Supporting information

'Imperfect but useful'- could pandemic response in the Global South benefit from greater use of mathematical modelling?

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Outline of the transmission model

Here we provide an overview of the transmission model underlying the simulator, with full technical details published previously [1]. We developed a compartmental, deterministic framework, illustrated schematically in figure S1. The model differentiates three different age groups: 0 – 17 years old (y.o.), 18 – 59 y.o., and >59 y.o. The model also incorporates essential features of the natural history of SARS-CoV-2, including: the fact that not all infections develop symptoms; that even asymptomatic infection can be infectious; and that the risk of severe disease and mortality increases sharply with age [2,3].

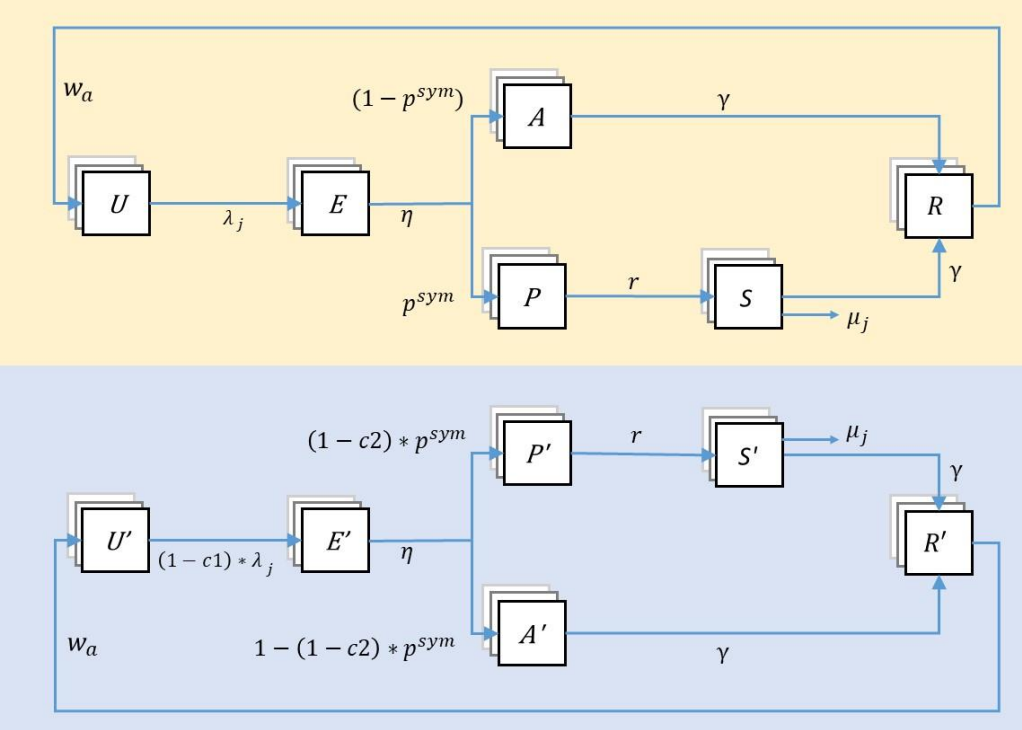

Figure S1. Schematic illustration of the model structure. The upper half of the figure (shaded in yellow) shows the unvaccinated population, while the lower half (shaded in blue) shows the vaccinated population. We modelled ongoing vaccination coverage as a rate of transition from compartments in the upper half to their corresponding compartments in the lower half. Boxes show states representing different stages in the natural history of SARS-CoV-2 infection, while arrows show flows between these states, as a result of infection, recovery, etc. States are as

follows: U, uninfected; E, exposed (latent infection); P, presymptomatic; A, asymptomatic; S, symptomatic; R, recovered and immune. This basic structure is further stratified by three different age groups.

Calibration and modelling of successive waves

The published version of the model [1] incorporated uncertainty in all model parameters, including those related to the natural history of SARS-CoV-2. However, for the purpose of the simulator we chose to present only central estimates in all model projections, for reasons discussed in the main text of the present paper. For the first wave, we modelled as a free parameter the rate-of-infection $\beta^{(1)}$, or the number of infections that would be caused per day by a single infected case in an otherwise susceptible population. We calibrated the value of $\beta^{(1)}$ so that the modelled number of individuals in the 'recovered' compartment in Figure S1 would match estimates of seroprevalence during India's second nationally representative seroprevalence survey in August-September 2020 [4]. At the country level, this seroprevalence was 7.1%, yielding an estimate of $\beta^{(1)} = 0.055$. Using standard methods for calculating the basic reproduction number R_0 [5], this suggests a value of 1.2. However, conditions varied in different states: the simulator thus allowed local users to enter their own locally relevant data for seroprevalence, and updated estimates for $\beta^{(1)}$ accordingly.

To simulate the second wave, we assumed that the end of the first wave would be followed by the introduction of a new virus with infection rate $\beta^{(2)}$. For simplicity we assumed that those infected during the first wave remained immune to the second-wave virus (model results are not substantially affected by relaxing this assumption, owing to the relatively modest size of the first wave). We calibrated the value of $\beta^{(2)}$ so that the relative heights (peak symptomatic incidence) of the second and first waves would be consistent with user-specified data. At the country level, daily reported cases were four times higher during the second wave than during the first wave: using this information, we estimated $\beta^{(2)} = 0.091$. However, the simulator allowed the user to generate their own estimates for this parameter, depending on the second wave peak height in their own settings.

The quantities $\beta^{(1)}$ and $\beta^{(2)}$, as mathematical parameters, are not typically readily recognised by lay users who are untrained in mathematical modelling. Instead, we took account of the fact that both parameters are proportional to the respective values of R_0 for each wave. The basic reproduction number, R_0, is by definition only applicable in the earliest stages of an epidemic where the population is fully susceptible. However, it is also useful as a measure of the *intrinsic* transmissibility of a pathogen, independent of any pre-existing population immunity. In particular, for given values of $\beta^{(1)}$ and $\beta^{(2)}$ for the first and second waves, we calculated corresponding values of R_0 by finding the spectral radius of the next-generation matrix [5].

To model the third wave, as described elsewhere [1], we incorporated three different possible mechanisms:

- Emergence of a novel variant, characterised by two parameters: (i) Its rate-of-infection $\beta^{(3)}$, (ii) the proportion of previously-infected individuals that were susceptible to reinfection with the new variant (to model immune escape in a simple way).
- Lockdown-release, permitting new opportunities for transmission.
- Waning of pre-existing immunity.

For the first of these mechanisms, as described above, the concept of R_0 was more familiar to users than $\beta^{(3)}$. The simulator therefore invited input on R_0 for the third-wave variant, explaining that this parameter was only being used to represent the intrinsic transmissibility of the variant. Using the next-generation matrix as described above, the value of R_0 was then used to calculate the value of $\beta^{(3)}$.

Modelling mitigation measures

As illustrated in Figure S1, we modelled vaccination in a simple way by distinguishing unvaccinated and vaccinated individuals, and assuming a constant rate of transition from the former to the latter. This rate was calculated from user-supplied scenarios for target vaccination coverage, and the duration over which this coverage would be

achieved. We assumed that vaccination protects against infection, with an efficacy consistent with the Covishield (ChAdOx1-S) vaccine [6].

In early discussions about the scope of the simulator we considered the possibility of modelling specific interventions such as school closures. However, we were advised that this approach may detract from the simplicity and transparency of the tool; we therefore chose to model all non-pharmaceutical interventions in a simple way, by assuming that they would act to reduce the rate-of-infection, β. The simulator invited user input for the effectiveness of interventions in reducing transmission, equating this to the reduction in β.

References

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