Calibrated Intervention and Containment of the COVID-19 Pandemic

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Abstract

COVID-19 has infected more than 823,000 people globally and resulted in over 40,000 deaths as of April 1, 2020. Swift government response to contain the outbreak requires accurate and continuous census of the infected population, particularly with regards to viral carriers without severe symptoms. We take on this task by converting the symptom onset time distribution, which we calibrated, into the percentage of the latent, pre-symptomatic and symptomatic groups through a novel mathematical procedure. We then estimate the reduction of the basic reproduction number $R_0$ under specific disease control practices such as contact tracing, testing, social distancing, wearing masks and staying at home. When these measures are implemented in parallel, their effects on $R_0$ multiply. For example, if 70\% of the general public wear masks and contact tracing is conducted at 60\% efficiency within a 4-day time frame, epidemic growth will be flattened in the hardest hit countries. Finally, we analyze the bell-shaped curves of epidemic evolution from various affected regions and point out the significance of a universal decay rate of $-0.32$/day in the final eradication of the disease.
The Coronavirus Disease 2019 (COVID-19) is a new contagious disease caused by the novel coronavirus (SARS-COV-2) (1), which belongs to the genera of betacoronavirus, the same as the coronavirus that caused the SARS epidemic between 2002 and 2003 (2). COVID-19 has spread to more than 170 countries, infected more than 823,000 people and claimed over 40,000 lives as of April 1, 2020 (3). The outbreak has been declared a pandemic and a public health emergency of international concern (4). Given its enormous span over a great variety of communities and nations, data from all sources are needed to formulate successful intervention strategies towards the containment of the disease. At this stage, there exists a wealth of carefully documented clinical data on disease progression and transmission, which can be mined to construct quantitative models of how individual outbreaks rise and cede. Coupled with a systematic characterization of how the disease has spread within and among different communities, a basket of intervention measures can be identified and assessed against the associated economic and social costs.

As the specific symptoms of COVID-19 are now well-publicized, symptomatic transmissions are being contained in most countries. However, disease transmission by pre-symptomatic and asymptomatic viral carriers is much harder to deal with due to its hidden nature (5). Clinical data reveals that viral load becomes significant before the symptom onset (6–8). The epidemiological investigation has identified cases of pre-symptomatic transmission based on the onset time interval studies (9–12). Estimates vary greatly among experts on the percentage of total transmission due to this group of viral carriers, ranging from as low as 18% to over 50% (13–15). A model-based study by Ferretti et al. (16) suggested that pre-symptomatic transmission alone could yield a basic reproduction number $R_{0,p} = 0.9$, close to the critical value of 1.0 that sustains epidemic growth. As the epidemic is driven by the most infectious group at any given period, it is not inconceivable that, as symptomatic transmission loses heat, pre-symptomatic and asymptomatic transmission takes over in fueling the outbreak (5).

To tackle the myriad of issues and questions regarding the outbreak, a simple quantitative model, based on the clinical facts of COVID-19 transmission, is much desired. The model can provide a timely assessment of current intervention measures and of new policies to change the course of the pandemic. In this paper, we show that such a goal is indeed reachable. By transforming the symptom onset time distribution into the reproductive rate since infection, we build a quantitative model that brings out universal features of individual outbreaks. The model allows one to convert the cumulative number of confirmed cases to the current size of the pre-symptomatic population. Subsequently, one can estimate the percentage reduction in the basic reproduction number $R_0$ (estimated to be around 3.68 at a growth rate of
0.3/day) due to contact tracing, wearing masks and other additional measures, individually or in combination. Additionally, we present our findings against the epidemic development curves around the world to highlight the level of social mobilization required to prevent COVID-19 from spreading further.

Model

In epidemiological studies, the central quantity is the average number of infections per unit time $r(t)$ by a viral carrier who was infected at $t = 0$\,(17, 18). In the case of COVID-19, disease transmission by a given individual peaks around his/her symptom onset time (7, 8), as illustrated by the infectiousness curve shown in Fig. 1. This property, when averaged over the population, gives an $r(t)$ that peaks at nearly the same time as the symptom onset time distribution, which we denote by $p_O(t)$. In fact, when the time window of transmission is narrow compared to the mean symptom onset time $\tau_O$, we have approximately

$$r(t) \approx R_0 p_O(t).$$

Equation (1) forms the basis of our analysis. Throughout this paper, we shall use $R_0$ to denote the mean number of secondary infections per infected individual in the population considered.

To incorporate the actual shape of the infectiousness curve, we developed a more complete model as presented in Supplementary Materials (SM). The model splits the pre-symptomatic period into two phases, a non-infectious latent phase L, followed by the infectious pre-symptomatic phase A. As usually done in SEIR type models (19), the infected population is grouped by the symptom phases, denoted as L, A, and S, except that a non-Markovian formulation is required here to accommodate an arbitrary onset time distribution. Starting from infection at $t = 0$, an individual first stays in the latent phase L. Transition to phase A takes place at a rate $\alpha_L(t)$, which increases with $t$. After some time in phase A, the individual develops symptoms and enters the symptomatic S phase at a rate $\alpha_A$, independent of how long the person has been in phase A. The mean duration of phase A is given by $\alpha_A^{-1}$, chosen to correspond to the size of the left-wing of the infectiousness curve. The transmission rate in phase A is set by $\beta_A$. Once in S, the person remains in this phase and the subsequent disease development is not followed. Disease transmission rate $\beta_S(\tau)$ is a function of $\tau = t - t_O$ that matches the right-wing of the infectiousness curve. The total area underneath the infectiousness curve is given by $R_0 = R_0^A + R_0^S$, with $R_0^A = \beta_A/\alpha_A$, and $R_0^S = \int_0^\infty \beta_S(\tau)\,d\tau$. Introducing a parameter $\beta_{\text{eff}} = \beta_A + \alpha_A R_0^S$, we have

$$R_0 = \frac{\beta_{\text{eff}}}{\alpha_A}.$$

Equation (2) forms the basis of our detailed model. In SM, we show that the $r(t)$ of this model can be written in the form of Eq. (1) with a slightly modified onset time distribution. In view of its mathematical simplicity, we will adopt Eq. (1) in the
following and leave the more technical discussions to SM. Denoting by \( A(t) \) the size of the infected population in phase A in a well-mixed community, we have

\[
\dot{A} = -\alpha_A A + \int_{-\infty}^{t} K(t - t_1) A(t_1) dt_1,
\]

with the kernel function given by

\[
K(t) = R_0 e^{-\alpha_A t} \frac{d}{dt} [e^{\alpha_A t} p_O(t)].
\]

An obvious advantage of our formulation, as compared to the more traditional approaches \((16, 20–22)\), is that its parameters have clear physical meaning and thus can be determined directly from clinical case studies. We undertake this task below using available data. By combining two data sets \((11, 29)\) with a total of 159 infection cases, we calibrated the statistical behavior of the symptom onset time as shown in Fig. 2a, and obtained a mean value \( \tau_O = 5.17 \) days, with a standard deviation of 2.93 days. The data is validated against a serial interval study on 468 infection pairs \((9)\) with excellent consistency (see SM). Starting from day 6, \( p_O(t) \) decays exponentially at a rate of \(-0.32/\text{day}\). As will be elaborated later, this decay rate is also seen in the decreasing rate of daily cases when the infectious population is fully isolated and can be attributed to the statistics of the latent period \( t_L \). The parameter \( \alpha_A \) controls the apparent duration of phase A, but its actual value has only a weak effect on our predictions. In our numerical exploration, we use the estimated value \( \alpha_A \approx 0.75/\text{day} \) based on a data set compiled by Xia et al. \((11)\). The basic reproduction number \( R_0 \) reflects the regional social contact pattern in a specific period.

Figure 2b shows the probabilities that a given individual is in one of the three phases at day \( t \) after infection, computed using the formula in Table S1 (SM). The red line marks the boundary between the pre-symptomatic and symptomatic phases. Dashed lines above the red line indicate probabilities that the individual is one day or two days into the symptomatic phase, respectively. The width of the orange region, on the other hand, is proportional to \( \alpha_A^{-1} = 1.5 \) days.

Figure 2c, obtained from the Laplace transforms of these curves, gives the percentage of the infected population in each of the three phases on a given day when the epidemic is growing at a rate \( \lambda \). These curves allow for estimation of the hidden population in L and A from the knowledge of S in real-time and form the basis for quantitative assessment of intervention measures. The actual size of S, which includes all individuals who have developed symptoms in the past, regardless of whether they have recovered from the disease, can be estimated from the total number of confirmed cases until that time point. Note that at high growth rates, probabilities at short times in Fig. 2b contribute more to the Laplace transforms, leading to a larger percentage of the total infectious population being constituted by the hidden population.

Under Eq. (1), the well-known Lotka–Euler estimating equation \((24)\) yields,
\[ R_0 = \frac{1}{\tilde{p}_O(\lambda)}. \]  

(5)

where \( \tilde{p}_O(\lambda) = \int_0^\infty p_O(t)e^{-\lambda t}dt \) is the Laplace transform of \( p_O(t) \). Using our calibrated numbers, we obtain from Eq. (5) the \( R_0-\lambda \) curve shown in Fig. 2d for COVID-19, which covers both growth and decline phases of the epidemic. The slope of the curve at \( R_0 = 1 \) is given by \( 1/T_g \), where \( T_g \) is the mean generation time and equals \( \tau_O = 5.17 \) days under Eq. (1) (see Fig. 2a). At the very high growth rate of \( \lambda = 0.3/\text{day} \) seen in China in late January 2020 and now in Europe and the US in March 2020, our estimated value of \( R_0 \) is 3.68. According to Eq. (2), \( R_0 \) is proportional to the transmission parameter \( \beta_{\text{eff}} \). Thus if one is to rely on social distancing alone, the number of close social contacts per individual needs to be reduced to 27\% of its original level to reach \( R_0 = 1.0 \) so as to halt exponential growth, highlighting the tremendous sacrifice required to curb a rapidly growing outbreak. The left end of the curve gives an ultimate epidemic decay rate of \( -0.32/\text{day} \) at \( R_0 = 0 \), i.e., a complete eradication of disease transmission.

**Evaluation of Intervention Measures**

To lessen the impact of social distancing practices on the general public, governments have mainly adopted two measures to track COVID-19 transmission: 1) testing and isolating infected individuals; and 2) tracing and quarantining contacts of infected individuals. For testing control, persons who had close contact with a confirmed infection case are asked to undergo voluntary or mandatory testing for infection, and quarantined when the result is positive. From Fig. 2b we see that, if the test is conducted too close to the day of infection, the individual has a high probability to still be in the latent phase, hence the test result is likely to be negative. On the other hand, if the test is conducted too late, the person may have already infected others so that the reduction of \( r(t) \) given by Eq. (1) is small. Therefore, there is an optimal interval between infection date and the test date, which we analyzed in SM. Figure 3a shows the function \( g(t) = r(t)/R_0 \) without intervention, and in three examples when the test was performed on day 2, day 3 and day 4 after infection, under the best-case scenario when all close contacts were traced and test results were immediately available. Relative change of the basic reproduction number \( R_0 \) is given by the sandwiched area in each case. In Fig. 3b, we show the reduction of \( R_0 \) as a function of the test date after infection for immediate reporting (red line) and delay of result by one day (blue line), with an initial value of \( R_0 = 3.68 \). The best result is obtained when the test is performed on day 4. This corresponds to the day when the width of the orange region in Fig. 2b is the widest. The actual values depend on the total width of the infectious interval, which should also extend to the symptomatic side if self-quarantine is not assumed.

For contact tracing and quarantining, we show our results for aggressive contact tracing under the scenario that all close contacts are traced and quarantined on day \( t \) after infection, without testing the individual for the virus (Fig. 3b). The reduction of \( R_0 \) is much greater if full tracing and quarantining are executed within 2-3 days
after infection. An 80% tracing efficiency shrinks the time window to 1 day for achieving the same effect. The above estimation procedure can be generalized to cases when testing or contact tracing is completed within a given period rather than on a particular day in terms of a weighted average of the reductions (see SM). These numbers can be used to design optimal strategies that combine social distancing, testing and contact tracing to contain the epidemic in a particular region, taking into account the local political, economic and social situations.

Other than government-led intervention, individual-led interventions, including social-distancing, mask-wearing, frequent hands washing, etc., may reduce disease transmission and slow down the outbreak. Among them, population-wide mask wearing is currently under debate (25). It is enforced in most Asian countries, but not recommended by the CDCs in the EU and USA as of 31 March, 2020. Given the now established risk of pre-symptomatic transmission, and the dominant role of droplet-mediated COVID-19 infections (26), masks with relatively low efficacy for personal protection may nevertheless reduce the overall infections in a population (27). Based on a previous study on influenza aerosols (28), we constructed a semi-quantitative model to show that mask-wearing reduces $\beta_{\text{eff}}$ and hence $R_0$ by a factor $(1 - e \cdot p_m)^2$, where $e$ is the efficacy of trapping viral particles inside the mask, and $p_m$ is the percentage of mask-wearing population (see SM). According to this model, mask-wearing at 96% alone could flatten an epidemic growing at a rate of 0.3/day by bringing down $R_0$ from its original value of 3.68 to 1. When combined with contact tracing (Fig. 3b), the two effects multiply. Figure 3c shows a heatmap of the reduced $R_0$ when contact tracing and isolation is completed within 4 days of infection. The solid yellow line indicates that the reduced $R_0$ reaches 1. For example, the combination of tracing of close contacts at 60% efficiency within 4 days and 70% of the general public wearing masks achieves the same purpose. This target line can be reached with lower percentages when close contacts can be found within 2 days of possible infection (red dashed line), but the numbers need to be higher when the time frame is relaxed to 7 days (green dashed line).

**Three-phase Epidemic Development**

From the time-series data of daily confirmed cases of COVID-19 obtained from the Johns Hopkins CSSE Repository (29), we identify three phases of COVID-19 epidemic development from different places in China following the Wuhan lockdown on January 23, 2020, with universal features at the beginning and end of regional outbreaks. These observations are interpreted within our model setting.

Phase I is characterized by exponential growth of the epidemic. In China, in the first week after the Wuhan lockdown on January 23, 2020, the number of daily confirmed cases continued to grow at a rate of approximately 0.3/day (Fig. 4a and 4b, dashed-black line), equivalent to an eight-fold increase per week. Data shows that most of the growth during this period is related to infections that can be traced to Hubei province, the epicenter of the initial outbreak. As we explain in the SM, the universal growth rate is set by latent, pre-symptomatic and symptomatic viral carriers from Hubei province, whose percentage among these travelers, while still low,
grows exponentially in that period. The fraction of local infections can be calculated using our model, and the result depends on the local value of $R_0$.

Phase II is a crossover phase where public policies on border control and to contain the disease spreading are taking effect. On a logarithmic scale, data from the most affected provinces apart from Hubei show consistent behavior. Close examination, however, reveals sporadic outbreaks in local communities that vary from province to province. Well-known examples include prison cases in Hubei, Shandong and Zhejiang provinces (30). Overall, under the swift and forceful implementation of contact tracing, isolation and social distancing policies, turnaround of the epidemic in provinces other than Hubei was reached in about three weeks after the regional lockdown. In Fig. 4b and the supplementary Fig. S5, we present simulation results using our model, assuming a linear decrease of $R_0$ from a local value of 2.0 to zero over a period $T$, which indeed reproduces the bell-shaped curve as seen in Fig. 4. The more gradual change of $R_0$ assumed in our simulations can be interpreted as due to the progressive government policies including additional lockdowns, which took place from February 4-10 (31, 32), as well as the time needed for these policies to take effect in communities that experienced new outbreaks.

Phase III, or the final descent, occurred when the intervention measures essentially eliminated new outbreaks. The few that surfaced were quickly identified and contained. Within our model, the newly confirmed cases in this period are identified with the shrinking number of individuals moving from the latent to the symptomatic phase, as one moves along the time axis in Fig. 2b. Strikingly, the observed decay rate in this phase reached the maximum value of 0.32/day, including data from Hubei province shown in Fig. 4a. This observation indicates that the infected cases were isolated at extremely high efficiency.

We now examine the situation elsewhere in the world. Exponential growth with a daily growth rate of around 0.3 is seen in the latest epidemic data from a number of countries (Fig. 4c). While these outbreaks were initially seeded by imported cases, they are now largely driven by local infections. Under successful interventions, a few countries have transitioned to the crossover phase observed in China in February (Fig. 4d). The government of Italy imposed a national quarantine on March 9 (33), after which growth in the number of newly confirmed cases slowed down (29). On the other hand, South Korea adopted very aggressive contact tracing and testing policies (34, 35), enabling the country to bring the initially rampaging outbreak to a much more manageable level at $R_0 \approx 1$ (Fig. 4d, stars). These countries are now facing the challenge of dealing with import driven growth, which can be analyzed using our model (see SM).

**Conclusions**

We have developed a simple yet powerful model to describe the spread of COVID-19 infection, which can be applied to communal outbreaks. The effect of intervention measures in a regional or national setting, such as border closing, social distancing, contact tracing, testing, quarantining and mask wearing, can be readily incorporated
into the model for quantitative assessment and prediction. In the ongoing battle in many countries, further reduction of the still positive growth rate requires quantitative evaluation of the proposed measures. While the data used to construct the reproduction rate function given by Eq. (1) will continue to be updated, our current predictions should already be informative to decision-makers. As the pandemic is fueled by transmission in the fastest growing community, reaching out to all sectors of society will pose the biggest challenge. In this respect, comprehensive monitoring of the pandemic development will be essential.

An important issue not treated explicitly in this work is the role played by asymptomatic carriers of the virus, i.e., those who never exhibit severe symptoms. We have made the implicit but plausible assumption that the reproduction rate function for this group of infected individuals is weaker or the same as the one given by Eq. (1), in which case their contribution is slaved to the group captured by Eq. (3), without altering the main structure of our model. We would like to emphasize that transmission by both pre-symptomatic and asymptomatic viral carriers can be much reduced under strict enforcement of social distancing and staying at home regulations in combination with wearing masks in public places, particularly when available resources and infrastructure do not allow for robust implementation of contact tracing and testing within the required time frames.

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Conflict of Interest Statement

Authors declare no conflict of interest.

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Figure 1. Transmission of COVID-19 during disease progression and intervention. COVID-19 disease progression and transmission: A person infected at time $t = 0$ first goes through a non-infectious latent phase (L) until $t_L$, marking the start of the infectious period. The infectious period consists of two phases, pre-symptomatic (A) and symptomatic (S). In phase A, the person is infectious without symptoms, during which the virus can be spread through pre-symptomatic transmission (orange dashed arrow). At the symptom onset time $t_O$, the person enters the S phase and infects others with symptomatic transmission (blue dashed arrow). The infectiousness of the person peaks around the symptom onset time. The basic reproduction number $R_0$ is split into $R_A$ and $R_S$, given by areas underneath the curve on either side of the symptom onset point, respectively. Interventions to limit transmission: contact tracing brings an infected person out of the transmission cycle at the point of isolation, while testing is effective only when the person has developed high viral load and is already in the infectious period.
Figure 2. Data and model predictions. a, The symptom onset time distribution. Two data sets from previous studies are shown (hollow circles (23) and squares (II)). The mean values and standard deviations are given in the legend. The distribution of the union of the two datasets is shown in solid circles. The red dashed line gives a reference exponential function shown in the legend. b, Probabilities for an infected individual being in each of the three phases at day $t$ after infection. The red curve indicates the boundary between the L+A and S phases. The probabilities that an individual is one day or two days into the S phase can be obtained from respective areas bounded by dashed and dashed-dotted curves, respectively. c, Percentage of the infected population in each phase when the epidemic is growing at a rate $\lambda$. The red curve indicates the boundary between the L+A and S phases. The percentages of the population one day or two days into the S phase are indicated by dashed and dashed-dotted lines, respectively. d, The relationship between the growth rate $\lambda$ and basic reproduction number $R_0$. At $\lambda = 0.3$, $R_0$ is 3.68.
Days after infection

Testing @ day 2
Testing @ day 3
Testing @ day 4

a

Probability of transmission

Testing @ day 2
Testing @ day 3
Testing @ day 4

0.15

Without testing
With testing

Days after infection

b

Traced/tested date (after infection)

Contact tracing
Testing (no report delay)
Testing (one day delay)

$R_0 = 3.88$ at $\lambda = 0.3$/day
80% of original $R_0$

Reduced reproduction number

Tracing and isolation within 4 days after infection

Probability of transmission

Percentage of traced contacts

Tracing and isolation within 4 days after infection

Percentage of mask-wearing population

Reduced reproduction number
Figure 3. Evaluation of Intervention Measures. **a,** Transmission probability (each day) from our model (black lines) and its revised values (unnormalized) under one-time testing (red dashed lines) performed on day 2 (top), day 3 (middle) and day 4 (bottom) after infection. The basic reproduction number $R_0$ is given by the area under the curve in each case. **b,** Reduction of the basic reproduction number $R_0$ against intervention time, calculated from the day of infection. Results are given for contact tracing and isolation (black line), and testing with 0 or 1 day reporting delay (red, blue curves), respectively. The value of $R_0$ at 3.68 corresponds to a growth rate $\lambda = 0.3$. Time is measured in days. **c,** Reduction of basic reproduction number $R_0$ under the combined measures of contact tracing and mask-wearing. The heatmap gives the reduced $R_0$ when close contacts are traced and isolated within 4 days after suspected infection, assuming a basal value of 3.68. The solid yellow line marks the percentages required to flatten the epidemic growth. The red dashed line and the green dashed line map out the percentages when the time frame for contact tracing is reduced to 2 days or relaxed to 7 days, respectively.
Figure 4. The COVID-19 epidemic development in various countries and regions. Daily confirmed cases in China and other affected countries since the Wuhan lockdown on January 23, 2020. a, Hubei province. The three phases of the epidemic development are marked in color: exponential growth (red), crossover
(yellow), and descent phase (green). Early exponential growth reached a rate $\lambda$ at approximately 0.3/day (left dashed line). Growth slowed and entered the crossover phase in the middle of the second week and reached the third phase nearly four weeks later. The final descent that began in the beginning of March is characterized by $\lambda = -0.32$/day (right dashed line). The incubation period distribution is shown in red circles to compare with the exponential decay. 

b, China (excluding Hubei province). The epidemic development in the main affected provinces followed a nearly identical three-phase pattern. Also shown is the model-predicted evolution of the number of daily confirmed cases (solid line), with details given in SM. The increase in the number of newly confirmed cases since the beginning of March is due to imported cases (white region). Data for the Diamond Princess cruise ship is included for comparison. 

c, Countries in phase I. The epidemic progress in these countries is still in the exponential growth phase with a daily rate $\lambda$ of around 0.3 (dashed line). 

d, Countries entering or in the middle of phase II. Italy, South Korea, and Switzerland have reached zero or negative growth in daily confirmed cases, while data from Iran indicates a slowing down of the exponential growth.
Supplementary Materials

Calibrated Intervention and Containment of the COVID-19 Pandemic

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In this Supplementary Material, a stochastic model of the infected population in a given communal outbreak is constructed and analyzed. The model is set up around the population dynamics of pre-symptomatic carriers. The size of the other infected groups can be inferred through simple mathematical equations.

1 The Mathematical Model

1.1 The governing equation

The basic structure of our model follows Fig. 1 in the Main Text, with model parameters defined in Fig. S1. The disease progression parameters $\alpha_A$ and $\alpha_L(t_L)$ are taken to be universal, while the transmission rates $\beta_A$ and $\beta_S(t_S)$ may vary significantly from community to community.

![Diagram](Image)

**Figure S1:** A stochastic model for disease progression and transmission. Disease progression of an infected individual is assumed to be described by a renewal process following the sequence of latent (L), pre-symptomatic (A) and symptomatic (S) phases. The transition rate from L to A is given by $\alpha_L(t_L)$ and depends on the duration $t_L$ of the latent phase. The transition from A to S is Poisson at a constant rate $\alpha_A$. Both A and S are infectious, with transmission rates to reproduce secondary cases at $\beta_A$ and $\beta_S(t_S)$, respectively. The latter is a function of $t_S$, the number of days since symptom onset.

We now consider the groups of infected individuals in L, A and S in a large population, using the same symbol to denote their size. The generation rate of L at time $t$ is given by,

$$J_L(t) = \beta_A A(t) + \int_{-\infty}^{t} \beta_S(t - t_2) dS(t_2).$$

1
Those “newly infected” eventually make their way to A. The flux to A due to a group infected at a time \( t_1 < t \) is given by,

\[
dJ_A(t) = \alpha_L (t - t_1) q_L (t - t_1) J_L(t_1) dt_1,
\]

where

\[
q_L(t) = e^{-\int_0^t \alpha_L(t_1) dt_1}
\]

is the probability that an individual infected at \( t = 0 \) remains in the latent phase \( L \). Adding up contributions from all such groups, we obtain

\[
J_A(t) = \int_{-\infty}^t \alpha_L (t - t_1) q_L (t - t_1) J_L(t_1) dt_1.
\]

Note that in our model, \( dS(t) = \alpha_A A(t) dt \), we have then

\[
J_A(t) = \int_{-\infty}^t \beta_A \alpha_L (t - t_1) q_L (t - t_1) A(t_1) dt_1
+ \int_{-\infty}^t \alpha_L (t - t_1) q_L (t - t_1) dt_1 \int_{-\infty}^{t_1} \beta_S (t_1 - t_2) \alpha_A A(t_2) dt_2
= \int_{-\infty}^t K(t - t_1) A(t_1) dt_1.
\]

Here the kernel function is given by,

\[
K(t) = \beta_A \alpha_L(t) q_L(t) + \alpha_A \int_0^t \alpha_L (t - t_1) q_L (t - t_1) \beta_S (t_1) dt_1. \tag{S1}
\]

The final equation for \( A \) takes the form,

\[
\dot{A} = -\alpha_A A + \int_{-\infty}^t K(t - t_1) A(t_1) dt_1. \tag{S2}
\]

1.2 Kernel function from observed symptom onset time distribution

As we show below, a major advantage of our model is that the kernel function in Eq. (S2) can be determined from the statistics of the time interval between infection and symptom onset \( \tau_O = \tau_L + \tau_A \), directly observable from clinical case studies. Denoting by \( p_O(t) \) the probability distribution function of \( \tau_O \). The probabilities for an individual infected at \( \tau = 0 \) to be in one of the three phases are given in Table S1.

In terms of Laplace transforms defined by \( \tilde{f}(\lambda) = \int_0^\infty f(t)e^{-\lambda t} dt \), we have

\[
\tilde{q}_L(\lambda) = \frac{1}{\lambda} \left( 1 - \frac{1}{\alpha_A} + \frac{1}{\lambda} \right) \tilde{p}_O(\lambda). \tag{S3}
\]

The Laplace transform of Eq. (S1) takes the form,

\[
\tilde{K}(\lambda) = \int_0^\infty K(t)e^{-\lambda t} dt = [\beta_A + \alpha_A \tilde{\beta}_S(\lambda)] \left[ 1 - \lambda \tilde{q}_L(\lambda) \right].
\]

With the help of Eq. (S3), we obtain

\[
\tilde{K}(\lambda) = [\beta_A + \alpha_A \tilde{\beta}_S(\lambda)] \left( 1 + \frac{\lambda}{\alpha_A} \right) \tilde{p}_O(\lambda). \tag{S4}
\]

Hence the dynamics of the disease transmission can be formulated in terms of the symptom onset time distribution.
Table S1: Probabilities an individual infected at $t = 0$ is in each of the disease phases at time $t$.

| Phase               | Probability | Expression                                      |
|---------------------|-------------|-------------------------------------------------|
| Latent              | $q_L(t)$    | $1 - \alpha_A^{-1} p_O(t) - \int_0^t p_O(t_1) \, dt_1$ |
| Pre-symptomatic     | $q_A(t)$    | $\alpha_A^{-1} p_O(t)$                           |
| Infectious w/o      |             |                                                  |
| Symptomatic         | $q_S(t)$    | $\int_0^t p_O(t_1) \, dt_1$                      |

1.3 Mean reproduction rate and $R_0$

Equation (S2) can be alternatively formulated in terms of the mean reproduction rate of an individual infected at $t = 0$. In our current setting,

$$r(t) = \beta_A \int_0^t q_L(t_1) \alpha_L(t_1) e^{-\alpha_A(t-t_1)} \, dt_1$$

$$+ \int_0^t q_L(t_1) \alpha_L(t_1) \, dt_1 \int_t^1 \beta_S(t-t_2) \alpha_A e^{-\alpha_A(t-t_2)} \, dt_2$$

$$= \int_0^t K(t_1) e^{-\alpha_A(t-t_1)} \, dt_1,$$

where the last step is written by comparing with the expression for $J_A(t)$.

The basic reproduction number is given by

$$R_0 = \int_0^\infty r(t) \, dt = \int_0^\infty \beta_A t e^{-\alpha_A t} \alpha_A dt + \int_0^\infty \beta_S(t) dt = \frac{\beta_{eff}}{\alpha_A}. \quad (S6)$$

Here

$$\beta_{eff} = \beta_A + \alpha_A \beta_S(0) \quad (S7)$$

is a key model parameter that sets the overall transmission speed of our model.

1.4 Exponential growth/decay

The self-sustained growth rate $\lambda$ of an outbreak can be obtained by seeking a solution $A(t) = e^{\lambda t}$ to Eq. (S2). Simple algebra gives

$$\lambda = -\alpha_A + \tilde{K}(\lambda). \quad (S8)$$

Combining Eq. (S4) with Eq. (S8), we obtain,

$$\begin{bmatrix} \beta_A + \alpha_A \beta_S(\lambda) \end{bmatrix} \left(1 + \frac{\lambda}{\alpha_A}\right) \tilde{p}_O(\lambda) = \lambda + \alpha_A$$

or

$$\begin{bmatrix} \beta_A + \alpha_A \beta_S(\lambda) \end{bmatrix} \tilde{p}_O(\lambda) = \alpha_A. \quad (S9)$$

In the epidemiological literature, it is customary to express the growth rate $\lambda$ in terms of $R_0$. From Eqs. (S6) and (S9), we obtain,

$$R_0 = \frac{1}{\tilde{p}_O(\lambda)} \frac{\beta_A + \alpha_A \beta_S(0)}{\beta_A + \alpha_A \beta_S(\lambda)} \quad (S10)$$

At $\lambda = 0$, $R_0 = 1$, as required, independent of the model parameters.

Equation (S10) expresses the fundamental mechanism for epidemic growth, i.e., the basic reproduction number of infected individuals drives the growth rate of the epidemic. The Laplace transform of the symptom onset time plays a pivotal role in our model. One immediate result from Eq. (S10) is that $R_0 = 0$ is at the pole of $\tilde{p}_O(\lambda)$. For $p_O(t) \sim e^{-\lambda t}$, the pole is at $\lambda = -\lambda_O$, which yields the rate of decay when transmission stops completely.
Table S2: Percentage of the infected in each group when the whole population grows at a rate $\lambda$.

| Phase                  | Probability | Expression                                      |
|------------------------|-------------|-------------------------------------------------|
| Latent                 | $Q_L(\lambda)$ | $1 - \tilde{p}_O(\lambda) - \frac{\lambda}{\alpha_A} \tilde{p}_O(\lambda)$ |
| Pre-symptomatic Infec- | $Q_A(\lambda)$ | $\frac{\lambda}{\alpha_A} \tilde{p}_O(\lambda)$ |
| tions w/o symptom      |             |                                                 |
| Symptomatic            | $q_S(\lambda)$ | $\tilde{p}_O(\lambda)$                        |

1.5 Generation time interval

Wallinger and Lipsitch [1] proposed a general equation between and based on the normalized “generation interval distribution”,

$$g(t) = r(t)/R_0.$$  \hfill (S11)

At the observed population growth rate $\lambda$, each individual produces $R_0(\lambda)$ offspring. Consequently,

$$\tilde{g}(\lambda) = \frac{\tilde{r}(\lambda)}{R_0} = \frac{1}{R_0},$$

known as the Lotka–Euler estimating equation. This equation is equivalent to (S10).

1.6 Percentage of subpopulations during exponential growth

Let $J_L(t) = J_L(0)e^{\lambda t}$ be the flux of newly infected individuals. The population size in each phase can be expressed as Laplace transforms of expressions in Table S1,

$$L(t) = \int_{-\infty}^{t} q_L(t - t_1) J_L(t_1) \, dt_1 = \left[ \frac{1}{\lambda} - \left( \frac{1}{\alpha_A} + \frac{1}{\lambda} \right) \tilde{p}_O(\lambda) \right] J_L(t),$$

$$A(t) = \int_{-\infty}^{t} q_A(t - t_1) J_L(t_1) \, dt_1 = \frac{1}{\alpha_A} \tilde{p}_O(\lambda) J_L(t),$$

$$S(t) = \int_{-\infty}^{t} q_S(t - t_1) J_L(t_1) \, dt_1 = \frac{1}{\lambda} \tilde{p}_O(\lambda) J_L(t).$$ \hfill (S12)

For easy reference, the percentages of subpopulations are collected in Table S2.
2 Simplifying Approximations

The expressions above that relate various quantities to \( p_O(t) \) are mostly expressed in Laplace transforms, which are inconvenient to use. Here we consider simplifying approximations which are handler when it comes to making analytical predictions.

2.1 Mapping symptom onset time distribution to generation time interval distribution

Under good self-quarantine practice, or when the public health and medical resources are not overstretched, symptomatic transmission is limited to the first one or two days after symptom onset. In such a scenario, \( \tilde{\beta}_s(\lambda) \) has a weak dependence on \( \lambda \):

\[
\tilde{\beta}_s(\lambda) = \int_0^\infty \beta_s(t)e^{-\lambda t}dt = \tilde{\beta}_s(0)(1 - \lambda \tau_s),
\]

(S13)

where \( \tilde{\beta}_s(0) \) is the area underneath the infectiousness curve on the symptomatic side of Fig. 1a in the Main Text, and

\[
\tau_s = \tilde{\beta}_s^{-1}(0)\int_0^\infty t\beta_s(t)dt
\]

is the width of the curve on the symptomatic side. Substituting (S13) into (S4), we obtain,

\[
\tilde{K}(\lambda) = \beta_{\text{eff}} \left( 1 + \frac{\lambda}{\sigma_A} \right) \tilde{p}_{O,\text{eff}}(\lambda),
\]

(S14)

where

\[
\tilde{p}_{O,\text{eff}}(\lambda) = (1 - \tau_{\text{eff}} \lambda) \tilde{p}_O(\lambda)
\]

(S15)

with

\[
\tau_{\text{eff}} = \tau_s \left( 1 - \frac{\beta_A}{\beta_{\text{eff}}} \right).
\]

Under this re-parameterisation, the integration kernel takes the explicit form,

\[
K(t) = \beta_{\text{eff}} \left[ p_{O,\text{eff}}(t) + \sigma_A^{-1} \frac{d}{dt} p_{O,\text{eff}}(t) \right].
\]

(S16)

From Eq. (S11), we obtain a very simple expression for the reproduction rate,

\[
\tau(t) = R_0 p_{O,\text{eff}}(t).
\]

(S17)

Hence, \( p_{O,\text{eff}}(t) \) is nothing but the generation time interval distribution \( g(t) \).

A few remarks with regard to the re-parameterized model are in order. As explained in the Main Text, \( \beta_{\text{eff}} \) incorporates contributions from both pre-symptomatic and symptomatic individuals (Fig. 1, Main Text). The mean value of the symptom onset time from the re-parameterized distribution is given by

\[
\langle t_O \rangle_{\text{eff}} = -\tilde{p}_{O,\text{eff}}(0) = \tau_{\text{eff}} + \tau_O,
\]

where \( \tau_O \) is the mean of the symptom onset time distribution in the original model. This is due to the incorporation of transmission by symptomatic patients.

**Important Note:** In the more general case, a model based on Eq. (S4) can be mapped to an effective model given by Eq. (S14) with a symptom onset time distribution given by the generation time interval \( g(t) \) whose Laplace transform is obtained from the data \( \tilde{p}_O(\lambda) \),

\[
\tilde{g}(\lambda) = \tilde{r}(\lambda) R_0 = \frac{\beta_A + \sigma_A \tilde{\beta}_s(\lambda)}{\beta_{\text{eff}}} \tilde{p}_O(\lambda).
\]

(S18)

In this effective model, an infected individual is infectious only in phase A, which may cause certain confusion when not explained properly. Nevertheless, it is mathematically fully equivalent to the original model in terms of Eq. (S2) for the epidemic dynamics that take care of transmission by both pre-symptomatic and symptomatic viral carriers.
2.2 A Markovian model

A number of modeling studies in the literature adopt a Markovian set up with $\alpha_L(t) = \text{const.}$ which is a special case of our more general non-Markovian approach. In particular,

$$\tilde{q}_L = \frac{1}{\alpha_L + \lambda}, \quad \tilde{p}_0 = \frac{\alpha_L}{\alpha_L + \lambda} \frac{\alpha_A}{\alpha_A + \lambda}. \quad (S19)$$

The onset time distribution in this case is given by

$$p_O(t) = \frac{\alpha_L \alpha_A}{\alpha_A - \alpha_L} \left[ e^{-\alpha_L t} - e^{-\alpha_A t} \right] = \frac{1}{\tau_L - \tau_A} \left( e^{-t/\tau_L} - e^{-t/\tau_A} \right),$$

with its mean and variance given by,

$$\tau_O = \langle t_O \rangle = \tau_L + \tau_A, \quad \sigma_O^2 = \langle t_O^2 \rangle - \langle t_O \rangle^2 = \tau_L^2 + \tau_A^2,$$

and

$$\frac{\sigma_O^2}{\tau_O^2} = \varepsilon^2 + (1 - \varepsilon)^2,$$

where $\tau_L = \varepsilon \tau_O$, $\tau_A = (1 - \varepsilon) \tau_O$. In the limit $\tau_L = \tau_A = 0$, $\varepsilon = 1/2$, $p_O(t) = \frac{1}{\tau_L} e^{-t/\tau_L}$.

The Laplace transform of the kernel function then takes the form,

$$\tilde{K}_M(\lambda) = \frac{\beta_A + \alpha_A \tilde{\beta}_S(\lambda)}{\alpha_L + \lambda} \approx \frac{\alpha_L}{\alpha_L + \lambda} \frac{\beta_{\text{eff}} (1 - \tau_{\text{eff}} \lambda)}{\alpha_{L,\text{eff}} + \lambda},$$

where $\alpha_{L,\text{eff}} = \alpha_L / (1 + \alpha_L \tau_{\text{eff}})$ gives the reduced effective rate to exit the latent phase, in agreement with Eq. (S15). Performing the inverse transform yields

$$K_M(t) \approx \beta_{\text{eff}} e^{-\alpha_{L,\text{eff}} t}. \quad (S20)$$

The re-parameterized model has the effective parameters $\tau_O = \alpha_{L,\text{eff}}^{-1} + \alpha_A^{-1}$ and $\varepsilon = \frac{\alpha_A}{\alpha_{L,\text{eff}} + \alpha_A}$, while transmission is assigned to phase A with an effective rate $\beta_{\text{eff}}$. Under the effective kernel function (S20), Eq. (S8) takes the form,

$$R_0 = \left( 1 + \frac{\lambda}{\alpha_{L,\text{eff}}} \right) \left( 1 + \frac{\lambda}{\alpha_A} \right),$$

i.e., a parabola with two nodes at $\lambda_L \approx -\alpha_{L,\text{eff}}$ and $\lambda_A \approx -\alpha_A$. 

6
3 Calibration of Model Parameters

3.1 Symptom onset time distribution from case studies

The incubation periods of individual patients before symptom onset were summarized in 2 preprints: 54 cases collected by Men et al. [2], and 105 cases collected by Xia et al. [3]. In total, we examined 159 cases with their incubation periods. For cases when the infection date can only be assigned to a time interval spanning more than one day, we simply assume equal probability for each day inside the interval. We then include all case counts to construct the histogram of symptom onset time, rendering the result shown in Fig. 2a of the Main Text. The mean onset time is 5.17 days, with a standard deviation of 2.93 days. The tail of the distribution can be well-fitted to an exponential decay function with a rate of $-0.32/day$.

3.2 Serial interval statistics

Du et al. [4] reported the statistics of the time lag in symptom onset between infector-infectee pairs in 468 confirmed serial infection cases, which we use to check against the symptom onset distribution function obtained above.

Figure S2: Predicted serial interval distribution vs. real data. The data collected by Du et al. [4] on the serial interval distribution is shown in black dots (all 468 pairs) and red squares (122 local infections). The solid line is our model prediction using the data for the incubation period as input. See text for details.

Let $t = 0$ be the time point when the infector X contracted the virus, the distribution of the transmission time $t_{X \rightarrow Y}$ from X to Y is $g(t)$.

We now consider the distribution of the difference in symptom onset time $t_{XY} = t_Y - t_X$ between X and Y. The distribution of $t_X$ is given by $p_0(t)$. The distribution of $t_Y = t_{X \rightarrow Y} + t_Y$ can also be expressed as a convolution of $g(t)$ and $p_0(t)$. According to Eq. (S18), $g(t) \approx p_0(t)$ when symptomatic transmission is limited to around the symptom onset time. In this case, all three quantities have the same distribution. Consequently, $\sigma_s = \frac{\sigma_X}{\sqrt{3}}$.

From Table S1 of Ref [2], we get SD value 2.74 days, in good agreement with the value 2.93 days from our analysis. Close examination of the data shows a discontinuity across the origin,
which may be attributed to the false assignment of infector and infectee in a pair. As noted by Du et al. [4], cluster infections (Such as intra-family transmission) could also yield a lower value for the serial interval on average, contributing to the discrepancy seen in the figure. Overall, the comparison lends strong support for the aggregated symptom onset time distribution obtained, as well as for our model assumptions.

3.3 Infectiousness around symptom onset and $\alpha_A$

Xia et al. [3] documented the infection date of 74 secondary cases in relation to the symptom onset date of the first generation. Data for the number of transmissions against days after symptom onset, which varies from individual to individual, are replotted in Fig. S3 with solid circles, ranging from 5 days before to 8 days after. Only 27% of the cases fall on or after the symptom onset date, while peak transmission happens two days before.

According to our model, the duration of the infectious phase A before symptom onset follows the Poisson distribution with a rate $\alpha_A$. Hence the left tail of the distribution is proportional to $e^{-\alpha_A(t_o-t)}$. Using the cumulative distribution (open circles) in Fig. S3, which is less sensitive to data fluctuations, we obtain a decay rate in the range $\alpha_A = 0.7$/day (solid line) to 0.82/day (dashed line). A value $\alpha_A = 0.75$/day is used in our numerical explorations. As seen in Tables S1 and S2, this parameter affects the size of the infectious A subpopulation. Since the actual reproduction rate is controlled by $R_0 = \beta_{eff}/\alpha_A$, the actual value of $\alpha_A$ is not important when the data is interpreted correctly. The outliers on the far right are not of concern in this work, as they are likely to be quarantined given the heightened attention on COVID-19.

![Figure S3: The number of secondary cases against the symptom onset date of the first generation cases. Data from Xia et al.[3]](image-url)
4 Model Exploration under Intervention

4.1 Testing and quarantining

Testing and quarantining of infected individuals is practiced in South Korea with great intensity. In the simplest scenario, a suspected individual undergoes testing of the COVID-19 infection. It takes a day or so for the test result to come back. If it is positive, the person will be quarantined and hence removed from the active infected population. Oral tests for the virus report only cases with a sufficiently high viral load. Therefore the test needs to be done around the time of symptom onset. However, by then the person may have already infected other people. Within our probabilistic framework, the efficiency of this procedure can be assessed from the reduction of the reproduction rate \( r(t) \).

Adopting the simplified model (S17), we can write, under testing,

\[
\tau_{\text{testing}}(t) = r(t) - \beta_{\text{eff}} \int_0^t dt_1 \eta_{\text{testing}}(t_1) q_A(t_1) e^{-\alpha_A(t-t_1-\tau_d)}.
\]  

(S21)

Here \( \eta_{\text{testing}} \) is the test protocol that gives the rate of testing per individual on day \( t \) since infection, and \( \tau_d \) is the delay in producing the report. To maximise reduction, the test time should be chosen to be around the peak of the probability in phase A:

\[
q_A(t) = \frac{1}{\alpha_A} p_0(t),
\]

which is around 4 days after the day of infection.

In the extreme case of testing on a particular day \( T \) with no reporting delay, we have

\[
\tau_{\text{testing}}(t) = \begin{cases} 
  r(t), & t < T, \\
  r(t) - q_T \alpha_A R_0 q_A(T) e^{-\alpha_A(t-T-\tau_d)}, & t > T.
\end{cases}
\]

(S22)

Here \( q_T \) is the percentage of close contacts of infected individuals being tested. Integrating the equation over time yields the reduction in \( R_0 \),

\[
\Delta R_0 = -R_0 q_A(T) e^{-\alpha_A \tau_d}.
\]

(S23)

The expression for \( q_A(T) \) can be found in Table S1.

4.2 Contact tracing

In contact tracing, all close contacts of a newly confirmed viral carrier are identified and quarantined soon after the contact took place, without testing. This procedure is more effective as it also covers those infected but still in the latent phase of their disease development. Thus \( r(t) \) is truncated on the day the contact is located, while the person could still be transmitting the disease before that time. The reduction of in this case is given by,

\[
\Delta R_0 = -q_c \int_0^\infty r(t) dt = -q_c R_0 [1 - q_S(T)].
\]

(S24)

Here \( q_c \) is the probability to locate the contact. Expression for \( q_S(T) \) is given in Table S1.

4.3 Mask wearing

Considering the dual-effects of mask-wearing in reducing both virus inhalation by susceptible individuals and exhalation by infectious individuals (including pre-symptomatic and asymptomatic groups), we calculated the reduction of \( \beta_{\text{eff}} \) against mask efficacy and the percentage of the population wearing masks under a simplifying approximation.
We assumed that there are COVID-19 positive (P) and negative (N) individuals. \( p_m \) is the percentage of the population that wear masks: \( p_{mP} \) for positive individuals and \( p_{mN} \) for negative individuals. Here, \( e \) denotes the efficacy of masks measured by the percentage of virus trapped by the mask: from inhalation (\( e_{in} \), important for COVID-19 positive individuals) and exhalation (\( e_{ex} \), important for COVID-19 negative individuals). In the absence of masks, the rate of transmission by a positive individual contacting a negative individual is \( \beta \).

**Table S3:** 4 types of encounters between a positive and a negative individual and reduction of the transmission rate \( \beta \).

| Prob. of contact type | P wearing mask | N wearing mask | Chance of transmission |
|-----------------------|----------------|----------------|------------------------|
| \( p_{mP} \cdot p_{mN} \) | Yes            | Yes            | \( \beta(1 - e_{ex})(1 - e_{in}) \) |
| \( p_{mP}(1 - p_{mN}) \) | Yes            | No             | \( \beta(1 - e_{ex}) \) |
| \( (1 - p_{mP})p_{mN} \) | No             | Yes            | \( \beta(1 - e_{in}) \) |
| \( (1 - p_{mP})(1 - p_{mN}) \) | No             | No             | \( \beta \) |

Therefore, the averaged chance of transmission is:

\[
\beta_{\text{mask}} = \beta(1 - e_{in} \cdot p_{mN}) \cdot (1 - e_{ex} \cdot p_{mP}).
\]

Under the totally symmetric assumption \( e_{ex} = e_{in} = e \), \( p_{mN} = p_{mP} = p_m \), the result becomes:

\[
\beta_{\text{mask}} = \beta(1 - e \cdot p_m)^2. \tag{S25}
\]

Note that this equation can be applied separately to pre-symptomatic and symptomatic transmission, with their own \( p_m \). In the numerical examples presented below and in the Main Text, we used the same \( p_m \) for both types of transmission. In the more general case, the reduction of \( \beta_{\text{eff}} \) can be calculated when contributions of the two types to the total are known.

**Figure S4:** Estimation on the impact of mask-wearing on the transmission rate. a. Schematic representation on the dual-effects of masks in reducing \( \beta \). b. Relationship between the factor multiplying \( \beta \) (heatmap color) with the mask efficacy (\( x \)-axis) and the fraction of the population wearing masks (\( y \)-axis). Black lines show the contours for reducing \( \beta \) to 0.75, 0.5, 0.27 of its original value.

The above discussion shows, in a semi-quantitative way, that mask-wearing brings benefits to one-self if not infected but more importantly to others. Even with masks at a moderate efficacy of 50%, reduction of the transmission rate can be substantial when practiced by the whole population.
Previous research on influenza suggested that surgical mask reduces 70% of the viral aerosol shedding [5]. Also, WHO suggested that respiratory droplets (> 5 ~ 10 \( \mu m \) in diameter) and contacts are the primary routes for COVID-19 to transmit between people [6]. Surgical mask reduces more than 90% of droplets in this size range [7]. However, general public may not be able to fully comply with the usage guidance of surgical masks. Therefore, in generating results in the Main Text, we take a simpler assumption that the efficacy of surgical masks is at 50%.

4.4 Solution with imported cases

Border control measures can effectively stop imported cases of viral carriers. To examine the time needed for their effect to take place, let us first consider growth driven by imported cases when unchecked. Under a daily flux \( \dot{J}_{\text{ext}}(t) \) of imported cases, Eq. (S2) is modified to,

\[
\dot{A} = -\alpha_A A + \int_{-\infty}^{t} K(t - t_1) A(t_1) \, dt_1 + \dot{J}_{\text{ext}}(t). 
\]

(S26)

Consider a simple situation where the imported cases grow exponentially with a rate \( \lambda \), i.e. \( \dot{J}_{\text{ext}}(t) = \lambda e^{\lambda t} \). Let's seek an exponentially growing solution where the local population is driven by the imported cases,

\[
A(t) = A_{\text{all}} e^{\lambda_A t},
\]

\[
(\lambda_I + \alpha_A) A_{\text{all}} = A_{\text{all}} K(\lambda_I) + J_I,
\]

\[
A_{\text{all}} = \frac{J_I}{\lambda_I + \alpha_A - K(\lambda_I)}.
\]

The fraction of local infections after the initial transient is given by,

\[
\frac{A_{\text{local}}}{A_{\text{all}}} = \frac{K(\lambda_I)}{\lambda_I + \alpha_A}.
\]

(S27)

In many cases, the ratio of imported and local infections is known. Equation (S27) can then be used to calibrate the level of local transmission when the imported cases grow exponentially at a rate greater than the one given in Table S2 for local outbreaks.

4.5 Crossover behavior under an evolving social distancing practice

We consider here a situation where the disease transmission rate per infected individual in a given community gradually weakens according to the schedule,

\[
\eta(t) = 1 - (1 - \eta_I) \frac{t - t_0}{T}, \quad t_0 < t < t_0 + T.
\]

Here \( t_0 \) is the starting date and \( T \) is the duration of the change. After this period, \( \eta(t) = \eta_{\text{eq}} \). Equation (S2) then takes the form,

\[
\dot{A} = -\alpha_A A + \int_{-\infty}^{t} K(t - t_1) \eta(t_1) A(t_1) \, dt_1.
\]

(S28)

We used Eq. (S28) to simulate the bell-shaped epidemic development curves in China after the Wuhan lockdown (Figs. 4a and 4b, Main Text), taking \( \eta_I = 0 \). The starting time of the social distancing policy \( t_0 \) is chosen to be one week after the lockdown. The blue, red and black curves in Fig. S5 correspond to three different values of \( T \) given in the legend. The situation represented by blue and red curves is initially driven by imported cases whose number grows exponentially at a rate \( \lambda_0 = 0.2 \) prior to the lockdown, with an amplitude that decreases linearly and vanishes on the 10th day after the lockdown. No imported cases were introduced to generate the black curve. Taken at face value, our model can reproduce fast or slow crossovers seen in the data from various provinces of China. Further discussion can be found in the Main Text.
4.6 Staying at home

The situation on the cruise ship Diamond Princess is close to a sudden complete confinement of the passengers. Such a scenario is described by the schedule function

\[ \eta(t) = \begin{cases} 
1, & t < t_0 \\
\eta_1, & t > t_0 
\end{cases} \]

at \( \eta_1 = 0 \). Under the Markovian assumption introduced above, the change over from exponential growth to exponential decay can be solved analytically.

Without loss of generality, we may define our time such that \( t_0 = 0 \). Take \( A(t) = \lambda(t) e^{\lambda_0 t} \) for \( t < t_0 = 0 \), we may rewrite Eq. (S28) as,

\[
\dot{A} = -\alpha_A A + \beta_0 \int_{-\infty}^{0} \alpha_L(t-t_1) q_L(t-t_1) A(t) e^{\lambda_0 t_1} \, dt_1 + \beta_1 \int_{0}^{t} \alpha_L(t-t_1) q_L(t-t_1) A(t_1) \, dt_1. 
\]

(S29)

Let

\[ S(t) = \beta_0 A_0 \int_{-\infty}^{0} \alpha_L(t-t_1) q_L(t-t_1) e^{\lambda_0 t_1} \, dt_1, \]

Eq. (S29) can be rewritten as,

\[
\dot{A} = -\alpha_A A + S(t) + \beta_1 \int_{0}^{t} \alpha_L(t-t_1) q_L(t-t_1) A(t_1) \, dt_1. 
\]

(S30)

Under the Markovian assumption (S19), \( \alpha_L(t) q_L(t) = \alpha_L e^{-\alpha_L t} \). Consequently,

\[ S(t) = \beta_0 A_0 \frac{\alpha_L}{\alpha_L + \lambda_0} e^{-\alpha_L t}. \]
Performing the Laplace transform of Eq. (S30), we obtain,
\[
\tilde{A}(\lambda) = \frac{\tilde{S}(\lambda) + A_0}{\alpha_A + \lambda - \tilde{K}_1(\lambda)},
\]
(S31)
where
\[
\tilde{S}(\lambda) = \beta_0 A_0 \frac{\alpha_L}{\alpha_L + \lambda_0} \frac{1}{\alpha_L + \lambda}.
\]
The kernel function under the new policy is given by
\[
\tilde{K}_1(\lambda) = \frac{\alpha_L}{\alpha_L + \lambda} \beta_1.
\]
Carrying out inverse transform of Eq. (S31), we obtain
\[
A(t) = A_0 \left( B_+ e^{\lambda_+ t} + B_- e^{\lambda_- t} \right).
\]
(S32)
Here,
\[
\lambda_{\pm} = -\frac{(\alpha_L + \alpha_A) \pm \sqrt{(\alpha_L - \alpha_A)^2 + 4R_1\alpha_A\alpha_L}}{2},
\]
\[
B_+ = \frac{\beta_0\alpha_L}{(\alpha_L + \lambda_0)(\lambda_+ - \lambda_-)} \frac{\lambda_+ + \alpha_L}{\lambda_+ - \lambda_-},
\]
\[
B_- = -\frac{\beta_0\alpha_L}{(\alpha_L + \lambda_0)(\lambda_+ - \lambda_-)} \frac{\lambda_- + \alpha_L}{\lambda_+ - \lambda_-},
\]
with \( R_1 = \beta_1/\alpha_A \).
The solution is shown for several examples in Fig. S6. Note that the duration of the crossover is set by \( \alpha_L^{-1} \approx 3 \text{ days} \) for the parameters chosen.

Figure S6: Simulation of daily confirmed infections under a sudden reduction of \( \beta_{\text{eff}} \). The epidemic initially grows at a rate \( \lambda_0 = 0.3/\text{day} \) before the jump. Different curves correspond to different values of \( R_1 \) under the new policy. Here \( \alpha_L = 0.3/\text{day}, \alpha_A = 0.5/\text{day} \).
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