and r_j is the corresponding eigenvalue. We assume that S varies very slowly compared to the exponential growth of ϕ. This is true when the number of susceptibles is much larger than the number of infected. The coefficients

$$\alpha_j = \left[\begin{pmatrix} | & | & | \\ \hat{e}_1 & \dots & \hat{e}_N \\ | & | & | \end{pmatrix}^{-1} I(0) \right]_j \tag{4}$$

are determined by the initial condition and the contact matrix. Note that because the generator matrix is non-Hermitian, the coefficients are not simply the overlap between the corresponding eigenvector and the state. The theoretical growth rate becomes

$$r_{\text{theo}} = \frac{d}{dt} \ln \left(\sum_k I_k(t) \right) = \frac{\sum_j \sum_k \alpha_j r_j (\hat{e}_j)_k e^{r_j t}}{\sum_j \sum_k \alpha_j (\hat{e}_j)_k e^{r_j t}} \,. \tag{5}$$

Let us arrange the eigenvalues such that r_1 is the largest real eigenvalue and rewrite this as

$$r_{\text{theo}} = \frac{\alpha_1 r_1 \sum_k (\hat{e}_1)_k + \sum_{j>1} \sum_k \alpha_j r_j (\hat{e}_j)_k e^{(r_j - r_1)t}}{\alpha_1 \sum_k (\hat{e}_1)_k + \sum_{j>1} \sum_k \alpha_j (\hat{e}_j)_k e^{(r_j - r_1)t}} \,. \tag{6}$$

Clearly, the components r_j, $j > 1$ are exponentially suppressed for large enough separation between the eigenvalues, but as long as the time is on the same scale as the inverse of this separation, non-equilibrium effects are visible. The time scale $\tau = \frac{1}{\max_{j>1}(\text{Re}\,(r_j) - r_1)}$ thus sets the upper bound of the convergence time, where convergence occurs at time $t \gg \tau$. Note that this is an upper bound as faster decaying contributions may be the largest ones in the beginning. That is, the balance between α_j and r_j in Equation (6) may be such that the convergence is faster than this scale τ.

III. NUMERICAL ILLUSTRATION OF SOLUTION CONVERGENCE

For the β-matrices we use the contact matrices used by the Danish expert group, where different societal structures under different lockdown conditions are used individually as an ensemble of contact matrices [13]. For each matrix, the largest eigenvalue is normalized to the infectiousness of either the Delta or the BA.1 Omicron variant at seasonal strength equivalent to the start of December. The parameters $\eta = 1/4.3$ and $\gamma = 1/3.3$ are used, consistent with [13]. The population is divided into 10 age groups, namely ages 0-4, 5-11, 12-19, 20-29,... , 70-79, 80+, according to the age distribution of the population of Denmark. We initialize with 10^{-5} of the age group population in the respective E-state in three different age groups, one at a time, and follow the progression of the observed growth rate over time. That is, a fit on log scale to the 10 surrounding days. The younger age groups are usually more active, and we therefore expect the growth rate of outbreaks starting in those groups to converge to the dominant eigenvalue faster. This turns out to be the case, and the convergence consistently happens within the first three weeks, see Figure 1 for an illustration. It is clear that an outbreak starting in a less active group will take longer to find the equilibrium state than one starting in the more active group. It is also clear that a higher growth rate leads to faster convergence. Neither of these facts are surprising, but the scale that it happens on is significant. It shows that waiting three weeks before measuring the growth rate is enough to negate this non-equilibrium effect, and it is therefore safe to estimate the growth rate at this point. This effect does not depend on the infectiousness, only the spacing between the eigenvalues as illustrated in Equation (6). Note also that the difference between 10 and 20 points is only a slight shift in the estimated growth rate.

Interestingly, the growth rate starts above the expected irrespective of which age group is infected first. This is not possible in Equation (6), as it is a weighted average of the different eigenvalues, but the larger growth rate comes from the finite-size effect of measuring only at certain times (i.e. once a day).

IV. DISCUSSION

We see that 10 days fit interval gives the fastest convergence, but in reality there are weekly testing patterns which means that at least 14 or 21 days of data are needed to get robust estimates of the growth rates. Furthermore, some groups are more likely to be tested than others so the observed incidences may misrepresent in which group the disease