covid19takecontrol.nectar.auckland.ac.nz/covid19_takeControl/

Apologies if you have mentioned this but how do you deal with estimating R when there are multiple imported sources of infection, especially in other countries when you might not have this data?

Your question alludes to an important assumption of many traditional methods used to estimate reproduction number R from case data, which is that any change in numbers of new daily cases are driven by community transmission, as opposed to by changes in testing regime or by imported cases. Where case numbers are low and dominated by imported cases (as is the case in NZ), models that do not account for imported cases will tend to over-estimate R. As you suggest, we were unable to obtain data on number of imported cases for the 25 countries included in our international review so were not able to separate these out from cases arising by community transmission. However, most of the countries included in our review were undergoing large outbreaks with high case numbers where community transmission was likely the dominant driver of case number trends. This means that imported cases should not be a major source of bias in our R estimates. Estimates of R for New Zealand were obtained by simulating our stochastic SEIR model (or branching process) using a range of possible R values (i.e. the effective R under each Alert Level is a model input), then selecting the value that resulted in the best fit between predicted reported case numbers and the real data. Imported cases do not bias R estimates using this approach. The model uses NZ's actual daily numbers of imported cases (with known arrival dates) and simulates daily numbers of domestic cases and sub-clinical (undetected) imported cases. We also performed sensitivity checks and found our estimates to be relatively robust to changes in other model parameters.

In mid-March, Stanford University epidemiologist John Ioannidis wrote that policy makers were creating an economic fiasco based on data that he called 'unreliable'. Has this argument -- which gained real traction in the US and in some circles in the US -- been muted by new data availability?

We believe so. Since mid-March, the volume and variety of available data providing evidence of the severity of SARS-CoV-2 has increased significantly. Current total number of COVID-19-related deaths recorded in the US and globally (which are under-estimates) provides a very simple and inaccurate measure but the sheer scale of these numbers is still a strong indicator for the seriousness of the pandemic. Since John Ioannidis published his article, there have been further studies reporting infection fatality rates: those considered most reliable are tending to report an IFR in the range 0.5% to 1% (more crude estimates place the case fatality rate in the range 0.25-10%, c.f. seasonal flu 0.1-0.2%). However, these are total population estimates and different groups (e.g. people over 65) can have much higher IFRs. Of particular concern for New Zealand, is our finding that infection fatality rates are likely to be much higher (approx. 50% higher) for Māori than for non-Māori (Steyn et al, 2020) and similar findings for indigenous peoples in other countries worldwide. Several seroprevalence studies have been conducted and suggest that cumulative incidence is likely far higher than what has been reported in several countries, but still nowhere near the levels required for herd immunity. The number of studies providing evidence that stringent policy

interventions (e.g. population-wide social distancing) are effective at reducing transmission has also grown. However, this is not to say that the issue of unreliable data has been solved. E.g. changes in testing regimes over time still make it incredibly challenging to accurately model infection trends. There are also major knowledge gaps remaining around the long-term effects of the virus. For example, to what extent people are immune after being infected and how long immunity may last. It is also too early to say what (and how prevalent) the long-term health impacts (e.g. long-term psychological impacts, heart/lung damage, blood clotting, ME/CFS), that could develop in people who survive the initial infection (across all age groups), may be.

My question is about how much better placed you think we are, as a result of this experience, for addressing a similar challenge with a human, animal or plant disease outbreak? What gaps have you seen gaps in NZ's capability? Are they going to be filled?

The current global Covid-19 outbreak illustrates the importance of coordinated interdisciplinary efforts which consider systems-approaches to animal and human disease preparedness. 1) The need for more data, but the right kind: on lesson learn is the need for speed! Quantity, quality, and speed of data access/sharing were key challenges; real-time data collection and sharing is therefore important. We had to adapt through quick fixes and workarounds. How can we keep successful innovation going forward? 2) There is no ethical guidance for model-making. Developing a useful ethical framework for future use will depend on: i) the creation of science–policy partnerships to mutually define policy questions and communicate results; ii) harmonized international standards for model development; iii) strong data stewardship and iv) improvement of traceability and transparency via searchable archives of policy-relevant models. 3) Transparent routes of communication with government: the latter has been essential to build meaningful relationships with policymakers and facilitate effective science-policy translation and knowledge exchange. It ensured that TPM work adds value, avoids unnecessary duplication and is complementary to government priorities. How can we sustain these relationships in "peace-time" as well as during animal disease emergencies? All the above need to be approached in partnership with Māori to ensure the science aligns with Māori values and aspirations