Supplementary Information for:

Managing COVID-19 spread with voluntary public-health measures: Sweden as a case study for pandemic control

Supplementary Notes:

Spread of COVID-19 in the hospital setting depends on three fundamental exposure routes: encounters with patients who are diagnosed or suspected COVID-19-positive, encounters with patients who are not suspected of being COVID19-positive yet still transmitting virus, and spread between healthcare staff (in breakrooms and other areas where PPE are not used). Available evidence suggests that appropriate personal protective equipment (PPE) can almost eliminate the first—in settings where PPE use is standard, the incidence of COVID-19 among healthcare workers is lower than in the surrounding general population [1, 2]. Preliminary data from at least once hospital in Sweden, however, suggest that the opposite may be true: serological survey data indicate over twice the seropositivity to SARS-CoV-2 compared to tests mailed to the general population during the same time period [3, 4]. These precise risks are difficult to quantify at this time and will vary from site to site, but overall hospitals can be assumed to be lower risk than the same assembly of people in other workplaces but non-zero.

Our models did not explicitly include congregate living facilities or home health care for older adults. Congregate living facilities have been a major source of spread in a number of countries. We note, however, that in Sweden, only 5% of adults over 70 are in such facilities (11% of adults over 80), while approximately twice that number receive home health care (8% of adults 65+ and 22% of adults 80+) [5]. The median size of such congregate living facilities in 2019 was 39 residents, with a median of 11 staff [6]. Infection-control practices and PPE use in both of these settings will also impact transmission. Government directives regarding PPE use in congregate living facilities in Sweden have been less stringent than in many other European countries; at this time there are not sufficient data available to rigorously parameterize spread in Swedish congregate living facilities.

The transmission model described below likely recapitulates household and workplace contacts reasonably well, as in a small household or workplace every individual has a chance of infecting every other individual. The simple distance-based gravity kernel used for community transmission is likely an oversimplification, and more discrete representations of contact networks in the community would improve fidelity at a substantial cost in complexity.

Supplementary Methods:

The functional forms for household, workplace, and community transmission are adopted from prior work by Ferguson and colleagues [7] and are briefly summarized as follows:

Infection of each susceptible individual is calculated via a Monte Carlo criterion at each (1-day) time step with probability 1 - e^{λ}, where λ is the sum of the household, workplace, and community transmission for each individual *i* as follows:

Household transmission for individual *i* in household h_i is calculated as:

$$\sum_{k \in h_i} I_k \beta_h \kappa(t - \tau_k) \rho_k [1 + C_k(\omega - 1)] / {n_i}^{\alpha}$$

where I_k is 1 if individual *k* is infectious, otherwise 0; β_h is a household transmission coefficient set to 1.1; κ describes infectiousness over time and is set to a log-normal distribution with mean of -0.72 and standard deviation of 1.8 as in previous work [7]; ρ_k is set to the identity function for an approximation of uniform infectiousness modulo case severity; C_k is 1 if individual *k*'s infection is severe and 0 otherwise, and ω is set to 2 such that severely affected individuals are twice as infectious as mildly infectious ones; n_i is the household size for individual *i*, and α is set to 0.8 as in previous work [7-9].

Workplace (or school) transmission for workplace *j* is calculated as:

$$\sum_{k \in j} I_k \beta_w \kappa(t - \tau_k) \rho_k [1 + C_k(\omega \psi_w(t - \tau_k) - 1)] / m_j$$

where β_w is a workplace transmission coefficient set to 0.627 for normal workplaces, 1.254 for schools, and 0.156 for hospitals [10]; ψ_w reflects illness-based absence from work and school and is set to 0.1 for preschools, 0.2 for primary schools, 0.25 for high schools and universities, and 0.5 for workplaces and hospitals as in previous work [7-9]; m_j is the number of individuals in workplace *j*.

Community transmission for individual *i* is calculated as:

$$\sum_{k} \frac{I_k \varsigma(i) \beta_c \kappa(t - \tau_k) \rho_k f(d_{i,k}) [1 + C_k(\omega - 1)]}{\sum_k f(d_{i,k})}$$

by summing over all individuals *k*, where β_c is set to 0.865; ς(*i*) depends on the age of individual *i* and is set to 0.1 for age less than 5, 0.25 for age 5-10 and over 75, 0.50 for age 10-15 and 70-75, 1.0 for age 20-65, and 0.75 for age 10-15 and 65-70; and $f(d_{i,k})$ is a gravity-based distance kernel depending on the distance between individuals *i* and *k* such that $f(d) = 1/(1 + (d/4\ km)^3)$ as previously used [7-9].

Infectious individuals are treated as follows: 33% are estimated to be asymptomatic or subclinical as previously estimated [7]; all infected individuals become infectious 4.6 days after exposure and symptomatic individuals develop symptoms 5.1 days after exposure as previously estimated [7, 11]; 50% of symptomatic individuals are estimated to have severe symptoms. Hospitalization and ICU admission need are estimated as in prior work, with age-dependent hospitalization probabilities for symptomatic individuals of 0.1% for

ages 0-9, 0.3% for 10-19, 1.2% for 20-29, 4.9% for 40-49, 10.2% for 50-59, 16.6% for 60-69, 24.3% for 70-79, and 27.3% for ≥ 80 as estimated by Verity and colleagues [12]; age-dependent ICU need probabilities for hospitalized individuals were taken as 5% for ages 0-39, 6.3% for 40-49, 12.2% for 50-59, 27.4% for 60-69, 43.2% for 70-79, and 70.9% for ≥ 80 as previously estimated [7, 12]. Overall ICU survival rates were set to 50%, and non-ICU patients had age-dependent mortality of 0.00161% for age 0-9, 0.00695% for 10-19, 0.0309% for 20-29, 0.0844% for 30-39, 0.161% for 40-49, 0.595% for 50-59, 1.93% for 60-69, 4.28% for 70-79, and 7.8% for ≥80 as estimated by Ferguson and colleagues [7]. Death was taken to occur after 10 days of symptoms; recovery of non-hospitalized individuals after 11 days of symptoms, of hospitalized individuals after 13 days of symptoms. Individuals in intensive care were transferred to standard hospital wards after 10 days and discharged/considered recovered 5 days later [10].

Transmissibility factors were determined based on fitting growth in cases or deaths in the date range Mar 21 to Apr 6 against Swedish or pan-European data as described in the main text. The predicted case and death curves from each value tested are plotted in Figure S6.

Supplementary Figures

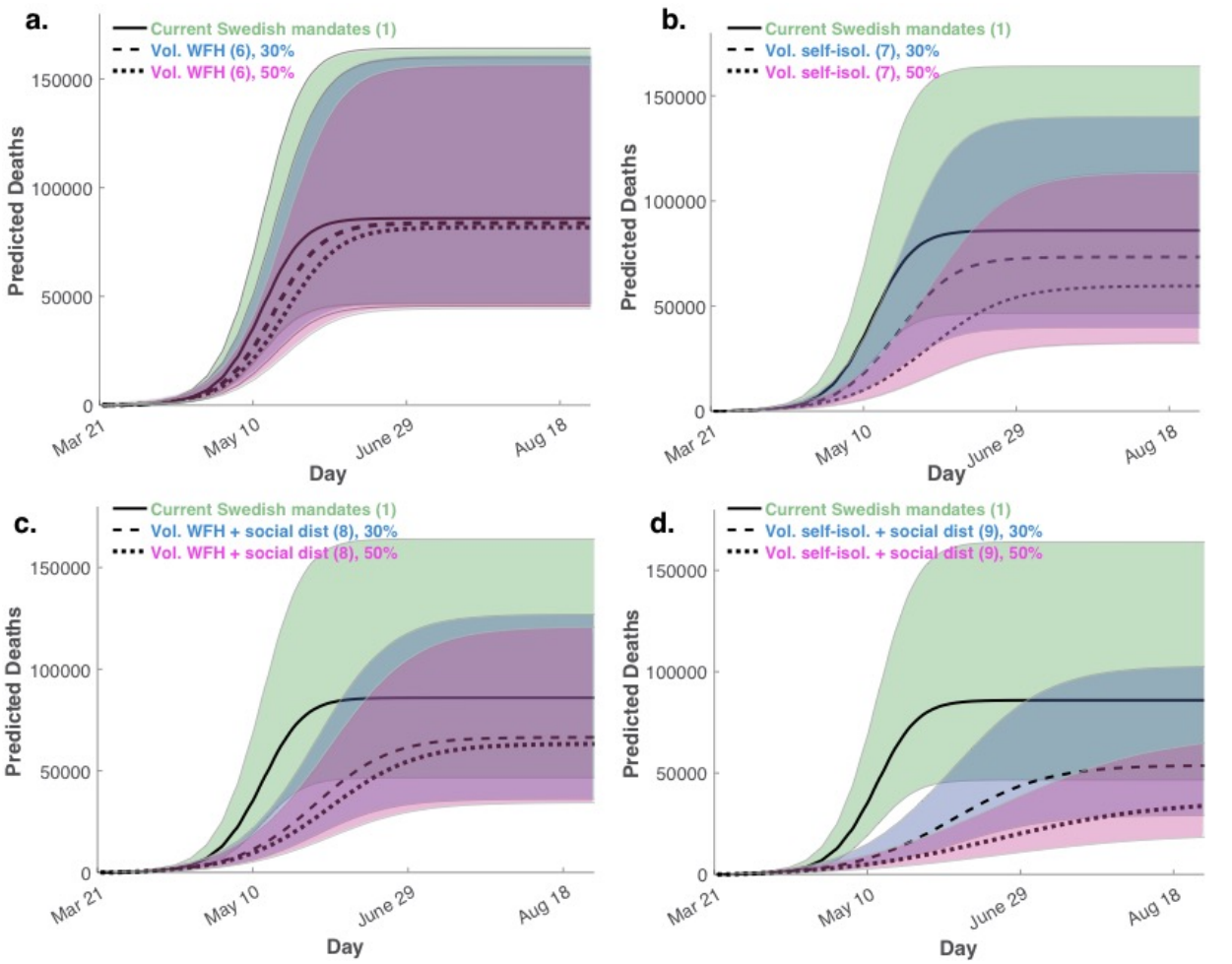

Figure S1. Confidence-interval analysis of predicted deaths with different voluntary-adherence strategies. Shaded regions show 95% confidence intervals on age-specific infection-fatality ratios propagated through each model. Although the uncertainties in computing infection-fatality ratios result in a large uncertainty for the absolute number of deaths predicted, the relative impact of each public-health intervention is only minimally affected. Statistically, this is a matched-sample calculation: each intervention i can be seen to produce $d_i(\mathbf{r}_j)$ predicted deaths for each sampled infection-fatality ratio $\mathbf{r}_j$ (vector over age quantiles) from the underlying distribution $\{\mathbf{r}\}$.

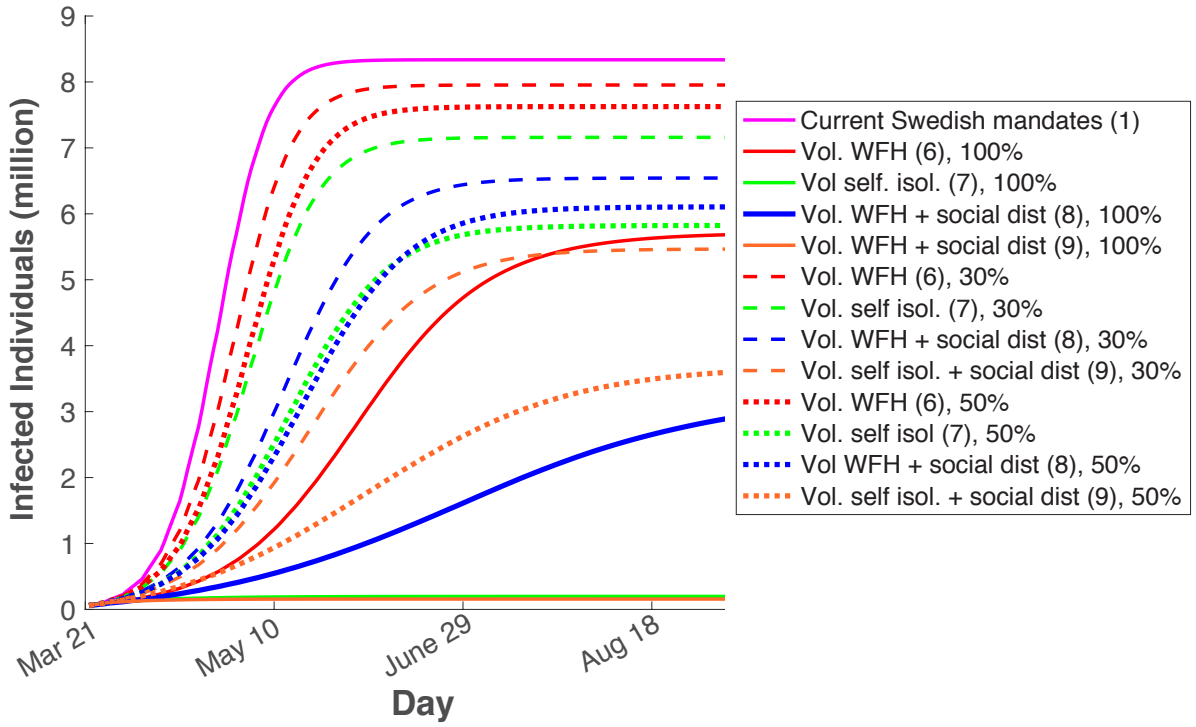

Figure S2. Predicted numbers of infections in Sweden with different voluntary-adherence strategies. The median number of cumulative infected individuals is plotted versus time for each of the mandates or voluntary-adherence regimes indicated. Numbers in parentheses match interventions listed in the Methods.

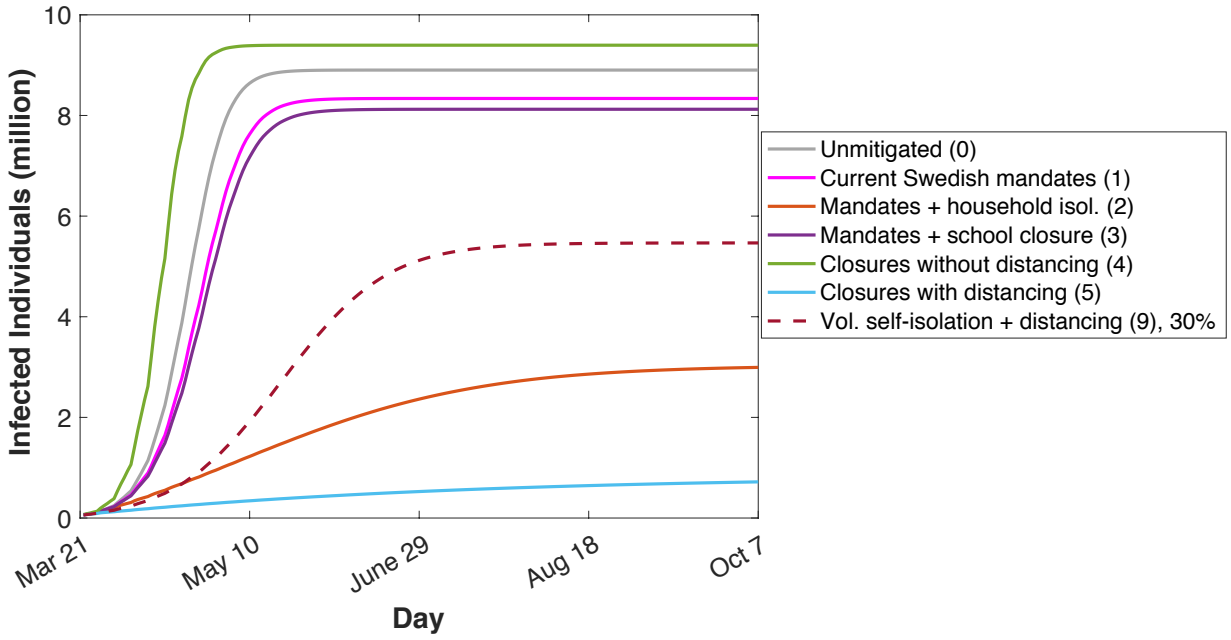

Figure S3. Predicted numbers of infections in Sweden with different public-health mandates. The cumulative number of infected individuals is plotted as a function of time for each indicated public-health mandate. Data represent the median of 10 independent model runs.

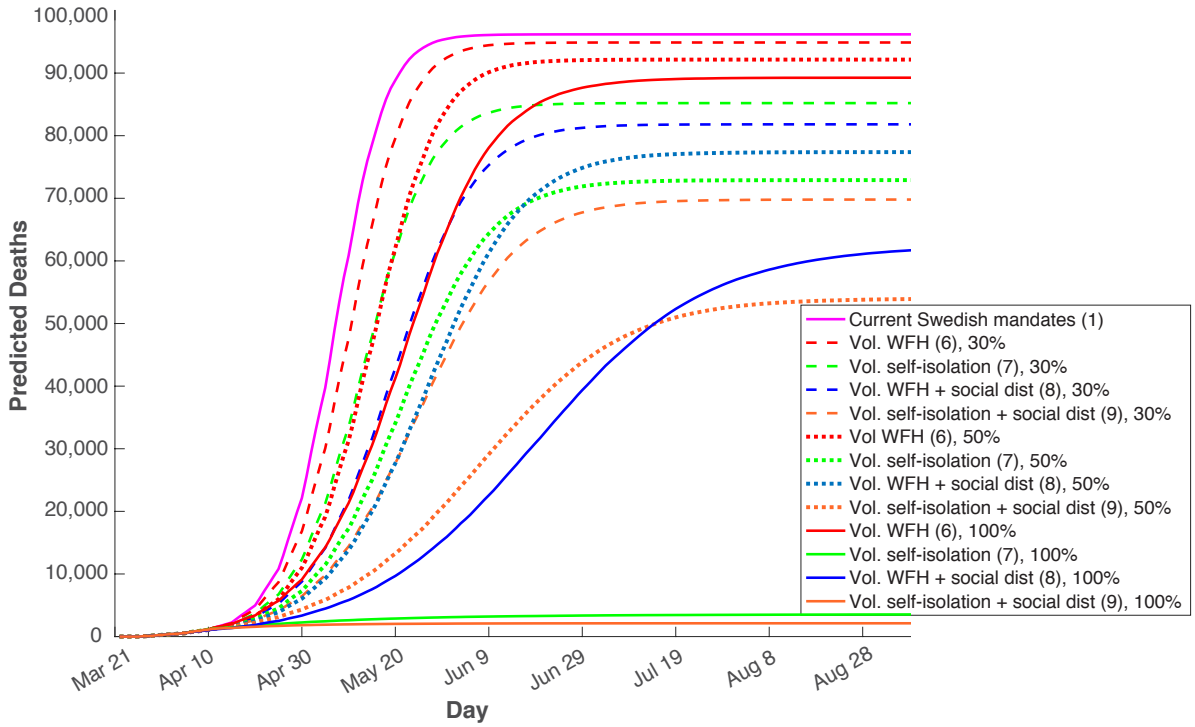

Figure S4. Predicted number of deaths with different voluntary-adherence strategies and alternate infectiousness parameters. The median number of predicted deaths is plotted for each indicated voluntary-adherence strategy. Data are plotted assuming a 3-day doubling time instead of a 5-day doubling time. Compared to the analogous data for a 5-day doubling time in Fig. 1, the fraction of adherence required for effective pandemic control increases with increased transmissibility (shorter doubling time).

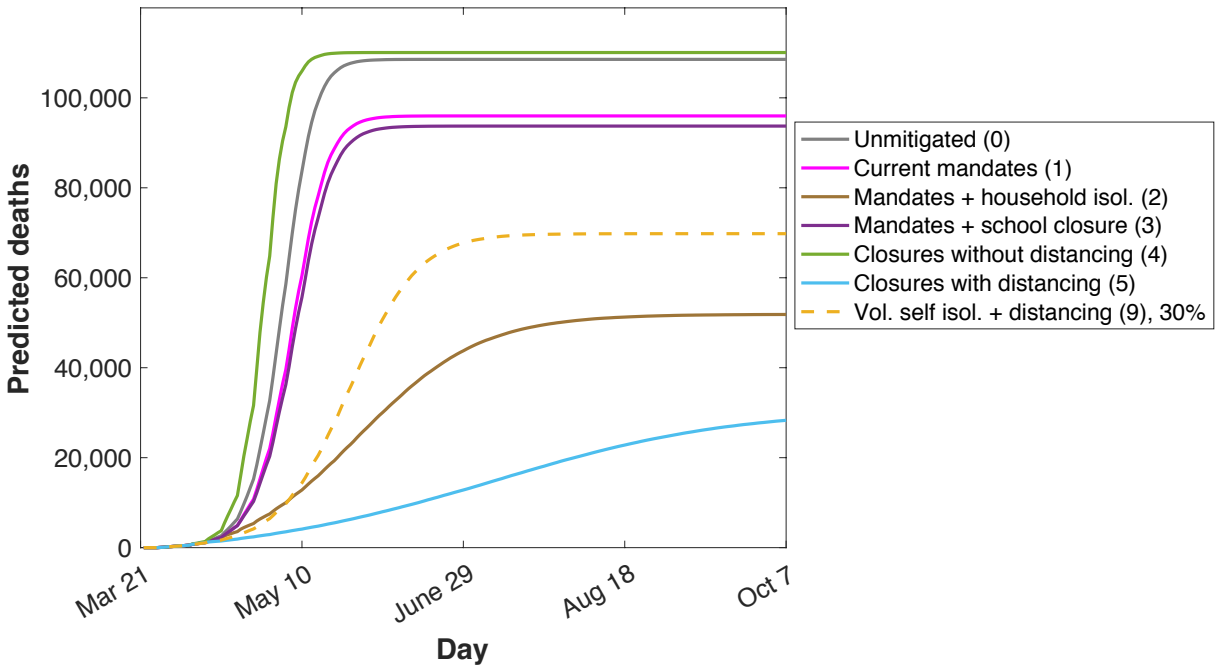

Figure S5. Predicted number of deaths with different public-health mandates and alternate infectiousness parameters. Data are plotted assuming a 3-day doubling time instead of a 5-day doubling time. Compared to the predictions for a 5-day doubling time, trends are similar but total deaths increased.

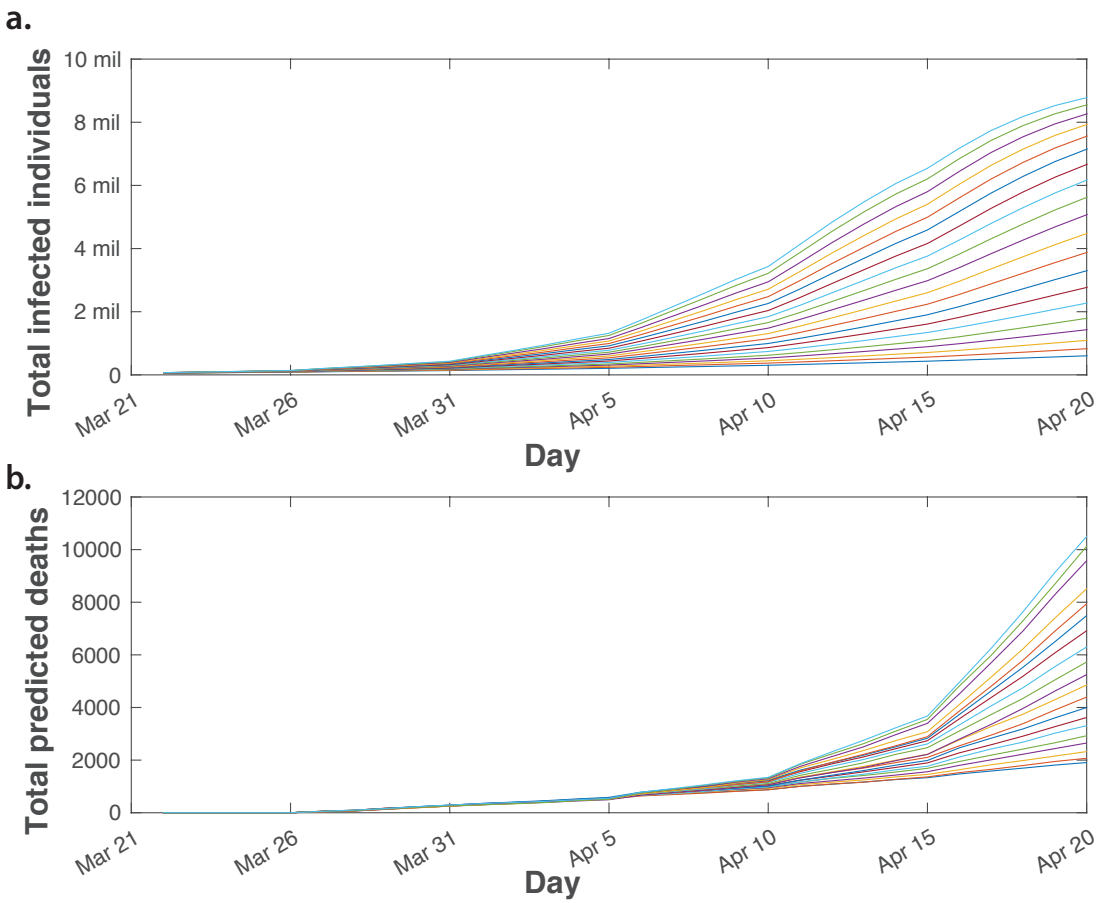

Figure S6. **Sensitivity testing and validation of transmissibility parameters**. Twenty values of the transmissibility parameter (range 1.0 to 2.9) were evaluated against growth in (a) cases and (b) deaths on the time range March 21 to April 6. Results from all twenty parameter values are plotted in ascending order. Parameter values were selected that yielded doubling times of approximately 5 days (main text) and 3 days (Figs. S4-S5).

Supplementary Tables

Country	Reported deaths per 100,000 population
Spain	58
Italy	52
United Kingdom	51
France	41
Sweden	**35**
Netherlands	32
Ireland	31
Portugal	12
Germany	9.4
Denmark	9.3
Austria	7.1
Romania	5.4
Finland	5.2
Slovenia	5.0
Estonia	4.7
Hungary	4.5
Norway	4.4
Czechia	2.8
Iceland	2.8
Poland	2.3
Croatia	2.3
Lithuania	1.9
Greece	1.5
Bulgaria	1.4
Latvia	1.0
Slovakia	0.5
Belgium	78*

Table S1. Deaths per capita in Sweden and other major European countries. Source: European Centre for Disease Prevention and Control, accessed May 15, 2020. * Belgium reported suspected deaths due to COVID-19 in addition to confirmed deaths, thereby increasing the count relative to other countries.

Supplementary References

1. Erickson M. Heartened by employees' COVID-19 test results, Stanford Health Care resumes almost all procedures. Stanford Medicine News. **2020** May 4, 2020.
2. Cuomo A. Amid Ongoing COVID-19 Pandemic, Governor Cuomo Announces Fourth Region Hits Benchmark to Begin Reopening May 15th. Albany, NY: Governor's Press Office, **2020**.
3. Persson D. En av fem anställda på Danderyds sjukhus smittad. Vård Fokus. **2020** April 27, 2020.
4. Ardell P. By early April, coronavirus had spread to 10 percent of Stockholm population. Stockholm: KTH Press Office, **2020**.
5. Statistics Sweden (SCB): Population statistics. Available at: http://www.statistikdatabasen.scb.se/pxweb/sv/ssd/. Accessed 12 May 2020.
6. Spangler D, Blomqvist P, Lindberg Y, Winblad U. Small is beautiful? Explaining resident satisfaction in Swedish nursing home care. Bmc Health Serv Res **2019**; 19(1).
7. Ferguson NM, Laydon D, Nedjati-Gilani G, et al. Impact of non-pharmaceutical interventions (NPIs) to reduce COVID-19 mortality and healthcare demand. **2020**: DOI: 10.25561/77482.
8. Ferguson NM, Cummings DA, Fraser C, Cajka JC, Cooley PC, Burke DS. Strategies for mitigating an influenza pandemic. Nature **2006**; 442: 448-52.
9. Ferguson NM, Cummings DA, Cauchemez S, et al. Strategies for containing an emerging influenza pandemic in Southeast Asia. Nature **2005**; 437: 209-14.
10. Gardner JM, Willem L, van der Wijngaart W, Kamerlin SCL, Brusselaers N, Kasson P. Intervention strategies against COVID-19 and their estimated impact on Swedish healthcare capacity. medRxiv **2020**.
11. Li Q, Med M, Guan X, et al. Early Transmission Dynamics in Wuhan, China, of Novel Coronavirus–Infected Pneumonia. N Engl J Med **2020**; 382: 1199-207.
12. Verity R, Okell LC, Dorigatti I, et al. Estimates of the severity of COVID-19 disease: a model-based analysis. Lancet Infect Dis **2020**; Online First: DOI: 10.1016/S473-3099(20)30243-7.