SUPPLEMENTARY INFORMATION

The intensity of COVID-19 outbreaks is modulated by SARS-CoV-2 free-living survival and environmental transmission

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The supplemental information here contains additional background information, elaborations on methods, and additional data.

Author statement on mathematical modeling, and the purpose of this study.

In any outbreak, mathematical modeling efforts are crucial for organizing the available information, transforming the unknowns into testable hypotheses, and providing projections of how the disease may progress under a set of assumptions (45-46).This last point is an especially important, often under-treated aspect of computational and mathematical modeling of epidemics: predictions are useful, but only apply to sets of circumstances commensurate with the model assumptions.

Note that this study is not tasked with making any predictions about how any particular COVID-19 outbreak will happen in any setting. That models like the ones in this manuscript might apply to hypothetical scenarios doesn't mean, however, that they are wholly irrelevant. We believe that our study offers a unique perspective on COVID-19 that can be used to develop a better understanding of disease dynamics, and perhaps, more intelligent prevention and interventions.

It is, however, important to communicate what the purpose of a given mathematical model is. We believe that the model in this study will aid our understanding of epidemics like COVID-19. We reiterate, however, that the purpose of this model is not to explain any particular COVID-19 outbreak, nor make specific projections about any setting. Alternatively, our general results and perspectives are likely broadly applicable to many disease types.

SUPPLEMENTAL METHODS

1. Elaborated derivation of formulas 8a and 8b (from the main text).

Here we provide a detailed derivation of several equations described in the main text. First, we provide equations 8a and 8b, as described in the main text.:

$$R_p = \frac{\epsilon(\beta_A(\mu_S+\nu) + \beta_S(1-p)\omega)}{(\mu+\epsilon)(\mu+\omega)(\mu_S+\nu)},\ R_e^2 = \frac{\epsilon\,\beta_W(\sigma_A(\mu_S+\nu) + \sigma_S(1-p)\omega)}{k(\mu+\epsilon)(\mu+\omega)(\mu_S+\nu)} \qquad \text{(8a, 8b)}$$

First, R_p represents the number of secondary infections of people through person-to-person contact. This avenue is captured by the β_A and β_S terms in equations 1 and 2 in the main text. Namely, the rate of converting susceptible individuals to exposed individuals through person-to-person contact with infectious individuals is given by $\beta_A\ S\ I_A / N$ for asymptomatic individuals and by $\beta_S\ S\ I_S / N$ for symptomatic individuals. Near the disease-free-equilibrium (DFE), $N \sim S_0$ (i.e. the total population is initially susceptible) and so the rate of infection conversion for each, near the DFE, is given (approximately) by $\beta_A\ I_A$ and by $\beta_S\ I_S$. And so, the rate of conversion *per* infectious individual (I_A and I_S) is given simply by β_A and by β_S respectively. The average time that an individual remains in the infectious state is given by the reciprocal of the "exit"-rate of that state, which is $1/(\mu + \omega)$ for I_A, and by $1/(\mu_S + \nu)$ for I_S.

Thus the average number of secondary infections by individuals in the asymptomatic-infectious state is given by $\beta_A /(\mu + \omega)$ and by $\beta_S/(\mu_S + \nu)$ for the symptomatic-infectious state. It is not sufficient to simply add these quantities together as the rates/probabilities of entering the I_A and the I_S differ from one another. In order to properly combine these two rates, we need to determine the fraction of individuals, early-on, that will be in each infectious compartment—or, if one prefers, the *probability* of being in either infectious state. Individuals entering the I_A compartment can only do so by leaving the E compartment. And individuals can only leave the E compartment by entering the I_A compartment or through death.

The fraction of individuals that move on to the I_A compartment is therefore given by $\varepsilon / (\mu + \varepsilon)$, i.e. the ratio of the rate of entering the I_A compartment to the rate of leaving the E compartment, per person. And, the fraction of individuals that move on from the I_A compartment to the I_S compartment is, analogously, $(1 - p)\ \omega / (\mu + \omega)$, i.e. the ratio of the rate of entering the I_S compartment to the rate of leaving the I_A compartment. Note that individuals who make it to the I_S compartment must also have made it through the I_A compartment, and only a fraction $\varepsilon / (\mu + \varepsilon)$ do. So the rate of new infections caused by asymptomatic individuals and by symptomatic individuals needs to be weighted accordingly; $\varepsilon / (\mu + \varepsilon)$ for the former and $\varepsilon / (\mu + \varepsilon) \times (1 - p)\ \omega / (\mu + \omega)$ for the latter—the latter needs both factors since individuals who make it to I_S must also make it through I_A first. Thus, weighting the rates in a sum accordingly, one finds that the rate of secondary person-to-person infections is given by, $[\ \beta_A / (\mu + \omega) \times \varepsilon / (\mu + \varepsilon)\ ] + [\ \beta_S / (\mu_S + \nu) \times \varepsilon / (\mu + \varepsilon) \times (1 - p)\ \omega / (\mu + \omega)\ ]$, the first term in brackets corresponding to infections by asymptomatic individuals, and the second term to symptomatic individuals. This expression simplifies to the form given in equation 8a.

A similar story can be told for the number of secondary infections of people, mediated by the environment, R_e^2. Although, in this case, we can break down this reproductive ratio further into two sub-components of its own: one representing the flow of infection *to* the environment *from* people, and another *from* the environment *to* people. We will call the former R_{pe} and the latter R_{ep}.

The derivation for R_{pe} is identical to R_p described above except that we replace β_A and β_S by σ_A / S_0 and σ_S / S_0 respectively, since these latter quantities represent the rates of depositing infection to the environment (near the DFE), as opposed to the rates of depositing infection to people as in the case of β_A and β_S. The derivation is otherwise identical since the probability of an individual making it to the I_A or the I_S compartments remains unchanged. σ_A / S_0 and σ_S / S_0 can be seen to represent the rate of environmental infection per infectious individual (for asymptomatic and symptomatic individuals respectively) by examining the full rate in equation 6 (in the main text), namely ($\sigma_A\ I_A$ +

$\sigma_S I_S) (1 - W) /N$. Near the DFE, the factor $1 - W$ is nearly equal to 1, and $N \sim S_0$. Thus, as above, the individual rates of infecting the environment *per* infectious person is given by σ_A/S_0 for asymptomatic individuals and by σ_S/S_0 for symptomatic individuals. The apparent dependence on S_0 in the $\mathcal{R}_0$ is a curious feature of the model, although as we will soon see, this dependence cancels out entirely, leaving the full $\mathcal{R}_0$ devoid of any such dependence on population density. Following the lines of the derivation for R_p described above, but with β_A and β_S replaced by σ_A/S_0 and σ_S/S_0 respectively, one arrives at,

$$\frac{\epsilon(\sigma_A(\mu_S+\nu)+\sigma_S\,(1-p)\,\omega)}{S_0(\mu+\epsilon)(\mu+\omega)(\mu_S+\nu)} \tag{S1}$$

This quantity can be interpreted as the *fraction* of the environment infected by a single infectious individual near the DFE. Note that we do not specify their infectious-type (asymptomatic or not) as this feature is implicit in the weighted sum we took to arrive at equation S1 (and equation 8a), representing the *expectation value* of the fraction of new environmental infections. Note also that we are justified in discussing increments in the *fraction* of the environment infected as these calculations are performed near the DFE, where we can assume only a very small fraction of the environment is infectious and thus need not be concerned by scaling the fractional quantities to values exceeding unity.

Lastly, the derivation for R_{ep} is straightforward. The rate of infection from the environment is given by $\beta_W W S$ (equations 1 and 2 in the main text). Near the DFE, $S \sim S_0$, and so the rate of people infected by the environment *per fraction* of the infected environment (W) is given simply by $\beta_W S_0$. The average time any fraction of the infected environment remains infected is given by $1/k$, i.e. the reciprocal of the exit rate of the W compartment. Thus, the number of new infections of people, per fraction of the environment, near the DFE, is given simply by

$$\frac{\beta_W S_0}{k} \tag{S2}$$

Thus, the number of *people* infected by an individual who has first deposited infection to the environment (parameterized here as some "fraction" of the environment) is simply the product of the fraction of the environment infected per infectious individual (equation S1) and the number of people infected per infectious fraction of the environment (equation S2).

$$\frac{\epsilon\,\beta_W(\sigma_A(\mu_S+\nu)+\sigma_S(1-p)\omega)}{k(\mu+\epsilon)(\mu+\omega)(\mu_S+\nu)} \tag{S3}$$

As promised, the dependency on S_0 cancels out in the product. The identification of this product with R_e^2 rather than with R_e may be better understood now having gone over its derivation by hand. A reproductive ratio representing the number of new infections of people per infectious person that are mediated through the environment is really the product of two reproductive ratios: one representing the spread of infection from people to the environment, and another representing the spread of infection from the environment to people. If one follows the lines of previous efforts for calculating reproductive ratios in general ODE-systems (47), then they would find that for the system of ODEs (equations 1-6 in the main text), the full $\mathcal{R}_0$ expression is composed of the two subcomponents (R_p and R_e^2) described here in a way expressed by equation 7 in the main text.

A brief explanation of the final $\mathcal{R}_0$ form (which is made clearer by following methods as described in prior studies (47) is that the form in equation 7 in the main text is the maximum eigenvalue of the next-generation matrix G:

$$G = \begin{pmatrix} R_p & R_{ep} \\ R_{pe} & 0 \end{pmatrix} \tag{S4}$$

This matrix represents the amount by which the infected populations (taken as a 2 x 1 vector of inputs to the matrix) are scaled from one infection generation to the next (47). For simplicity, here we only consider two infectious components: people and the infectious portion of the environment, as opposed to splitting people into all of the infected categories in our model (*E, I_A, & I_S*), simplifying *G* to a 2 x 2 matrix. As explained in more detail in Diekmann et al. 2010 (47), this simplification will preserve the maximum eigenvalue of the system, although one could just as well follow the lines of the calculation using the full 4 x 4 version of *G*, accounting for all infected compartments *E, I_A, I_S, & W*.

One will notice that *G* in equation S4 includes the three subcomponents of the $\mathcal{R}_0$ discussed above (equation 8a, and equations S1 & S2). Each element represents an $\mathcal{R}_0$ from *a* to *b* where *a* and *b* could represent people or the environment, making the 0-0 component of *G* (using indices beginning at 0) the $\mathcal{R}_0$ of people to people, the 0-1 component the $\mathcal{R}_0$ of the environment to people, the 1-0 component the $\mathcal{R}_0$ of people to the environment, and lastly the 1-1 component gives the $\mathcal{R}_0$ of the environment to the environment, which is zero in this case as the environment does not infect itself. Note that under this basis, the vector of inputs is the column vector given by (infected people at time t, infected fraction of environment at time t). As is readily verifiable, solving for the maximum eigenvalue of *G* gives equation 7 in the main text, with the identification that $R_e^2 = R_{ep}R_{pe}$.

2. Model fitting and parameter estimation

Mathematical models like the ones developed in this study require the use of parameters, terms that dictate the way that the different parts of the model interact. In the case of SARS-CoV-2 transmission, we are fortunate that many early studies have provided estimates for many terms, like the incubation period, and rate of recovery. That said, there remains many terms for which there are no solid estimates. In this scenario, we must estimate these values. There are many ways to attempt this. One way to do this is to fit the model (using the fixed parameters based on values that we do have less uncertainty for) in order to estimate the unknown parameters. To do this, we use real-

world data on the COVID-19 outbreak from 17 countries, in their “early-stage” outbreaks.

We define the “early stage” of the epidemic as the 30 days following the first day with ≥10 cumulative infected individuals within a particular region. This allowed us to standardize our comparisons between different regions, leading to more robust fitting results. We choose case counts ≥10 in order to avoid early difficulties with testing and recording and to give the infection sufficient time to settle into a more consistent doubling time. The window of 30 days was chosen in order to maximize the number of data points while also allowing enough room to include countries who have had a long enough exposure to SARS-CoV-2 to be included in the analysis.

We conduct our analysis using early stage data (30 days) from the following 17 countries (in alphabetical order): Australia, Austria, Canada, China, Denmark, France, Germany, Iran, Italy, Netherlands, Norway, Spain, Sweden, South Korea, Switzerland, the United Kingdom, and the United States. These countries were chosen because they had both the highest cumulative COVID-19 cases (of the 181 total countries affected) as of 03/30/2020, and because the outbreak had developed for at least 30 days following the first day with ≥10 cumulative infected cases within each country (37,42).

Fitting our model to data from each of these countries, we deduce values for 6 model parameters, β_A, β_S, β_W, σ_A, σ_S, *and* ε in the SEIR-W case, and 3 model parameters β_A, β_S, *and* ε in the SEIR case. Using the fitted parameters, we calculate Akaike Information Criterion (AIC) values (equation S6, discussed below) for the two versions of the model: one with the environmental reservoir included (SEIR-W) and one without it (SEIR). The AIC is an estimator representing the quality of a statistical model given a particular set of data and thus provides a means for model selection.

The environment can be “turned off” by setting certain parameters (arrows in the compartmental diagram) to 0. These include β_W, σ_A, and σ_S; each representing some coupling between the environment and people. The reciprocal of ε gives the number of

days an individual is expected to remain in the *E* compartment before becoming asymptomatically infectious. We include this parameter in both fits (SEIR-W and SEIR) since little is known about how long the period is, after being initially exposed to SARS-CoV-2, before an individual becomes infectious (which we assume occurs *before* symptom onset). The only constraint on ε in the curve fitting is that $1/\varepsilon$ lies between 0 and 5.5 days (the incubation period). We set $1/\omega$ (the expected time in the I_A compartment) to be the remainder of the time in the incubation period; i.e. $\omega^{-1} = \eta - \varepsilon^{-1}$ where η is the incubation period (5.5 days). All other fitting parameters are constrained to lie between 0 and 100, which is expected to provide ample room in the parameter space to locate an appropriate fit.

We use the python module *scipy.optimize.curve fit* to fit our model to the data from each country. This program uses the Levenberg-Marquardt algorithm to perform a least-squares regression analysis (48). Using the optimal parameters, we computed the log-likelihood, *L*, using the following formula:

$$L = -\frac{N}{2} ln\, 2\pi - N\, ln\, \sigma - \frac{\sum_{t=1}^{N}\left(y_t - f(t,q)\right)^2}{2\sigma^2} \quad \text{(S5)}$$

$f(t,q)$ is the number of people infected on day *t* predicted by the model, *y* represents the data, *N* is the number of data points (in this case, 30), and σ^2 is taken to be the maximum-likelihood estimation (MLE) of the variance, given by (49).

$$\sigma_{MLE}^2 = \frac{1}{N}\sum_{t=1}^{N}\left(y_t - f(t,q)\right)^2 \quad \text{(S6)}$$

From this we compute an AIC value using the formula,

$$AIC = 2(k_\theta - L) \quad \text{(S7)}$$

where k_{θ} is the number of fitting parameters (6 for SEIR-W and 3 for SEIR) and L is the log-likelihood with MLE-calculated variance shown above. As mentioned above, we compute AIC values with and without the environmental reservoir. In each case, we perform the least-squares regression and arrive at the optimal parameters: β_A, β_S, β_W, σ_A, σ_S, and ε in the former, and β_A, β_S, and ε in the latter.

Having established parameter values from this analysis, our primary aim is to assess the impact of SARS-CoV-2 environmental transmission (via copper, steel, cardboard, and plastic) on both the general (averaged) and country specific dynamics of the COVID-19 pandemic.

As stated previously, these 17 countries were chosen because they both had the highest cumulative COVID-19 cases (of the 181 total countries affected) as of the end of March, 2020.

Initial values

Country	**Value of S_0 (people)**	**Value of I_{S0}**	**Source (S_0 & I_{S0})**
Australia	25,499,881	11	(37)
Austria	9,006,400	10	(37)
Canada	37,742,157	11	(37)
China	60,000,000	27	(37)
Denmark	5,792,203	10	(37)
France	65,273,512	11	(37)
Germany	83,783,945	11	(37)

Iran	83,992,953	18	(37)
Italy	60,461,828	17	(37)
Netherlands	17,134,873	13	(37)
Norway	5,421,242	15	(37)
South Korea	51,269,183	12	(37)
Spain	46,754,783	12	(37)
Sweden	10,099,270	12	(37)
Switzerland	8,654,618	12	(37)
United Kingdom	67,886,004	13	(37)
United States	331,002,647	11	(37)

Table S1. Initial values used for susceptible and symptomatically infected individuals for each country.

***Fitted* parameter values, SEIR-W (by country)**

Country	β_A	β_S	β_W	σ_A	σ_S	$1/\varepsilon$	**Source**
Australia	0.233	1.231	0.000	2.143	33.960	5.445	[Fitted]
Austria	0.000	0.000	0.071	2.983	0.001	0.000	[Fitted]
Canada	1.503	5.015	0.001	0.359	30.454	5.032	[Fitted]

China	0.915	0.000	0.000	1.641	12.829	2.403	[Fitted]
Denmark	0.211	0.000	0.128	0.000	11.471	2.749	[Fitted]
France	0.777	0.000	0.000	0.079	26.404	2.426	[Fitted]
Germany	0.649	0.000	0.000	4.349	75.298	2.451	[Fitted]
Iran	0.470	0.000	0.036	0.000	0.000	2.749	[Fitted]
Italy	0.479	0.000	0.032	0.000	0.482	1.744	[Fitted]
Netherlands	0.000	0.000	0.037	6.054	0.001	0.003	[Fitted]
Norway	0.525	0.000	0.075	0.000	0.000	2.748	[Fitted]
South Korea	0.752	2.100	0.000	2.306	3.804	2.434	[Fitted]
Spain	0.724	0.000	0.020	0.000	2.243	2.591	[Fitted]
Sweden	0.523	0.000	0.027	0.000	9.322	2.747	[Fitted]
Switzerland	0.590	0.000	0.100	0.000	0.000	2.747	[Fitted]
United Kingdom	0.421	0.000	0.002	27.754	8.622	1.323	[Fitted]
United States	0.573	0.000	0.000	10.199	14.474	2.540	[Fitted]

Table S2. Fitted parameters used in the SEIR-W model for each of the 17 selected countries.

***Fitted* parameter values, SEIR (by country)**

Country	β_A	β_S	β_W	σ_A	σ_S	$1/\varepsilon$	**Source**
Australia	3.042	1.058	0.000	0.000	0.000	5.445	[Fitted]
Austria	0.000	12.203	0.000	0.000	0.000	0.009	[Fitted]
Canada	0.000	9.176	0.000	0.000	0.000	5.445	[Fitted]
China	0.915	0.000	0.000	0.000	0.000	2.404	[Fitted]
Denmark	0.000	9.626	0.000	0.000	0.000	0.022	[Fitted]
France	0.777	0.000	0.000	0.000	0.000	2.426	[Fitted]
Germany	0.649	0.000	0.000	0.000	0.000	2.451	[Fitted]
Iran	0.000	12.323	0.000	0.000	0.000	5.307	[Fitted]
Italy	0.233	16.127	0.000	0.000	0.000	5.445	[Fitted]
Netherlands	0.001	12.427	0.000	0.000	0.000	0.008	[Fitted]
Norway	0.001	9.250	0.000	0.000	0.000	0.009	[Fitted]
South Korea	1.097	1.956	0.000	0.000	0.000	3.389	[Fitted]
Spain	0.000	17.840	0.000	0.000	0.000	0.014	[Fitted]
Sweden	0.000	9.680	0.000	0.000	0.000	0.106	[Fitted]
Switzerland	0.000	12.949	0.000	0.000	0.000	0.014	[Fitted]

United Kingdom	0.000	11.715	0.000	0.000	0.000	5.445	[Fitted]
United States	0.574	0.000	0.000	0.000	0.000	2.544	[Fitted]

Table S3. Fitted parameters used in the model for each of the 17 selected countries. β_W, σ_A, and σ_S are set to zero when we run the standard SEIR country fits.

3. Model fitting and parameter estimation II

Tables S1-S3 display all the data relevant to the model initial conditions (for each country), and the AIC values for the country fits. In Figure S1, we show graphs corresponding to individual country fits. Note that four countries with the most explosive early outbreaks—Spain, Italy, Iran and Switzerland—appear in the main text (Figure 2). Explosiveness was defined by the highest cumulative number of infected cases after 30 days following the first day when cases were greater than or equal to 10). These four also appear in the table, however, so that their fits can be compared to the other 14 countries in the set.

Akaike Information Criterion (by country)

	Australia	**Austria**	**Canada**	**China**	**Denmark**
AIC SEIR-*W*	94.6	400	311.9	388.5	332.6
AIC SEIR	88.5	422	307	382.5	349.9

	France	**Germany**	**Iran**	**Italy**	**Netherlands**
AIC SEIR-*W*	273.7	221.4	427	439.5	377.9

AIC SEIR	267.7	215.4	472.3	503.8	420.8

	Norway	**South Korea**	**Spain**	**Sweden**	**Switzerland**
AIC SEIR-*W*	344.1	356.1	456	322.7	416.5
AIC SEIR	363.4	350.1	498.1	339	439.9

	United Kingdom	**United States**
AIC SEIR-*W*	372.8	191.9
AIC SEIR	375	185.9

Table S4. The Akaike information criterion (AIC) for the fits conducted of the “early stage” (30 days) of the outbreak in each of the selected 17 countries with (SEIR-W) and without (SEIR) the WAIT component present. The larger of each pair of AIC scores is highlighted in red, and the smaller in green.

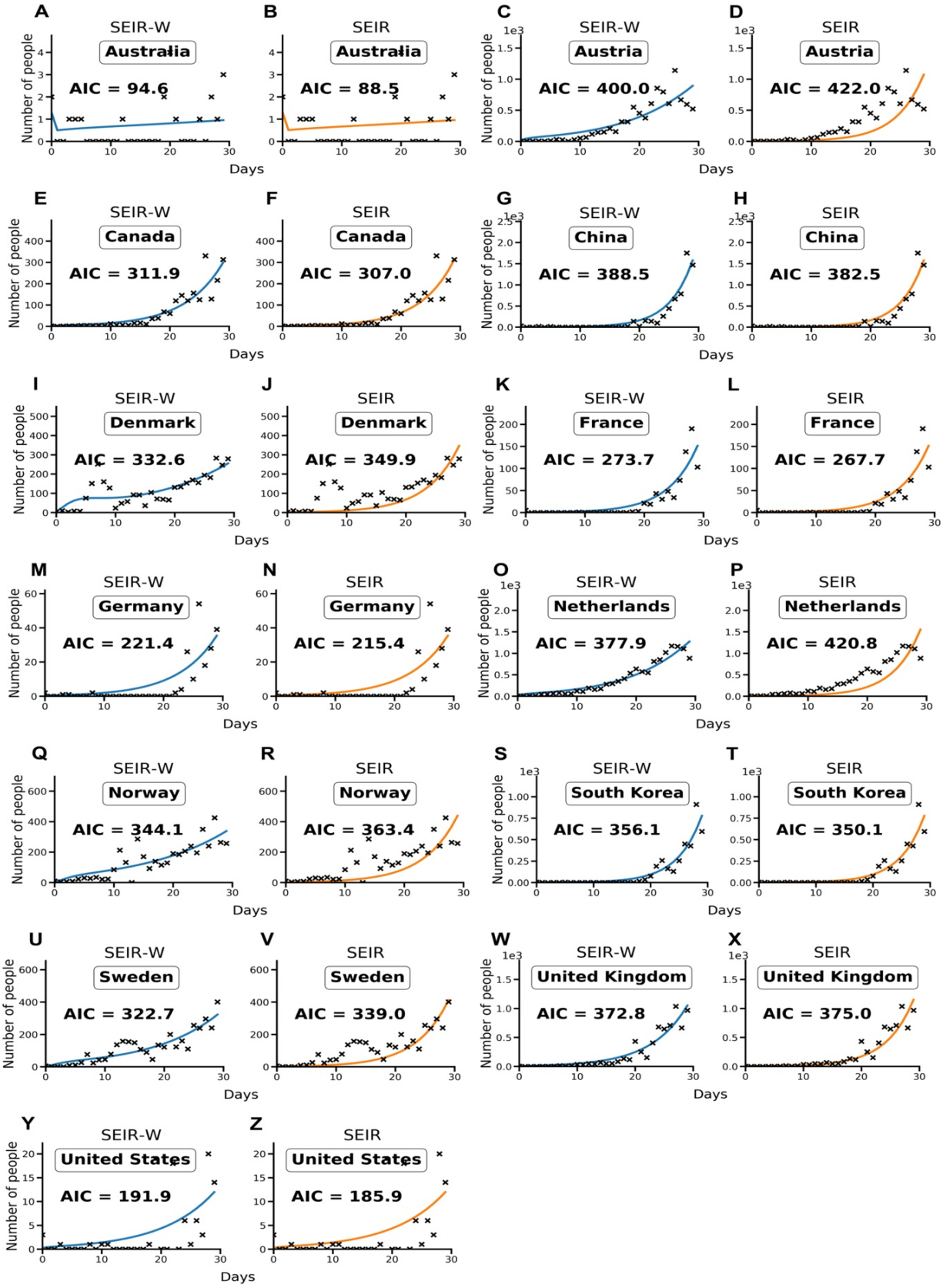
A
SEIR-W
Australia
AIC = 94.6
B
SEIR
Australia
AIC = 88.5
C
SEIR-W
Austria
AIC = 400.0
D
SEIR
Austria
AIC = 422.0
E
SEIR-W
Canada
AIC = 311.9
F
SEIR
Canada
AIC = 307.0
G
SEIR-W
China
AIC = 388.5
H
SEIR
China
AIC = 382.5
I
SEIR-W
Denmark
AIC = 332.6
J
SEIR
Denmark
AIC = 349.9
K
SEIR-W
France
AIC = 273.7
L
SEIR
France
AIC = 267.7
M
SEIR-W
Germany
AIC = 221.4
N
SEIR
Germany
AIC = 215.4
O
SEIR-W
Netherlands
AIC = 377.9
P
SEIR
Netherlands
AIC = 420.8
Q
SEIR-W
Norway
AIC = 344.1
R
SEIR
Norway
AIC = 363.4
S
SEIR-W
South Korea
AIC = 356.1
T
SEIR
South Korea
AIC = 350.1
U
SEIR-W
Sweden
AIC = 322.7
V
SEIR
Sweden
AIC = 339.0
W
SEIR-W
United Kingdom
AIC = 372.8
X
SEIR
United Kingdom
AIC = 375.0
Y
SEIR-W
United States
AIC = 191.9
Z
SEIR
United States
AIC = 185.9
Number of people
Days

Figure S1. Graphical depiction of data in the Table S4. A-Z correspond to 13 country fits of the mathematical models—SEIR and SEIR-W. Four other country fits are depicted in the main text Figure 2 (Spain, Italy, Iran, Switzerland).

4. Model sensitivity analysis

Partial Rank Correlation Coefficient (PRCC). A key aspect of model building is a "sensitivity analysis," or a test of how the model dynamics change as a result of changes in parameters.

We use the *Partial Rank Correlation Coefficient* (PRCC)—an established method—to assess the sensitivity of various aspects of our model with respect to changes in the parameter values of our model (50).

Using this method, we can identify certain parameters which may be quite influential to the dynamics of the infection. Here we briefly review the steps we took to compute the PRCC values for several aspects of the model. As a rough outline of the calculation, one begins by constructing *M* random samples of the parameter values around the predetermined set of expected values of parameters—let us say there are *n* parameters. For each of these *M* samples, which are selected using the *Latin Hypercube Sampling* (LHS) method (51) the value of whatever model aspect is calculated, such as the $\mathcal{R}_0$ or the number of people infected after 30 days. In the LHS method, the *n* parameters are varied independently of each other and so this procedure allows us to assess sensitivity of all parameters collectively, rather than assessing the sensitivity of the model with respect to changes in a single parameter at a time. Then the sampled values for each of the *n* parameters are numbered *1* through *M* depending on how they rank in the sample, resulting in *n* vectors of length *M* giving some permutation of the numbers *1* through *M*. The same ranking procedure is performed for the output of the model, whether that is the $\mathcal{R}_0$ or something else. Consequently, there are now *n+1* permutations of the numbers *1* through *M*. The next step is to compute the ordinary correlation coefficient between all pairs of these *n+1* vectors, and to arrange

them into a matrix *C*—note that *C* is a symmetric matrix. Thus, C_{ij} gives the correlation between the *ith* and *jth* parameter vector or model value vector. In general, most of these values are expected to be fairly close to 0, except for the diagonal (all ones) and the values associated with correlations with the model value vector. The last step is to compute the matrix inverse of *C*, which we call *B*. Finally, the PRCC values (one for each parameter) are defined by,

$$PRCC_i = -B_{i\,n+1}/\sqrt{B_{i\,i}B_{n+1\,n+1}} \quad \text{(S8)}$$

where *i* refers to the *ith* parameter.

We compute the PRCC values for four aspects of our model, (1) $\mathcal{R}_0$,(2) cumulative number of symptomatic infections after 30 days, (3) time to the symptomatic peak (t_{max}), and (4) symptomatic peak (I_{max}),

	Copper	**Cardboard**	**Stainless steel**	**Plastic**
SARS-CoV-2 decay time ($1/k$)	4 hours	24 hours	48 hours	72 hours
$\mathcal{R}_0$	2.4	2.67	2.94	3.18
Time to reach maximum # of I_S	88.4	65.1	56.6	52.6
The maximum number of I_S	1,023,100	1,177,930	1,254,260	1,292,010
The number of I_S after 30 days	5960	58240	155390	256420
The number of deaths after 30 days	55	461	1,133	1,814

Table S5. Summary of the “surface world” simulations key features.