Herd immunity thresholds for SARS-CoV-2 estimated from unfolding epidemics

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As severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) spreads, the susceptible subpopulation declines causing the rate at which new infections occur to slow down. Variation in individual susceptibility or exposure to infection exacerbates this effect. Individuals that are more susceptible or more exposed tend to be infected and removed from the susceptible subpopulation earlier. This selective depletion of susceptibles intensifies the deceleration in incidence. Eventually, susceptible numbers become low enough to prevent epidemic growth or, in other words, the herd immunity threshold is reached. Here we fit epidemiological models with inbuilt distributions of susceptibility or exposure to SARS-CoV-2 outbreaks to estimate basic reproduction numbers (R_0) alongside coefficients of individual variation (CV) and the effects of containment strategies. Herd immunity thresholds are then calculated as $1-(1/R_0)^{1/(1+CV^2)}$ or $1-(1/R_0)^{1/(1+2CV^2)}$, depending on whether variation is on susceptibility or exposure. Our inferences result in herd immunity thresholds around 10-20%, considerably lower than the minimum coverage needed to interrupt transmission by random vaccination, which for R_0 higher than 2.5 is estimated above 60%. We emphasize that the classical formula, $1-1/R_0$, remains applicable to describe herd immunity thresholds for random vaccination, but not for immunity induced by infection which is naturally selective. These findings have profound consequences for the governance of the current pandemic given that some populations may be close to achieving herd immunity despite being under more or less strict social distancing measures.

Scientists throughout the world have engaged with governments, health agencies, and with each other, to address the ongoing pandemic of coronavirus disease (COVID-19). Mathematical models have been central to important decisions concerning

contact tracing, quarantine, and social distancing, to mitigate or suppress the initial pandemic spread[1]. Successful suppression, however, may leave populations at risk to resurgent waves due to insufficient acquisition of immunity. Models have thus also addressed longer term SARS-CoV-2 transmission scenarios and the requirements for continued adequate response[2]. This is especially timely as countries relax lockdown measures that have been in place over recent months with varying levels of success in tackling national outbreaks.

Here we demonstrate that individual variation in susceptibility or exposure (connectivity) accelerates the acquisition of immunity in populations. More susceptible and more connected individuals have a higher propensity to be infected and thus are likely to become immune earlier. Due to this selective immunization by natural infection, heterogeneous populations require less infections to cross their herd immunity threshold (HIT) than suggested by models that do not fully account for variation. We integrate continuous distributions of susceptibility or connectivity in otherwise basic epidemic models for COVID-19 which account for realistic intervention effects and show that as coefficients of variation (CV) increase from 0 to 5, HIT declines from over 60%[3,4] to less than 10%. We then fit these models to series of daily new cases to estimate CV alongside basic reproduction numbers (R_0) and derive the corresponding HITs.

Effects of individual variation on SARS-CoV-2 transmission

SARS-CoV-2 is transmitted primarily by respiratory droplets and modelled as a susceptible-exposed-infectious-recovered (SEIR) process.

Variation in susceptibility to infection

Individual variation in susceptibility is integrated as a continuously distributed factor that multiplies the force of infection upon individuals[5] as

$$\dot{S}(x) = -\lambda(x)xS(x), \quad (1)$$

$$\dot{E}(x) = \lambda(x)x[S(x) + \sigma R(x)] - \delta E(x), \quad (2)$$

$$\dot{I}(x) = \delta E(x) - \gamma I(x), \quad (3)$$

$$\dot{R}(x) = (1 - \phi)\gamma I(x) - \sigma\lambda(x)xR(x), \quad (4)$$

where $S(x)$ is the number of individuals with susceptibility x, $E(x)$ and $I(x)$ are the numbers of individuals who originally had susceptibility x and became exposed and infectious, while $R(x)$ counts those who have recovered and have their susceptibility reduced to a reinfection factor σ due to acquired immunity. δ is the rate of progression from exposed to infectious, γ is the rate of recovery or death, ϕ is the proportion of individuals who die as a result of infection and $\lambda(x) = (\beta/N)\int[\rho E(y) + I(y)]\,dy$ is the average force of infection upon susceptible individuals in a population of size N and transmission coefficient β. Standardizing so that susceptibility distributions have mean $\int xg(x)\,dx = 1$, given a probability density function $g(x)$, the basic reproduction number is

$$R_0 = \beta\left(\frac{\rho}{\delta} + \frac{1}{\gamma}\right), \quad (5)$$

where ρ is a factor measuring the infectivity of individuals in compartment E in relation to those in I. The coefficient of variation in individual susceptibility $CV = \sqrt{\int(x-1)^2 g(x)\,dx}$ is explored as a parameter. Non-pharmaceutical interventions (NPIs) designed to control transmission typically reduce β and hence R_0. We denote the resulting controlled reproduction number by R_c. The effective reproduction

number R_{eff} is another useful indicator obtained by multiplying R_c by the susceptibility of the population, in this case written as $R_{eff}(t) = R_c(t) \int xS(x,t)\, dx/N(t)$ to emphasize its time dependence.

Figure 1 depicts model trajectories fitted to suppressed epidemics (orange) in 4 European countries (Belgium, England, Portugal and Spain) assuming gamma distributed susceptibility and no reinfection ($\sigma = 0$). We estimate: R_0 rounding 5 (Belgium), 2.9 (England), 4.3 (Portugal) and 4.1 (Spain); individual susceptibility CV reaching 3.9 (Belgium), 1.9 (England), 4.3 (Portugal) and 3.2 (Spain); and overall intervention efficacy at maximum (typically during lockdown) being 60% (Belgium), 48% (England), 69% (Portugal) and 63% (Spain). Another estimated parameter is the day when NPIs begin to affect transmission, after which we assume a linear intensification from baseline over 21 days, remaining at maximum intensity for 30 days and linearly lifting back to baseline over a period of 120 days (although we have confirmed that the results do not change significantly if measures are lifted over slightly longer time frames, such as 150 or 180 days). Denoting by $d(t)$ the proportional reduction in average risk of infection due to interventions, in this case we obtain $R_c(t) = [1 - d(t)]R_0$ which is depicted for each country, alongside $R_{eff}(t)$, underneath the respective epidemic trajectories. Overlaid on the R_c plots are mobility data from Google[6], showing excellent agreement with our independently chosen framework and estimate for the time R_{eff} starts declining. To assess the potential for case numbers to overshoot if NPIs had not been applied, we rerun the model with $d(t) = 0$ and obtain the unmitigated epidemics (black). Further details and sensitivity analyses are described in Methods.

Variation in connectivity

In a directly transmitted infectious disease, such as COVID-19, variation in exposure to infection is primarily governed by patterns of connectivity among individuals. We incorporate this in the system (Equations 1-4) assuming that individuals mix at random (but see Methods for more general formulations that enable other mixing patterns). Under random mixing and heterogeneous connectivity, the force of infection[7] is written as $\lambda(x) = (\beta/N)(\int y[\rho E(y) + I(y)]\, dy / \int y g(y)\, dy)$, the basic reproduction number is

$$R_0 = (1 + CV^2)\beta\left(\frac{\rho}{\delta} + \frac{1}{\gamma}\right), \quad (6)$$

$R_c(t)$ is as above and $R_{eff}(t)$ is derived by a more general expression given in Methods. Applying this model to the same epidemics as before we estimate: R_0 rounding 7.1 (Belgium), 3.8 (England), 7.9 (Portugal) and 6.6 (Spain); individual susceptibility CV reaching 2.9 (Belgium), 1.6 (England), 4.0 (Portugal) and 2.7 (Spain); and intervention efficacy during lockdown being 73% (Belgium), 58% (England), 80% (Portugal) and 72% (Spain).

Comparing the two models, variation in connectivity systematically leads to estimates that are higher for R_0, lower for CV, and higher for the efficacy of non-pharmaceutical interventions. Nevertheless, the percentage of the population required to be immune to curb the epidemic and prevent future waves when interventions are lifted appears remarkably conserved across models: 9.6 vs 11% (Belgium); 20 vs 21% (England); 7.3 vs 6.0% (Portugal); and 12 vs 11% (Spain). This property is further explored below.

Herd immunity thresholds and their conserveness across models

Individual variation in risk of acquiring infection is under selection by the force of infection, whether individual differences are due to biological susceptibility, exposure, or both. The most susceptible or exposed individuals are selectively removed from the susceptible pool as they become infected and eventually recover (some die), resulting in decelerated epidemic growth and accelerated induction of immunity in the population. In essence, the *herd immunity threshold* defines the percentage of the population that needs to be immune to reverse epidemic growth and prevent future waves. When individual susceptibility or connectivity is gamma-distributed and mixing is random, HIT curves can be derived analytically[8] from the model systems (Equations 1-4, with the respective forces of infections). In the case of variation in susceptibility to infection we obtain

$$HIT = 1 - \left[\frac{1 - \sigma R_0}{(1-\sigma)R_0}\right]^{\frac{1}{1+CV^2}}, \quad (7)$$

while variable connectivity results in

$$HIT = 1 - \left[\frac{1 - \sigma R_0}{(1-\sigma)R_0}\right]^{\frac{1}{1+2CV^2}}. \quad (8)$$

In more complex cases HIT curves can be approximated numerically. Figure 3 shows the expected downward trends in HIT and the sizes of the respective unmitigated epidemics for SARS-CoV-2 without reinfection ($\sigma = 0$) as the coefficients of variation are increased (gamma distribution shapes adopted here are illustrated in Extended Data Figure 1; for robustness of the trends to other distributions see Gomes et al[9]). Values of R_0 and CV estimated for our study countries are overlaid to mark the respective HIT and final epidemic sizes. While herd immunity is expected to require 60-80% of a homogeneous population to have been infected, at the cost of

157 infecting almost the entire population if left unmitigated, given an R_0 between 2.5 and
158 5, these percentages drop to the range 10-20% or lower when CV is roughly between
159 2 and 5.

160 When acquired immunity is not 100% effective ($\sigma > 0$) HITs are relatively higher
161 (Extended Data Figure 2). However, there is an upper bound for how much it is
162 reasonable to increase σ before the system enters a qualitatively different regime.
163 Above $\sigma = 1/R_0$ – the *reinfection threshold*[10,11]– infection becomes stably endemic
164 and the HIT concept no longer applies. Respiratory viruses are typically associated
165 with epidemic dynamics below the reinfection threshold, characterized by seasonal
166 epidemics intertwined with periods of undetection.

167 Individual variation in exposure, in contrast with susceptibility, accrues from complex
168 patterns of human behaviour which have been simplified in our model. To explore the
169 scope of our results we generalise our models (Methods) by relaxing some key
170 assumptions. First, we enable mixing to be assortative in the sense that individuals
171 contact predominantly with those of similar connectivity. Formally, an individual
172 with connectivity x, rather than being exposed uniformly to individuals of all
173 connectivities y, has contact preferences described by a normal distribution on the
174 difference $y - x$. We find this modification to have negligible effect on HIT
175 (Extended Data Figure 3). Second, we allow connectivity distributions to change in
176 shape (not only scale) when subject to social distancing. In particular we modify the
177 model so that CV reduces in proportion to the intensity of social distancing (Extended
178 Data Figure 4) and replicate the fittings to epidemics in our study countries (Extended
179 Data Figure 5). We find a general tendency for this model to estimate higher values
180 for R_0 and CV while HIT remains again remarkably robust to the change in model

assumptions.

Herd immunity thresholds and seroprevalence at sub-national levels

As countries are conducting immunological surveys to assess the extent of exposure to SARS-CoV-2 in populations it is of practical importance to understand how HIT may vary across regions. We have redesigned our analyses to address this question. Series of daily new cases were stratified by region. Fitting the models simultaneously to the multiple series enabled the estimation of local parameters (R_0 and CV) while the effects of NPIs were estimated at country level. Extended Data Figures 6-9 show how the modelled epidemics fit the regional data and include an additional metric to describe the cumulative infected percentage. These model projections are comparable to data from seroprevalence studies such as Spain[12]. We emphasise that seroprevalence estimates generally lie slightly below our cumulative infection curves (Extended Data Figure 9) consistently with recent findings that a substantial fraction of infected individual does not exhibit detectable antibodies[13]. In addition to their practical utility these results begin to unpack some of the variation in HIT within countries: Belgium (9.4-11%), England (16-26%), Portugal (7.1-9.9%) and Spain (7.5-21%).

Discussion

The concept of *herd immunity* was developed in the context of vaccination programs[14,15]. Defining the percentage of the population that must be immune to cause infection incidences to decline, HITs constitute useful targets for vaccination coverage. In idealized scenarios of vaccines delivered at random and individuals mixing at random, HITs are given by a simple formula $(1 - 1/R_0)$ which, in the case of SARS-CoV-2, suggests that 60-80% of randomly chosen subjects of the population

would need be immunized to halt spread considering estimates of R_0 between 2.5 and 5. This formula does not apply to infection-induced immunity because natural infection does not occur at random. Individuals who are more susceptible or more exposed are more prone to be infected and become immune, providing greater community protection than random vaccination[16]. In our model, the HIT declines sharply when coefficients of variation increase from 0 to 2 and remains below 20% for more variable populations. The magnitude of the decline depends on what property is heterogeneous and how it is distributed among individuals, but the downward trend is robust as long as susceptibility or exposure to infection are variable (Figure 3 and Extended Data Figures 3) and acquired immunity is efficacious enough to keep transmission below the reinfection threshold (Extended Data Figure 2).

Several candidate vaccines against SARS-CoV-2 are showing promising safety and immunogenicity in early-phase clinical trials[17,18], although it is not yet known how this will translate into effective protection. We note that the reinfection threshold[10,11] informs not only the requirements on naturally acquired immunity but, similarly, it sets a target for how efficacious a vaccine needs to be in order to effectively interrupt transmission. Specifically, given an estimated value of R_0 we should aim for a vaccine efficacy of $1 - 1/R_0$ (60% or 80% if R_0 is 2.5 or 5, respectively). A vaccine whose efficacy is insufficient to bring the system below the reinfection threshold will not interrupt transmission.

Heterogeneity in the transmission of respiratory infections has traditionally focused on variation in exposure summarized into age-structured contact matrices. Besides overlooking differences in susceptibility given exposure, the aggregation of

individuals into age groups reduces coefficients of variation. We calculated CV for the landmark POLYMOD matrices[19,20] and obtained values between 0.3 and 0.5. Recent studies of COVID-19 integrated contact matrices with age-specific susceptibility to infection (structured in three levels)[21] or with social activity (three levels also)[22] which, again, resulted in coefficients of variation less than unity. We show that models with coefficients of variation of this magnitude would appear to differ only moderately from homogeneous approximations when compared with our estimates, which are consistently above 1 in England and above 2 in Belgium, Portugal and Spain. In contrast with reductionistic procedures that aim to reconstruct variation from correlate markers left on individuals (such as antibody or reactive T cells for susceptibility, or contact frequencies for exposure), we have embarked on a holistic approach designed to infer the whole extent of individual variation from the imprint it leaves on epidemic trajectories. Our estimates are therefore expected to be higher and should ultimately be confronted with more direct measurements as these become available. Adam at et[23] conducted a contact tracing study in Hong Kong and estimated a coefficient of variation of 2.5 for the number of secondary infections caused by individuals, attributing 80% of transmission to 20% of cases. This statistical dispersion has been interpreted as reflecting a common pattern of contact heterogeneity which has been corroborated by studies that specifically measure mobility[24]. According to our inferences, 20% of individuals may be responsible for 47-94% infections depending on model and country. In parallel, there is accumulating evidence of individual variation in the immune system′s ability to control SARS-CoV-2 infection following exposure[25,26]. While our inferences serve their purpose of improving accuracy in model predictions, diverse studies such as these are necessary for developing interventions targeting individuals who may be at higher risk of being

infected and propagating infection in the community.

Country-level estimates of R_0 reported here are in the range 3-5 when individual variation in susceptibility is factored and 4-8 when accounting for variation in connectivity. The homogeneous version of our models would have estimated R_0 between 2.4 and 3.3, in line with other studies[27]. Estimates for England suggest lower baseline R_0 and lower CV in comparison with the other study countries (Belgium, Portugal and Spain). The net effect is a slightly higher HIT in England which nevertheless we estimate around 20%. The lowest HIT, at less than 10%, is estimated in Portugal, with higher R_0 and higher CV. NPIs reveal less impact under variable susceptibility (48-69%), followed by variable connectivity (58-80%), and finally appear to inflate and agree with Flaxman et al[27] when homogeneity assumptions are made (65-89%), although this does not affect the HIT which relates to pre-pandemic societies.

More informative than reading these numbers, however, is to look at simulated projections for daily new cases over future months (Figures 1 and 2). In all four countries considered here we foresee HIT being achieved between July and October and the COVID-19 epidemic being mostly resolved by the end of 2020. Looking back, we conclude that NPIs had a crucial role in halting the growth of the initial wave between February and April. Although the most extreme lockdown strategies may not be sustainable for longer than a month or two, they proved effective at preventing overshoot, keeping cases within health system capacities, and may have done so without impairing the development of herd immunity.

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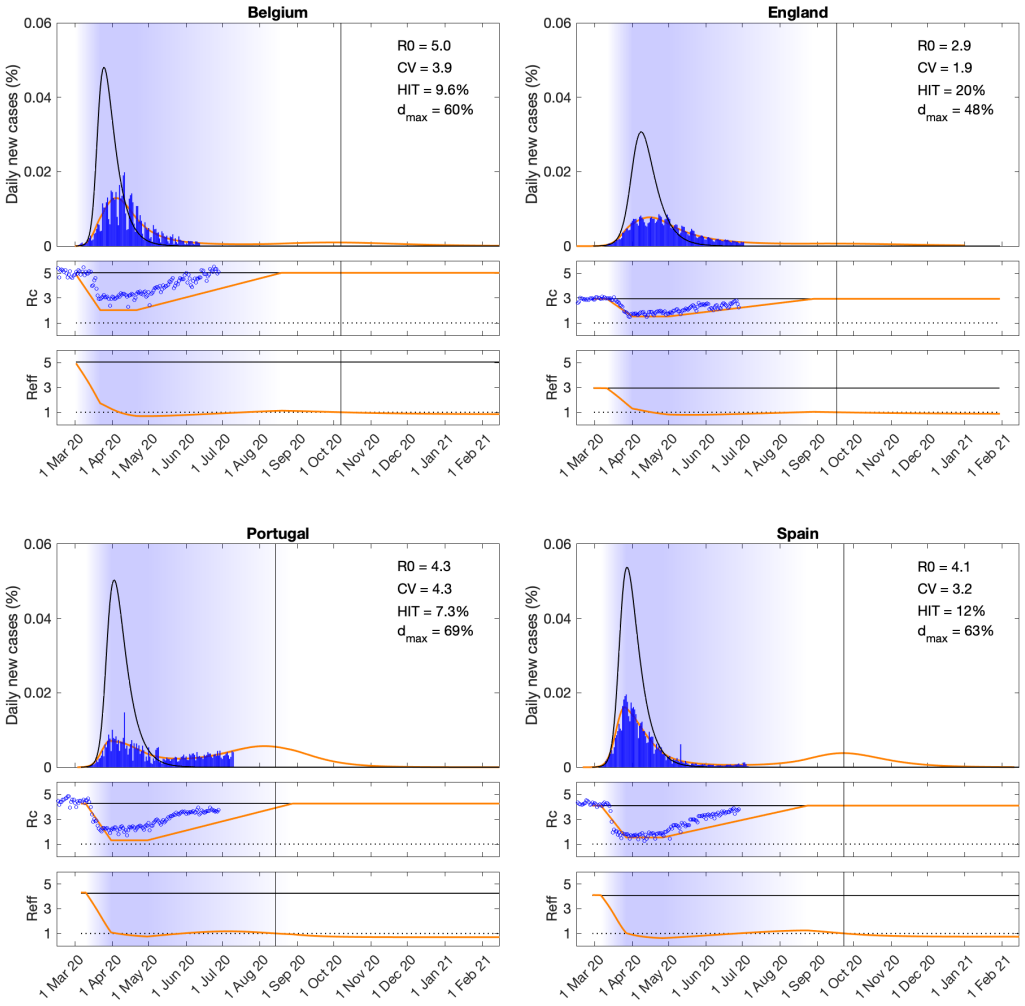

Fig. 1| SARS-CoV-2 transmission with individual variation in susceptibility to infection. Suppressed wave and subsequent dynamics in Belgium, England, Portugal and Spain (orange). Estimated epidemic in the absence of interventions revealing overshoot (black). Blue bars are daily new cases. Basic (R_0) and effective ($R_{eff} = \{\int \lambda(x)x[S(x) + \sigma R(x)]\,dx / \int \rho E(x) + I(x)\,dx\}\{\rho/\delta + 1/\gamma\}$) reproduction numbers are displayed on shallow panels underneath the main plots. Blue shades represent social distancing (intensity reflected in R_0 trends and shade density). Susceptibility factors implemented as gamma distributions. Consensus parameter values (Methods): $\delta = 1/4$ per day; $\gamma = 1/4$ per day; and $\rho = 0.5$. Fraction of infected individuals identified as positive (reporting fraction): 0.06 (Belgium); 0.024 (England); 0.09 (Portugal); 0.06 (Spain). Basic reproduction number, coefficients of variation and social distancing parameters estimated by Bayesian inference as described in Methods (estimates in Extended Data Table 1). Curves represent mean model predictions from 10^4 posterior samples. Orange shades represent 95% credible intervals. Vertical lines represent the expected time when herd immunity threshold will be achieved. Circles depict independent mobility data Google[6] not used in our parameter estimation.

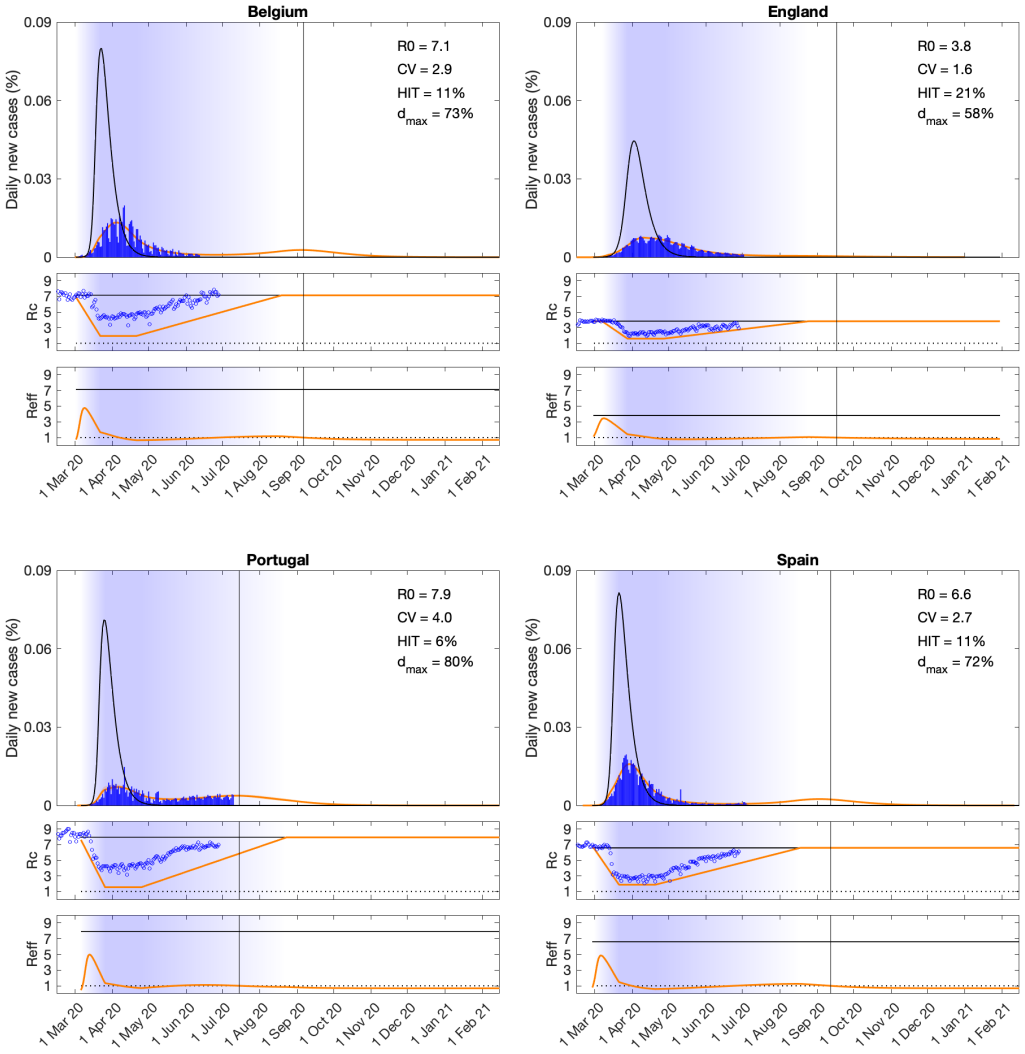

Fig. 2| SARS-CoV-2 transmission with individual variation in exposure to infection. Suppressed wave and subsequent dynamics in Belgium, England, Portugal and Spain (orange). Estimated epidemic in the absence of interventions revealing overshoot (black). Blue bars are daily new cases. Basic (R_0) and effective ($R_{eff} = \{\int \lambda(x)x[S(x) + \sigma R(x)]\, dx / \int \rho E(x) + I(x)\, dx\}\{\rho/\delta + 1/\gamma\}$) reproduction numbers are displayed on shallow panels underneath the main plots. Blue shades represent social distancing (intensity reflected in R_0 trends and shade density). Exposure factors implemented as gamma distributions. Consensus parameter values (Methods): $\delta = 1/4$ per day; $\gamma = 1/4$ per day; and $\rho = 0.5$. Fraction of infected individuals identified as positive (reporting fraction): 0.06 (Belgium); 0.024 (England); 0.09 (Portugal); 0.06 (Spain). Basic reproduction number, coefficients of variation and social distancing parameters estimated by Bayesian inference as described in Methods (estimates in Extended Data Table 2). Curves represent mean model predictions from 10^4 posterior samples. Orange shades represent 95% credible intervals. Vertical lines represent the expected time when herd immunity threshold will be achieved. Circles depict independent mobility data Google[6] not used in our parameter estimation.

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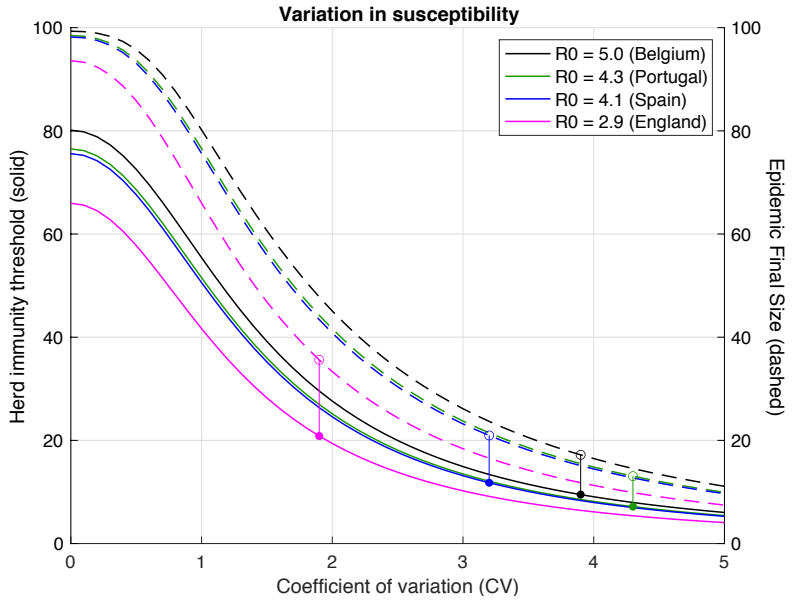

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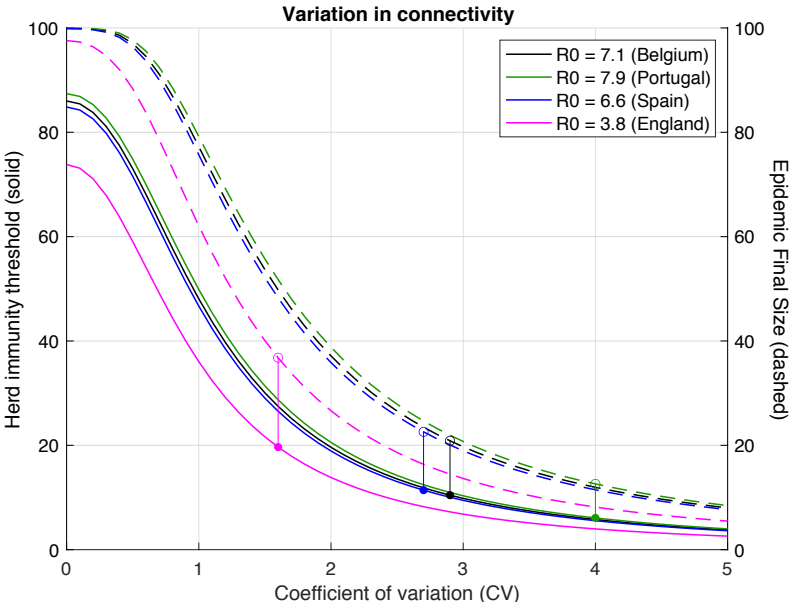

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388 **Fig. 3| Herd immunity threshold with gamma-distributed susceptibility or**
389 **exposure to infection.** Curves generated with the SEIR model (Equation 1-4)
390 assuming values of R_0 estimated for the study countries (Extended Data Tables 1 and
391 2) assuming gamma-distributed: susceptibility (top); connectivity (bottom). Herd
392 immunity thresholds (solid curves) are calculated according to the formula $1 -$
393 $(1/R_0)^{1/(1+CV^2)}$ for heterogeneous susceptibility and $1 - (1/R_0)^{1/(1+2CV^2)}$ for
394 heterogeneous connectivity. Final sizes of the corresponding unmitigated epidemics
395 are also shown (dashed).

METHODS

Model structure and underlying assumptions

The model presented here is a differential equation SEIR model, where susceptible individuals become exposed at a rate that depends on their susceptibility, the number of potentially infectious contacts they engage in, and the total number of infectious people in the population per time unit. Upon exposure, individuals enter an asymptomatic incubation phase, during which they slowly become infectious[29-32]. Thus, infectivity of exposed individuals is made to be $1/2$ of that of infectious ones ($\rho = 0.5$). After a few days, individuals develop symptoms – on average 4 days after the exposure to the virus ($\delta = 1/4$) – and thus become fully infectious[33-35]. They recover, i.e., they are no longer infectious 4 days after that ($\gamma = 1/4$), on average[36].

Efficacy of acquired immunity

We conducted the core of our analysis under the assumption that no reinfection occurs after recovery due to acquired immunity ($\sigma = 0$). To analyse the sensitivity of these results to leakage in immune response ($\sigma > 0$) we calculated herd immunity thresholds (HIT) as a function of coefficients of variation (CV) for different values of σ. The results displayed in Extended Data Figure 2 confirm the expectation that as the efficacy of acquired immunity decreases (σ increases) larger percentages of the population are infected before herd immunity is reached. Less intuitive is that there is an upper bound for how much it is reasonable to increase σ before the system enters a qualitatively different regime – the reinfection threshold[10-11] ($\sigma = 1/R_0$) – above which infection becomes stably endemic and the notion of herd immunity threshold

no longer applies. Respiratory viruses are typically associated with epidemics dynamics below the reinfection threshold.

Effective reproduction number

The effective reproduction number (R_{eff}, also denoted by R_e or R_t by other authors) is a time-dependent quantity which we calculate as the incidence of new infections divided by the total number of active infections (affected by ρ for individuals in E) multiplied by the average duration of infection (also affected by ρ for individuals in E)

$$R_{eff}(t) = \frac{\int \lambda(x,t)x[S(x,t) + \sigma R(x,t)]\, dx}{\int \rho E(x,t) + I(x,t)\, dx} \left(\frac{\rho}{\delta} + \frac{1}{\gamma}\right). \quad (9)$$

Assortative mixing

In the main text we assumed random mixing among individuals, but human connectivity patterns are assortative due societal structures and human behaviours. To explore the sensitivity of our results to deviations from random mixing, we develop an extended formalism that allows individuals to connect preferentially with those with similar connectivity, formally $\lambda(x) = (\beta/N)(\int y\, h(y-x)[\rho E(y) + I(y)]\, dy / \int y g(y)\, dy)$, where $h(y-x)$ is a normal distribution on the difference between connectivity factors (Extended Data Figure 3).

Dynamic coefficients of variation

The formulation of the variable connectivity model in the main text assumes that coefficients of variation are constant irrespective of interventions. Social distancing has been assumed to reduce connectivity of every individual by the same factor (from

439 x to $[1-d]x$) leaving the coefficient of variation unchanged. The possibility that CV
440 might reduce with social distancing (d), causing a drop in the intensity of selection,
441 might affect our results. To study sensitivity to this type of CV dynamics, we
442 formulate an extended model where connectivity is reformulated as $(1-d)[1+$
443 $(1-d)(x-1)]$, and whose CV decreases with social distancing (Extended Data
444 Figure 4). This does not change the way the model is written but special care is
445 needed in analysis and interpretation to account for the new dynamics. The basic
446 reproduction number, in particular, depends explicitly on a CV which is now
447 dependent on social distancing

448 $$R_0 = [1 + CV(d)^2]\beta\left(\frac{\rho}{\delta}+\frac{1}{\gamma}\right), \quad (10)$$

449 which is noticeable in the curvilinear shape of the controlled R_0 (R_c) trajectories
450 (Extended Data Figure 5).

451 *Non-pharmaceutical interventions*

452 We implemented non-pharmaceutical interventions (NPI) as a gradual decrease in
453 viral transmissibility in the population and thus a lowering of the controlled and
454 effective reproduction numbers (R_c and R_{eff}). Once containment measures are put in
455 place in each country, we postulate it takes 21 days until the maximum effectiveness
456 of social distancing measures is reached. In the simulations presented throughout we
457 have held this condition (maximum "lockdown" efficacy) for 30 days, after which
458 period, social distancing measures are progressively relaxed, slowly returning to pre-
459 pandemic conditions. Both the implementation and relaxing of the social distancing
460 measures are imposed to be linear in this scheme.

Bayesian Inference

The model laid out above is amenable to theoretical exploration as presented in the main manuscript and provides a perfect framework for inference. Fundamentally, to be able to reproduce the inception of any epidemic, we would need to estimate when local transmission started to occur (t_0), and the pace at which individuals infected each other in the very early stages of the epidemic (R_0). All countries, to different extents and at different timepoints of the epidemic, enforced some combination of social distancing measures. To fully understand the interplay between herd immunity and the impact of NPIs, we then set out to estimate the time at which social distancing measures started to have an impact on daily incidence (t_0^d), what their maximum effectiveness (d_{max}) is, the basic reproduction number (R_0) and what the underlying variance in heterogeneity is for both susceptibility to infection and number of infectious contacts.

In order to preserve identifiability, we made two simplifying assumptions: (*i*) the fraction of infectious individuals reported as COVID-19 cases (reporting fraction) is constant throughout the study period and is comparable between countries proportionally to the number of tests performed per person; (*ii*) local transmission starts (t_0) when countries/regions report 1 case per 5 million population in one day. To calculate the reporting rates, we used the Spanish national serological survey[12] as a reference and divided the total number of reported cases up to May 11th by the estimated number of people that had been exposed to the virus. This gives us a reporting rate for Spain around 6%. Unfortunately, there are no other national serological surveys that could inform the proportion of the population infected in other countries, so we had to extrapolate the reporting rate for those. Assuming the

485 reporting rate is highly dependent on the testing effort employed in each country,
486 reflected in the number of tests per individual, we estimate the reporting rate by
487 scaling the reporting rate recorded in Spain according to the ratio of PCR tests per
488 person in other countries relative to the Spanish reference of 0.9 tests per thousand
489 people (https://ourworldindata.org/coronavirus-testing). This produced estimated case
490 reporting rates (ratio of reported cases to infections) of 9% for Portugal, 6% for
491 Belgium (and Spain) and 2.4% for England.

492 Whist national case and mortality data is easily available for most countries, more
493 spatially resolute data is difficult to find in the public domain. Thus, we restricted our
494 analysis to countries for which disaggregated regional case data was easily available.
495 We collected the data at two time points. First, we compiled all available data from
496 the day the countries started reporting COVID-19 cases to the initial collection date
497 (May 20th) and later collated available data from May 21st to July 10th.

498 Parameter estimation was performed with the software MATLAB, using PESTO
499 (Parameter EStimation Toolbox)[37], and assuming the reported case data can be
500 accurately described by a Poisson process. We first fixed the beginning of local
501 transmission (parameter t_0) in each data series as the day in which reported cases
502 surpassed 1 in 5 million individuals. Next, we optimized the model for the set of
503 parameters $\theta = \{R_0, CV, t_0^d, d_{max}\}$ by maximizing the logarithm of the likelihood
504 (LL) (Equation 11) of observing the daily reported number of cases in each country
505 $D = \{(k, \tilde{y}_k)\}_{k=0}^{n}$:

$$LL(\theta|D) = -\sum_{k=1}^{n} y(k,\theta) + \left(\sum_{k=1}^{n} \tilde{y}(k)\ \ln\left(y(k,\theta)\right)\right) - ln\left(\prod_{k=1}^{n} \tilde{y}(k)!\right) \quad (11)$$

in which $y(k,\theta)$ is the simulated model output number of COVID-19 cases at day k (with respect to t_0), and n is the total number of days included in the analysis for each country.

When fitting the model to disaggregated data, we follow the procedure outlined above and estimate region-specific R_0 and CV, with common t_0^d and d_{max}. To ensure that the estimated maximum is a global maximum, we performed 50 multi-starts optimizations, and selected the combination of parameters resulting in the maximal Loglikelihood as a starting point for 10^4 Markov Chain Monte-Carlo iterations. From the resulting posterior distributions, we extract the median estimates for each parameter and the respective 95% credible intervals for the set of parameters $\theta = \{R_0, CV, t_0^d, d_{max}\}$. We used uniformly distributed priors with ranges {1-9, 0.0025-8,1-60, 0-0.7}.

This fitting procedure was applied to 4 countries (Belgium, England, Portugal and Spain) for both the national and disaggregated case data series and repeated for each of the 4 model variants considered here (homogeneous, heterogeneous susceptibility, heterogeneous connectivity with constant CV, and heterogeneous connectivity with CV reducing in proportion to social distancing). In the fitting procedures using sub-national data, we assumed regions had the same start date for interventions that mitigate transmission (t_0^d), and that these measures produced the same maximum impact on transmission (d_{max}) everywhere. Thus, the only region-specific parameters to be estimated are $\{R_{0_i}, CV_i\}$. Parameter estimates obtained from each of the model variants are displayed in Extended Data Table 1 (heterogeneity in susceptibility), Extended Data Table 2 (heterogeneity in connectivity with constant CV), Extended Data Table 3 (heterogeneity in connectivity with dynamic CV) and Extended Data

Table 4 (homogeneous model), are comparable to those obtained in other studies[27,38-43]. Finally, we apply the Akaike information criterion (AIC) for each estimation procedure to inform on the quality of each model's fit to the datasets of reported cases (Extended Data Table 5). In all cases, heterogeneous models are preferred over the homogeneous approximation. Homogeneous models systematically fail to fit the maintenance of low numbers of cases after the relaxation of social distancing measures in many countries and regions (images not shown). The three heterogeneous models are roughly equally well supported by the data used in this study. Further research should complement this with discriminatory data types and hybrid models to enable the integration of different forms of individual variation.

Data and code availability

Datasets are publicly available at the respective national ministry of health websites (44-48). Core models implemented in MATLAB available from: https://github.com/mgmgomes1/covid

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590

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596

597 **Author contributions**

598 M.G.M.G. conceived the study. R.A. and R.M.C. and M.G.M.G. performed the
599 analyses. All authors interpreted the data and wrote the paper.

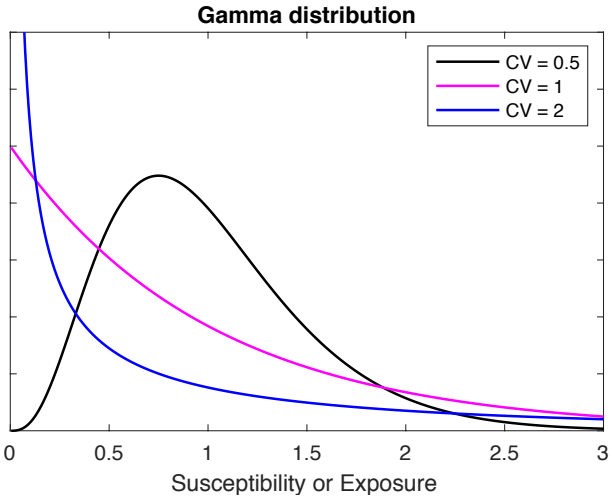
600

601 **Competing interests**

602 The authors declare no competing interests.

603

604



605

606 **Extended Data Fig. 1| Distributions used for variable susceptibility and**
607 **connectivity.** Gamma distribution probability density functions with mean 1 and
608 various coefficients of variation: $\left[x^{1/CV^2-1}e^{-x/CV^2}\right]/\left[\Gamma(1/CV^2)CV^{(2/CV^2)}\right]$, where Γ
609 is the Gamma function. For numerical implementations we discretized gamma
610 distributions into N bins, calculated the susceptibility or connectivity factor as well as
611 the fraction of the population in each bin, and derived the associated $4N$-dimensional
612 systems of ordinary differential equations.
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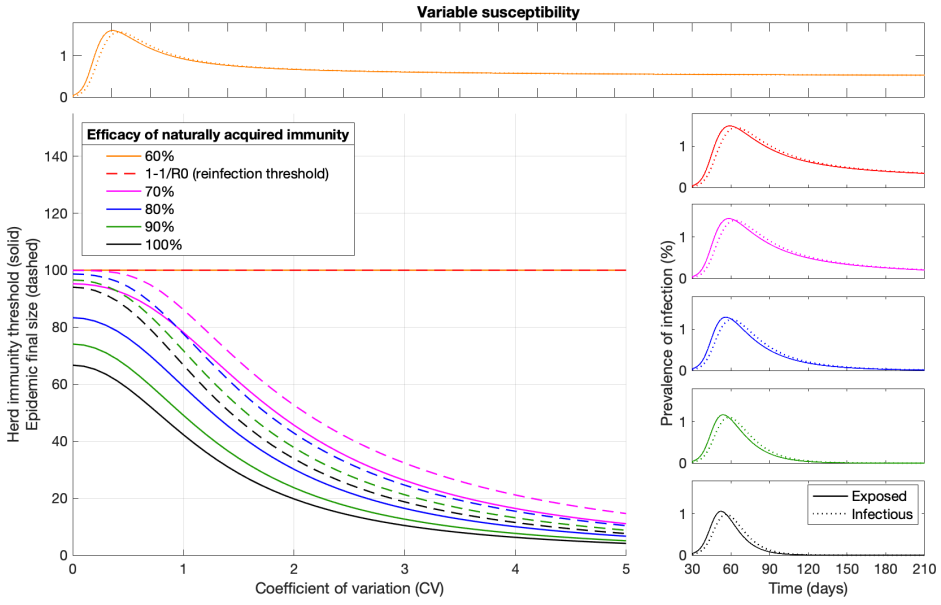

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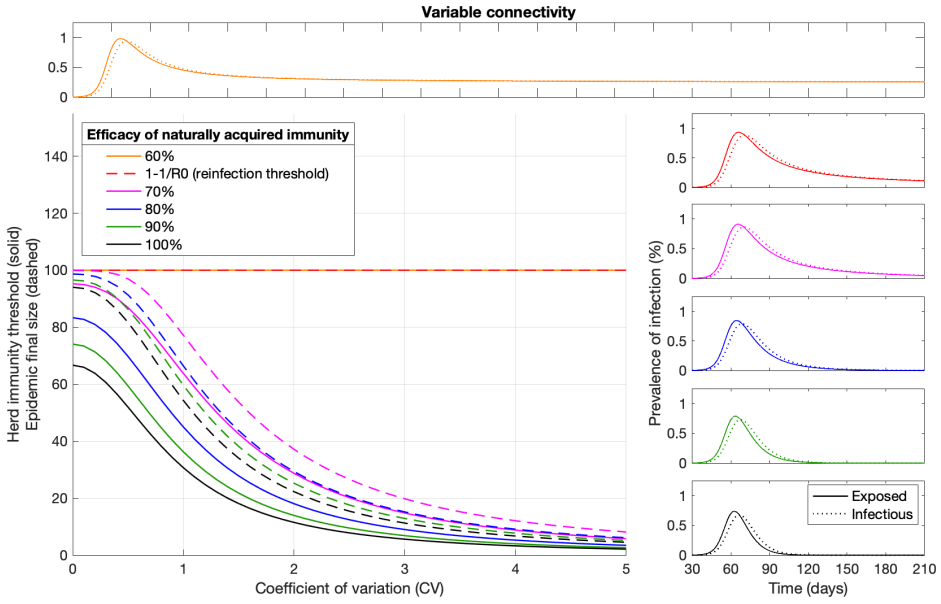

616

617 **Extended Data Fig. 2| Herd immunity threshold and epidemic final size with**
618 **reinfection.** Curves in the main panels generated with the SEIR model (Equation 1-4)
619 assuming $R_0 = 3$ and gamma-distributed susceptibility (top) or connectivity (bottom).
620 Efficacy of acquired immunity is captured by a reinfection parameter σ, potentially
621 ranging between $\sigma = 0$ (100% efficacy) and $\sigma = 1$ (0 efficacy). This illustration
622 depicts final sizes of unmitigated epidemics and associated HIT curves for 6 values of
623 σ: $\sigma = 0$ (black); $\sigma = 0.1$ (green); $\sigma = 0.2$ (blue); $\sigma = 0.3$ (magenta); $\sigma = 1/3$ (red);
624 and $\sigma = 0.4$ (orange);. Above $\sigma = 1/R_0$ (reinfection threshold (Gomes et al 2004;
625 2016)) the infection becomes stably endemic and there is no herd immunity threshold.
626 Representative epidemics of the regime $\sigma \leq 1/R_0$ are shown on the right while the
627 regime $\sigma > 1/R_0$ is illustrated on top. All depicted dynamics are based on the
628 rightmost CVs represented on the main panel.
629

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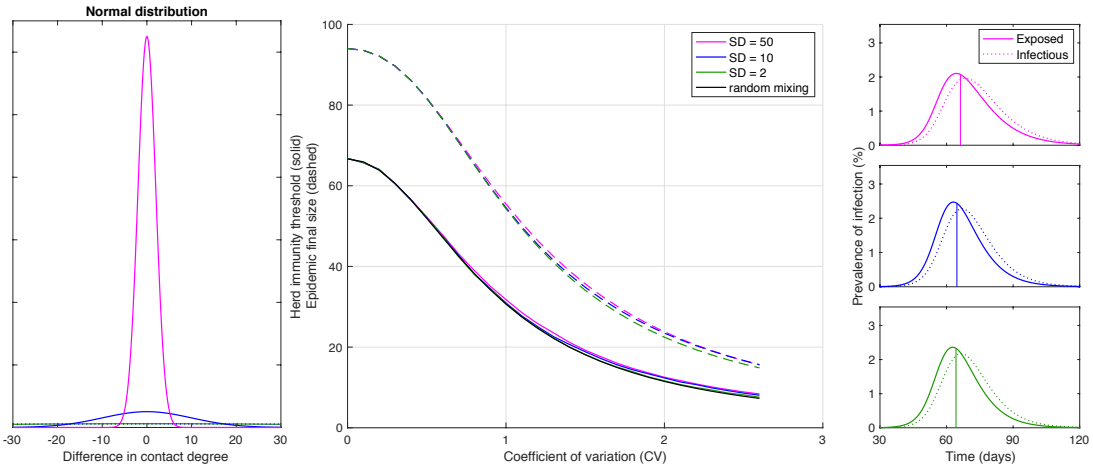
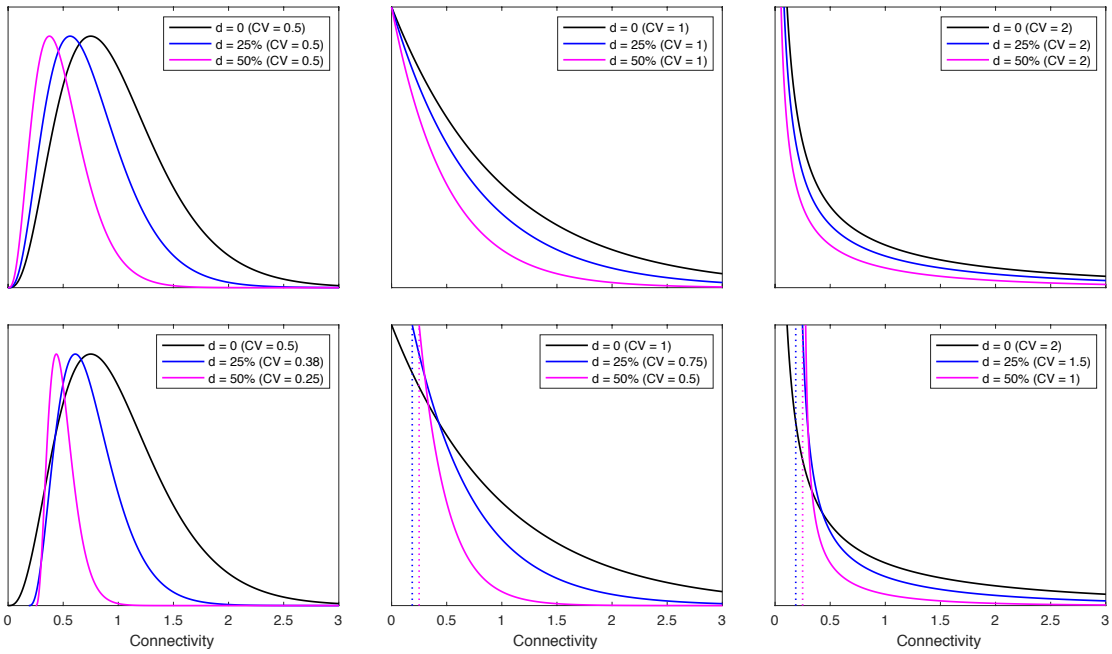
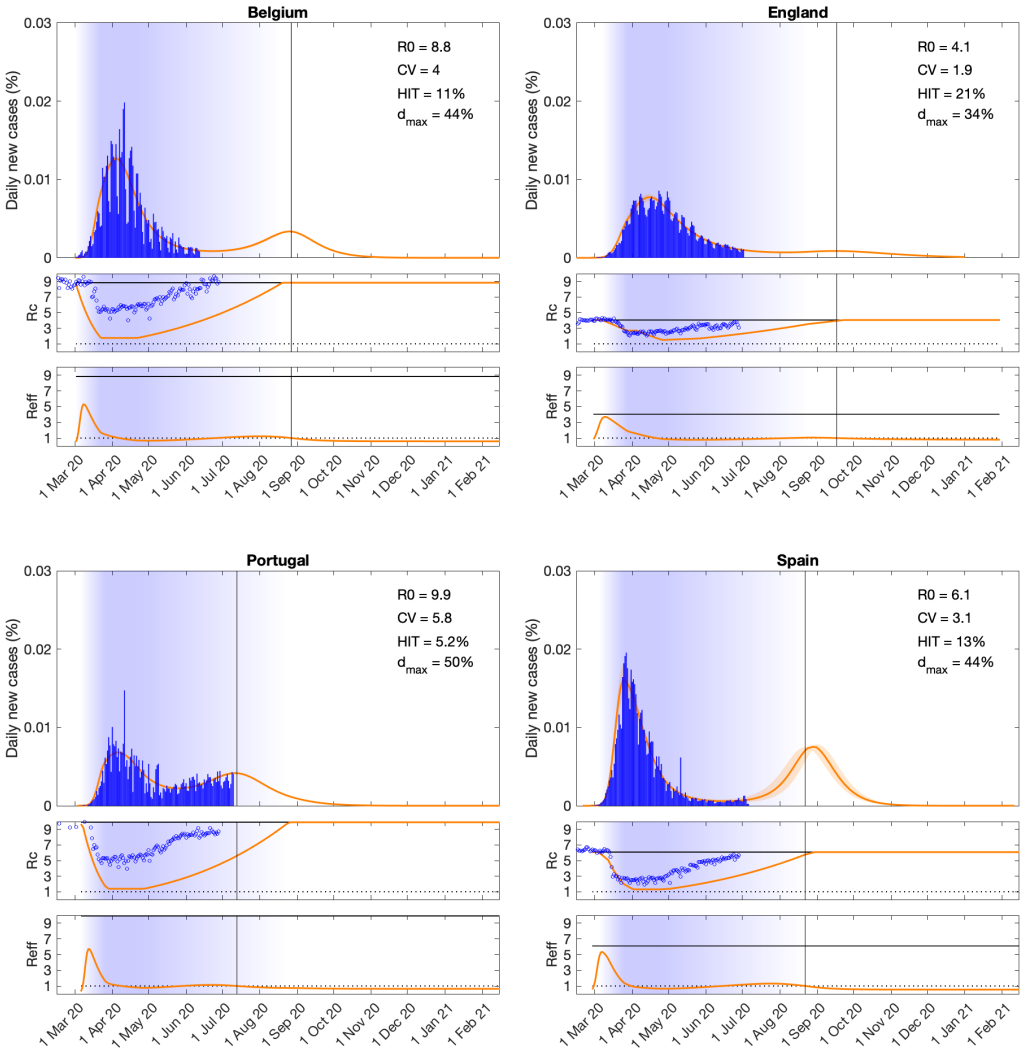


631

632 **Extended Data Fig. 3| Herd immunity threshold and epidemic final size with**
633 **gamma-distributed exposure to infection and assortative mixing.** Curves in
634 central panel generated with the SEIR model (Equation 1-4) assuming $R_0 = 3$ and
635 gamma-distributed connectivity. Assortative mixing is implemented by imposing a
636 normal distribution for contact preferences such that individuals contact preferentially
637 with those with the similar contact degree (left). This illustration used normal
638 distributions with standard deviation $SD = 50$ (green); $SD = 10$ (blue); and $SD = 2$
639 (magenta). More assortative mixing leads to more skewed epidemics. Herd immunity
640 thresholds were calculated numerically as the percentage of the population no longer
641 susceptible when new outbreaks are effectively prevented (approximately when the
642 exposed fraction crosses the peak in the absence of mitigation). Final sizes of the
643 corresponding unmitigated epidemics are also shown. Representative epidemics are
644 depicted on the right based on the rightmost CVs represented on the main panel (with
645 vertical lines marking the point when herd immunity is achieved).
646



Extended Data Fig. 4| Connectivity distributions with reducing coefficient of variation in proportion to social distancing. Individual variation in connectivity is originally implemented as a gamma distribution of mean 1 parameterised by the coefficient of variation (CV) (black). Social distancing is initially implemented as a reduction in connectivity by the same factor to every individual, from x to $(1-d)x$ (top panels). Sensitivity of the results to the possibility that CV might reduce with social distancing with replicated the analyses with a model connectivity is reformulated as $(1-d)[1+(1-d)(x-1)]$ (bottom panels).



Extended Data Fig. 5| SARS-CoV-2 transmission with individual variation in exposure reduced by social distancing. Suppressed wave and subsequent dynamics in Belgium, England, Portugal and Spain. Blue bars are daily new cases. Basic (R_0) and effective ($R_{eff} = \{\int \lambda(x)x[S(x) + \sigma R(x)]\, dx / \int \rho E(x) + I(x)\, dx\}\{\rho/\delta + 1/\gamma\}$) reproduction numbers are displayed on shallow panels underneath the main plots. Blue shades represent social distancing (intensity reflected in R_0 trends and shade density). Exposure factors implemented as gamma distributions. Consensus parameter values (Methods): $\delta = 1/4$ per day; $\gamma = 1/4$ per day; and $\rho = 0.5$. Fraction of infected individuals identified as positive (reporting fraction): 0.06 (Belgium); 0.024 (England); 0.09 (Portugal); 0.06 (Spain). Basic reproduction number, coefficients of variation and social distancing parameters estimated by Bayesian inference as described in Methods (estimates in Extended Data Table 3). Curves represent mean model predictions from 10^4 posterior samples. Orange shades represent 95% credible intervals. Vertical lines represent the expected time when herd immunity threshold will be achieved. Circles depict independent mobility data (Google 2020) not used in our parameter estimation.

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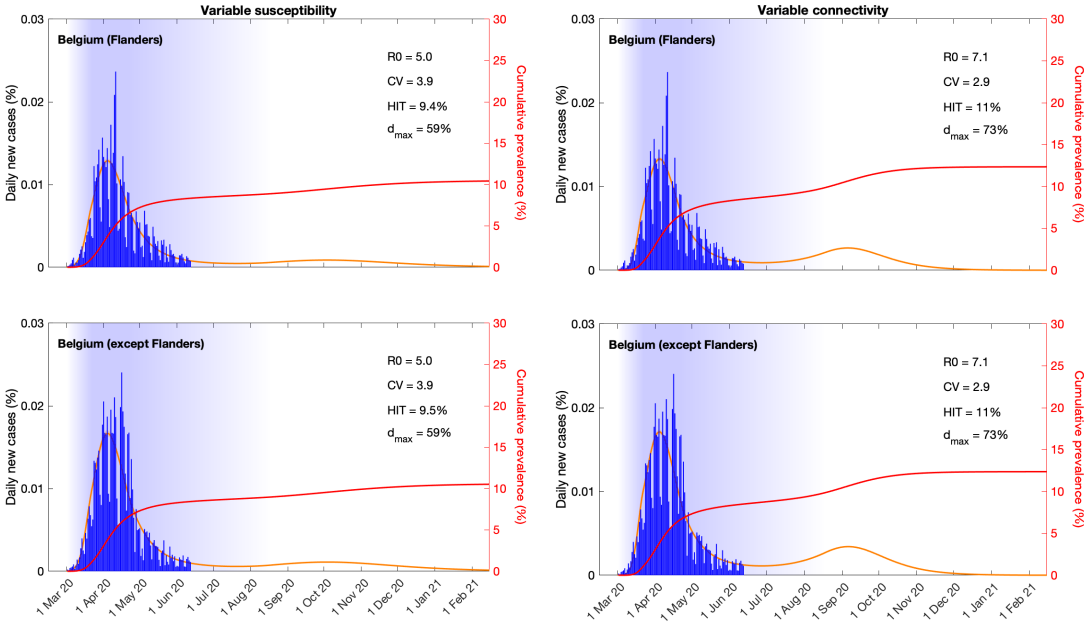

678 **Extended Data Fig. 6| SARS-CoV-2 transmission at subnational levels in Belgium.**
679 Suppressed wave and subsequent dynamics in Flanders and the rest of Belgium, with
680 individual variation in susceptibility (left) or exposure (right). Blue bars are daily new
681 cases. Shades represent social distancing (intensity reflected in shade density).
682 Susceptibility or exposure factors implemented as gamma distributions. Consensus
683 parameter values (Methods): $\delta = 1/4$ per day; $\gamma = 1/4$ per day; and $\rho = 0.5$.
684 Fraction of infected individuals identified as positive (reporting fraction): 0.06. Basic
685 reproduction number, coefficients of variation and social distancing parameters
686 estimated by Bayesian inference as described in Methods (estimates in Extended Data
687 Table 1 and 2). Curves represent mean model predictions from 10^4 posterior samples.
688 Orange shades represent 95% credible intervals. Red curves represent cumulative
689 infected percentages.
690

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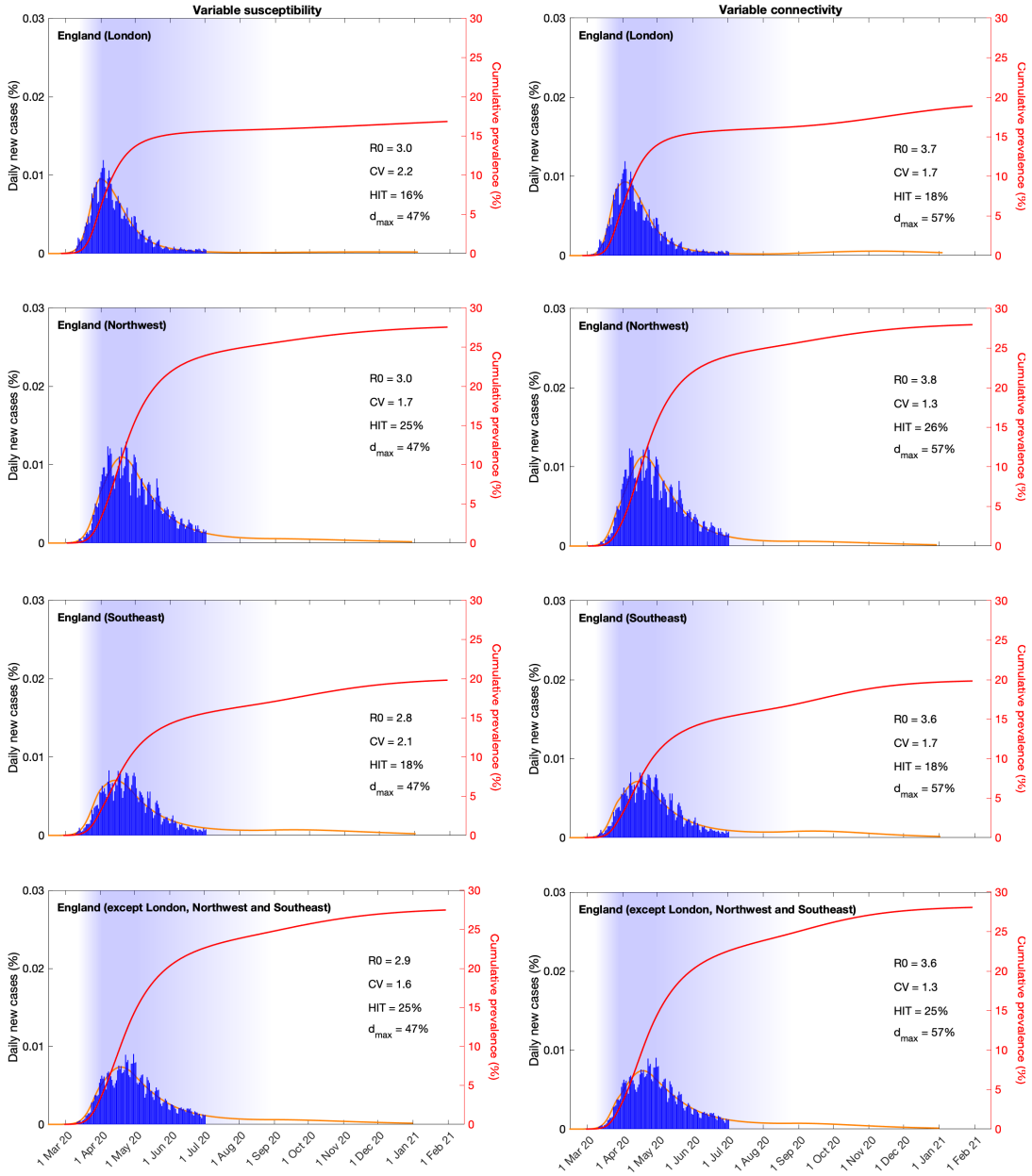
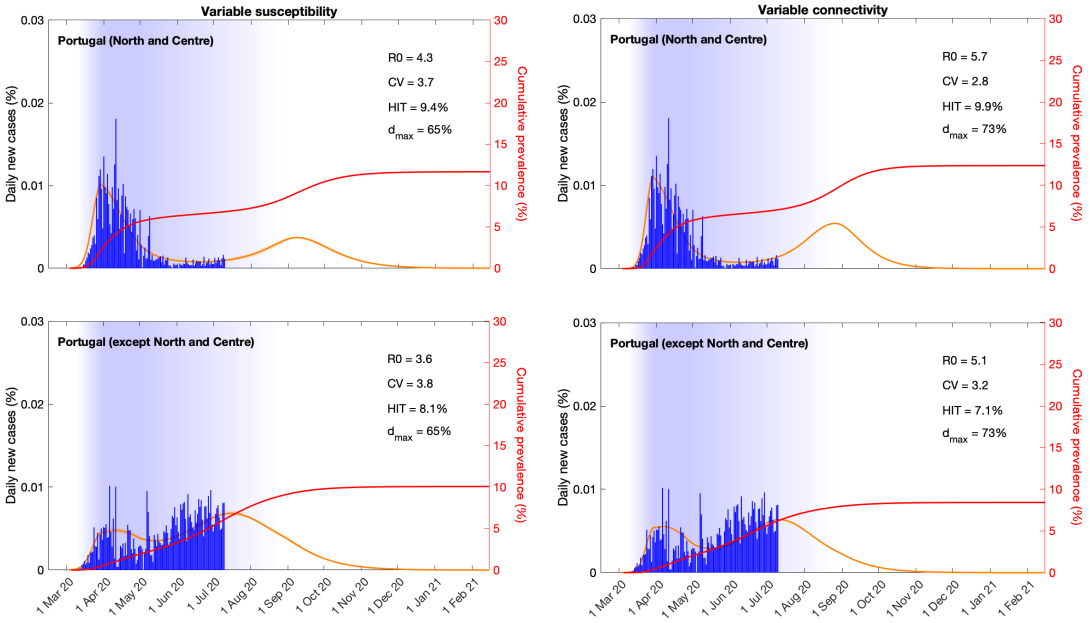


692 **Extended Data Fig. 7| SARS-CoV-2 transmission at subnational levels in England.**
693 Suppressed wave and subsequent dynamics in London, Northwest, Southeast and the
694 rest of England, with individual variation in susceptibility (left) or exposure (right).
695 Blue bars are daily new cases. Shades represent social distancing (intensity reflected
696 in shade density). Susceptibility or exposure factors implemented as gamma
697 distributions. Consensus parameter values (Methods): $\delta = 1/4$ per day; $\gamma = 1/4$ per
698 day; and $\rho = 0.5$. Fraction of infected individuals identified as positive (reporting
699 fraction): 0.024. Basic reproduction number, coefficients of variation and social
700 distancing parameters estimated by Bayesian inference as described in
701 Methods(estimates in Extended Data Table 1 and 2). Curves represent mean model
702 predictions from 10^4 posterior samples. Orange shades represent 95% credible
703 intervals. Red curves represent cumulative infected percentages.
704



Extended Data Fig. 8| SARS-CoV-2 transmission at subnational levels in Portugal. Suppressed wave and subsequent dynamics in the North and Centre regions versus the rest of Portugal, with individual variation in susceptibility (left) or exposure (right). Blue bars are daily new cases. Shades represent social distancing (intensity reflected in shade density). Susceptibility or exposure factors implemented as gamma distributions. Consensus parameter values (Methods): $\delta = 1/4$ per day; $\gamma = 1/4$ per day; and $\rho = 0.5$. Fraction of infected individuals identified as positive (reporting fraction): 0.09. Basic reproduction number, coefficients of variation and social distancing parameters estimated by Bayesian inference as described in Methods (estimates in Extended Data Table 1 and 2). Curves represent mean model predictions from 10^4 posterior samples. Orange shades represent 95% credible intervals. Red curves represent cumulative infected percentages.

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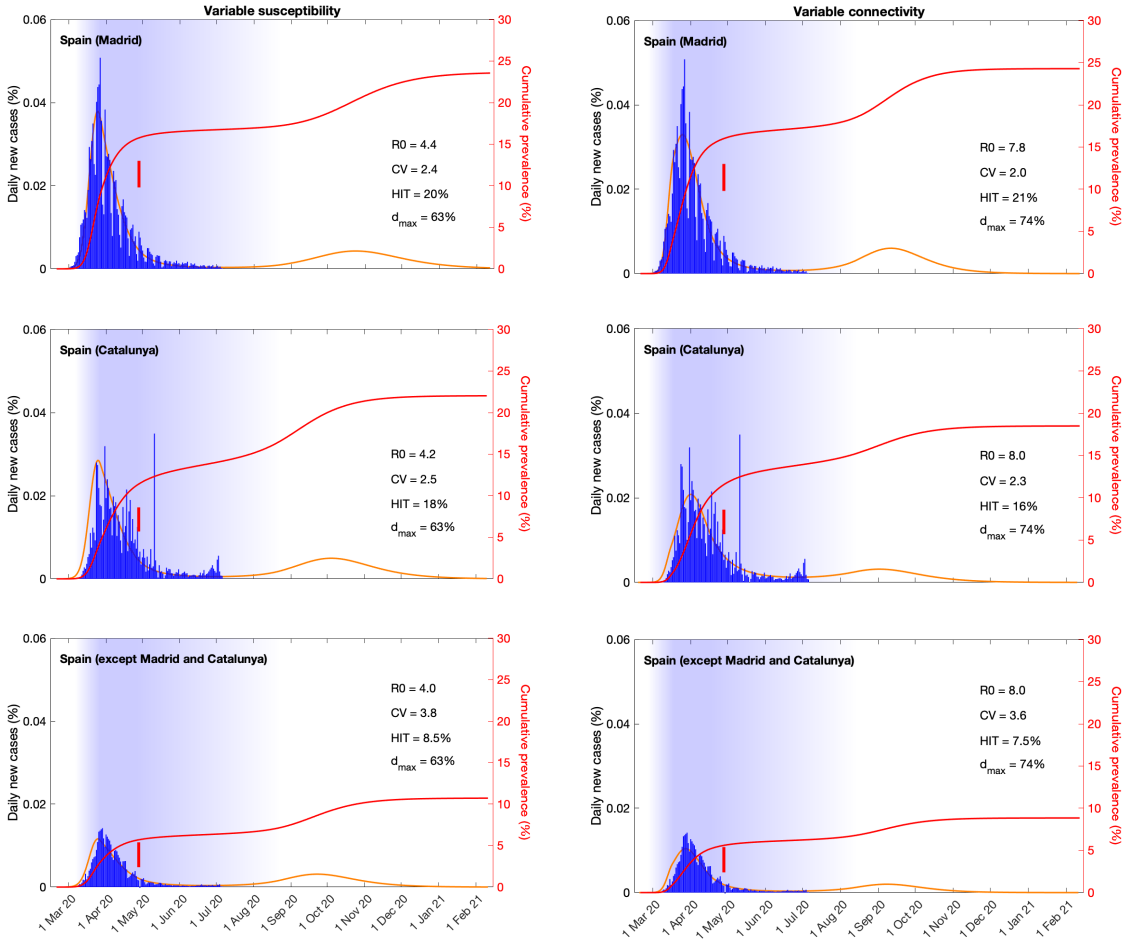

720 **Extended Data Fig. 9| SARS-CoV-2 transmission at subnational levels in Spain.**
721 Suppressed wave and subsequent dynamics in Madrid, Catalunya and the rest of
722 Spain, with individual variation in susceptibility (left) or exposure (right). Blue bars
723 are daily new cases. Shades represent social distancing (intensity reflected in shade
724 density). Susceptibility or exposure factors implemented as gamma distributions.
725 Consensus parameter values (Methods): $\delta = 1/4$ per day; $\gamma = 1/4$ per day; and $\rho =$
726 0.5. Fraction of infected individuals identified as positive (reporting fraction): 0.06.
727 Basic reproduction number, coefficients of variation and social distancing parameters
728 estimated by Bayesian inference as described in Methods (estimates in Extended Data
729 Table 1 and 2). Curves represent mean model predictions from 10^4 posterior samples.
730 Orange shades represent 95% credible intervals. Red curves represent cumulative
731 infected percentages and vertical red segments mark seroprevalences (95% CI)
732 according to a recent study[12].
733

Extended Data Table 1| Estimated parameters for heterogeneous susceptibility model. Estimates generated from model fit to the national datasets are in the grey shaded rows. The remaining rows provide the region-specific estimates. Best parameter estimates are presented as a bold median bounded by the lower and upper ends for the 95% credible interval. Model runs are initiated on the day (t_0) when reported cases surpassed 1 in 5 million individuals: Belgium (day 1); England (day 29); Portugal (day 3); Spain (day 8).

Country/Region	R_0			CV			$1-d_{max}$			t_0^d		
Belgium	4.99	**5.03**	5.06	3.87	**3.88**	3.91	0.40	**0.40**	0.41	1.00	**1.01**	1.12
Flanders	4.96	**5.00**	5.02	3.89	**3.91**	3.93	0.41	**0.41**	0.41	1.00	**1.02**	1.15
Rest	4.97	**5.01**	5.03	3.87	**3.89**	3.91						
Portugal	4.23	**4.26**	4.31	4.22	**4.26**	4.30	0.30	**0.31**	0.31	7.37	**7.71**	7.91
North/Centre	3.54	**3.58**	3.61	3.72	**3.76**	3.79	0.34	**0.35**	0.35	7.51	**7.73**	8.00
Rest	4.27	**4.32**	4.36	3.69	**3.72**	3.74						
Spain	4.08	**4.10**	4.11	3.20	**3.21**	3.22	0.37	**0.37**	0.37	16.02	**16.13**	16.23
Madrid	4.38	**4.39**	4.39	2.37	**2.37**	2.38	0.37	**0.37**	0.37	16.40	**16.41**	16.44
Catalunya	4.20	**4.21**	4.21	2.49	**2.50**	2.50						
Rest	3.96	**3.97**	3.97	3.80	**3.81**	3.82						
England	2.93	**2.94**	2.95	1.94	**1.94**	1.95	0.52	**0.52**	0.52	40.56	**40.69**	40.80
London	2.95	**2.96**	2.96	2.24	**2.24**	2.24	0.52	**0.53**	0.53	41.35	**41.51**	41.64
NorthWest	3.03	**3.04**	3.05	1.66	**1.67**	1.68						
SouthEast	2.80	**2.81**	2.82	2.07	**2.07**	2.07						
Rest	2.88	**2.88**	2.89	1.64	**1.64**	1.65						

Extended Data Table 2| Estimated parameters for heterogeneous connectivity model (constant CV). Estimates generated from model fit to the national datasets are in the grey shaded rows. The remaining rows provide the region-specific estimates. Best parameter estimates are presented as a bold median bounded by the lower and upper ends for the 95% credible interval. Model runs are initiated on the day (t_0) when reported cases surpassed 1 in 5 million individuals: Belgium (day 1); England (day 29); Portugal (day 3); Spain (day 8).

Country/Region	R_0			CV			$1-d_{max}$			t_0^d		
Belgium	7.12	**7.14**	7.17	2.86	**2.87**	2.88	0.27	**0.27**	0.27	1.00	**1.01**	1.04
Flanders	7.09	**7.11**	7.13	2.86	**2.87**	2.89	0.27	**0.27**	0.28	1.00	**1.01**	1.03
Rest	7.11	**7.13**	7.15	2.86	**2.87**	2.89						
Portugal	7.76	**7.94**	8.14	4.01	**4.04**	4.09	0.19	**0.20**	0.20	2.67	**2.98**	3.27
North/Centre	5.06	**5.08**	5.09	3.24	**3.24**	3.24	0.25	**0.25**	0.25	7.21	**7.22**	7.24
Rest	5.68	**5.69**	5.69	2.79	**2.81**	2.83						
Spain	6.59	**6.60**	6.60	2.73	**2.73**	2.73	0.28	**0.28**	0.28	10.00	**10.01**	10.02
Madrid	7.81	**7.83**	8.82	1.98	**1.99**	2.06	0.24	**0.26**	0.26	5.38	**7.02**	7.06
Catalunya	8.00	**8.02**	9.00	2.33	**2.34**	2.43						
Rest	7.97	**7.99**	8.96	3.58	**3.59**	3.72						
England	3.81	**3.82**	3.83	1.55	**1.55**	1.55	0.42	**0.42**	0.42	36.45	**36.52**	36.61
London	3.70	**3.70**	3.71	1.69	**1.69**	1.70	0.43	**0.43**	0.43	37.50	**37.52**	37.55
NorthWest	3.83	**3.83**	3.84	1.32	**1.32**	1.32						
SouthEast	3.58	**3.59**	3.59	1.66	**1.67**	1.68						
Rest	3.60	**3.60**	3.61	1.30	**1.31**	1.31						

Extended Data Table 3| Estimated parameters for heterogeneous connectivity model (dynamic CV). Estimates generated from model fit to the national datasets are in the grey shaded rows. The remaining rows provide the region-specific estimates. Best parameter estimates are presented as a bold median bounded by the lower and upper ends for the 95% credible interval. Model runs are initiated on the day (t_0) when reported cases surpassed 1 in 5 million individuals: Belgium (day 1); England (day 29); Portugal (day 3); Spain (day 8).

Country/Region	R_0			CV			$1-d_{max}$			t_0^d		
Belgium	8.79	**8.84**	8.88	3.98	**4.00**	4.02	0.56	**0.56**	0.56	1.00	**1.01**	1.05
Flanders	8.78	**8.82**	8.86	3.98	**4.00**	4.02	0.56	**0.56**	0.56	1.00	**1.01**	1.04
Rest	8.82	**8.86**	8.89	3.98	**4.00**	4.02						
Portugal	9.86	**9.92**	9.95	5.73	**5.77**	5.80	0.50	**0.50**	0.50	3.00	**3.01**	3.07
North/Centre	6.65	**6.72**	6.80	3.75	**3.78**	3.81	0.56	**0.57**	0.57	5.84	**6.02**	6.19
Rest	5.98	**6.05**	6.13	4.09	**4.14**	4.19						
Spain	5.97	**6.09**	6.10	3.04	**3.09**	3.10	0.56	**0.56**	0.56	14.46	**14.50**	14.89
Madrid	6.19	**6.20**	6.21	2.43	**2.43**	2.44	0.57	**0.57**	0.57	13.80	**13.81**	13.83
Catalunya	6.30	**6.32**	6.33	2.61	**2.62**	2.62						
Rest	6.34	**6.35**	6.36	3.80	**3.81**	3.82						
England	4.01	**4.05**	4.09	1.90	**1.92**	1.93	0.66	**0.66**	0.66	36.79	**37.03**	37.29
London	3.78	**3.79**	3.80	1.99	**2.00**	2.01	0.67	**0.67**	0.67	39.06	**39.20**	39.28
NorthWest	3.91	**3.92**	3.94	1.64	**1.65**	1.66						
SouthEast	3.67	**3.69**	3.70	1.89	**1.89**	1.90						
Rest	3.64	**3.65**	3.66	1.58	**1.58**	1.59						

Extended Data Table 4| Estimated parameters for the homogenous model.
Estimates generated from model fit to the national datasets are in the grey shaded rows. The remaining rows provide the region-specific estimates. Best parameter estimates are presented as a bold median bounded by the lower and upper ends for the 95% credible interval. Model runs are initiated on the day (t_0) when reported cases surpassed 1 in 5 million individuals: Belgium (day 1); England (day 29); Portugal (day 3); Spain (day 8).

Country/Region	R_0			$1-d_{max}$			t_0^d		
Belgium	3.298	**3.301**	3.310	0.208	**0.209**	0.210	16.299	**16.354**	16.357
Flanders	3.235	**3.239**	3.308	0.208	**0.212**	0.213	16.324	**17.039**	17.064
Rest	3.235	**3.238**	3.307						
Portugal	2.900	**2.910**	2.914	0.242	**0.243**	0.244	20.551	**20.622**	20.693
North/Centre	3.542	**3.578**	3.608	0.343	**0.345**	0.348	7.514	**7.725**	7.999
Rest	4.274	**4.321**	4.361						
Spain	3.028	**3.031**	3.034	0.149	**0.150**	0.150	28.329	**28.360**	28.393
Madrid	4.113	**4.116**	4.120	0.111	**0.111**	0.112	20.000	**20.000**	20.000
Catalunya	4.208	**4.214**	4.218						
Rest	3.735	**3.751**	3.752						
England	2.434	**2.435**	2.437	0.355	**0.355**	0.356	55.047	**55.070**	55.074
London	2.307	**2.308**	2.310	0.359	**0.360**	0.360	54.577	**54.578**	54.578
NorthWest	2.602	**2.603**	2.604						
SouthEast	2.368	**2.370**	2.371						
Rest	2.502	**2.503**	2.504						

Extended Data Table 5| Model selection criteria. Displays the maximum Loglikelihood obtained for each combination of model and data partitioning for each country, as well as the Akaike information criterion. Models are labelled by a sort name as follows: homog (homogenous); hetsus (heterogeneity in susceptibility); hetcon (heterogeneity in connectivity with constant CV); hetdyn (heterogeneity in connectivity with dynamic CV).

Country	*Model*	*LL*	*AIC*
Aggregate Data			
Portugal	homog	2.25E+05	-4.50E+05
	hetsus	2.30E+05	-4.59E+05
	hetcon	2.30E+05	-4.60E+05
	hetdyn	2.30E+05	-4.59E+05
Spain	homog	1.77E+06	-3.54E+06
	hetsus	1.79E+06	-3.59E+06
	hetcon	1.79E+06	-3.58E+06
	hetdyn	1.79E+06	-3.58E+06
England	homog	1.67E+06	-3.33E+06
	hetsus	1.68E+06	-3.36E+06
	hetcon	1.68E+06	-3.36E+06
	hetdyn	1.68E+06	-3.36E+06
Belgium	homog	3.30E+05	-6.60E+05
	hetsus	3.33E+05	-6.66E+05
	hetcon	3.33E+05	-6.65E+05
	hetdyn	3.33E+05	-6.66E+05
Regional Data			
Portugal	homog	3.36E+05	-6.73E+05
	hetsus	3.55E+05	-7.11E+05
	hetcon	3.55E+05	-7.10E+05
	hetdyn	3.55E+05	-7.11E+05
Spain	homog	1.87E+06	-3.75E+06
	hetsus	1.96E+06	-3.91E+06
	hetcon	1.96E+06	-3.91E+06
	hetdyn	1.47E+06	-2.95E+06
England	homog	2.59E+06	-5.18E+06
	hetsus	2.63E+06	-5.25E+06
	hetcon	2.62E+06	-5.25E+06
	hetdyn	2.62E+06	-5.25E+06
Belgium	homog	3.74E+05	-7.48E+05
	hetsus	3.78E+05	-7.56E+05
	hetcon	3.78E+05	-7.55E+05
	hetdyn	3.78E+05	-7.55E+05