Research Square

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Dynamics of the COVID-19 epidemic in Ireland under mitigation

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Abstract

Background: In Ireland and across the European Union, cases of COVID-19 continue to rise with recent increases in reported cases following a period of stability. Public health interventions continue in their attempts to control the epidemic in spite of a lack of information on the scale of silent transmission.

Methods: To tackle this challenge and the non-stationary aspect of the epidemic we used a modified SEIR stochastic model with time-varying parameters, following Brownian process. This model is coupled with Bayesian inference (PMCMC) for parameter estimation and used mainly confirmed reported hospitalized cases.

Results: Mitigation measures provided an 80% reduction in transmission between March and May 2020. By end of October our estimated seroprevalence rate was 1.1% (95% CI: 0.5%–2.8%) far from herd immunity. We estimated that the proportion of asymptomatic transmission was approximately 40% but with large uncertainty (95% CI: 14%–73%). Finally we demonstrate that the available observed confirmed cases are not reliable for any analysis owing to the fact that their reporting rate has greatly evolved.

Conclusion: We provide the first estimations of the dynamics of the COVID-19 epidemic in Ireland and its key parameters. We also quantify the effects of mitigation measures on the virus transmission before, during and after mitigation. Our results demonstrate that Ireland has significantly reduced transmission by employing mitigation measures, physical distancing and lockdown. This has to date avoided the saturation of healthcare infrastructures, flattened the epidemic curve and likely reduced mortality. However, as mitigation measures change silent transmission remain an ongoing challenge.

Background

In the last months of 2019, grouped pneumonia cases were described in China. The etiological agent of this new disease, a betacoronavirus, was identified in January and named SARS-CoV2. Meanwhile this novel coronavirus disease (COVID-19) spread rapidly from China across multiple countries worldwide. As of March 17, 2020, COVID-19 was officially declared a pandemic by the World Health Organization. COVID-19 has now spread throughout most countries causing premature loss of life and resulting in damaging social-economic impacts. At the end of September SARS-CoV-2 has infected more than 34 million people and produced more than 1 million deaths around the world [1]. Numbers of infection and mortality are predicted to continue to rise in the near future and there is likely to be second waves and future recurrence [2].

The first case in Ireland was declared on the 29th of February 2020 followed by a rapid increase in reported infections leading to a peak in daily incidence in the week of April 10th to 17th. This peak was followed by a steady decline in daily cases reported until mid-August when a slow but steady increase in cases emerged. This increase was sustained and on Friday the 18th of September as a result of this increase the capital city, Dublin, was placed on a level 3 alert with movement restrictions and various lockdown measures. On September 25th a rural region in close proximity to the border of Northern Ireland was also placed on this level 3 alert [3].

Our aim is to examine the dynamics of the COVID-19 epidemic in Ireland using public data and a simple stochastic model. As occurs with the majority of epidemics, the COVID-19 epidemic has and continues to modify greatly during its course. Taking account of the time-varying nature of the different mechanisms responsible for disease propagation is always a major challenge. To tackle this aspect, we have used a previously proposed framework [3]. This framework uses diffusion models driven by fractional Brownian motion to model time-varying parameters embedded in a stochastic modified SEIR model, coupled with Bayesian inference methods. The advantages of this approach are the possibility of (i) considering all the specific mechanisms of the transmission of the pathogen (*e.g.* asymptomatic transmission), (ii) using different datasets simultaneously, (iii) accounting for all the uncertainty associated with the data used and, most importantly (iv) following the time-evolution of some of the key model parameters. This framework allows us to follow changes in disease

transmission owing, for example, to Public Health interventions, which are of particular interest to us in the case the COVID-19 epidemic.

Materials And Methods

Data

Large uncertainties are associated with the reported number of cases of COVID-19. The lower number of reported cases is due to low detection and reporting rates, firstly because the testing capacity (RT-PCR laboratory capacity) was limited and has greatly varied during the course of this epidemic. Secondly, it is due to features of this new virus, such as transmission before the symptom onsets and important asymptomatic transmission, which results in a low fraction of infected people attending the health facilities for testing.

This suggests that hospitalized data is likely to be the most accurate COVID-19 related data. Thus we mainly focus on hospitalized data published by the Irish Public Health Authorities (Health Protection Surveillance Centre, HPSC). We have used the daily reports [5] and also a data hub launched last June [6].

We also mainly focus on incidence data to avoid all defects related to the use of cumulative data (see [7]), ie: daily hospitalized admission, daily ICU admission, daily deaths and daily hospital discharged. We also used “current bed used” both in hospital and in ICU as these are state variables of our model.

Since hospitalized data is only available from the 22th of March after the first mitigation measures (school closure) and that our aim was to model the dynamics of the epidemic before, during and after the NPI measures, we used daily incident infectious data available before the 25th of March. Nevertheless this data was associated with a low reporting rate and a large variance in the observational process used (see Inference part below).

Model

A simple model of extended stochastic Susceptible-Exposed-Infectious-Recovered (SEIR) also accounting for asymptomatic transmission and the hospital system has been developed (see eqs A1-A2). It is similar to others, which have been proposed to model and forecast the COVID-19 epidemic [8–11]. It includes the following variables: the susceptibles S, the infected non-infectious E, the infectious symptomatic I, the infectious asymptomatic A, the removed people R, and the hospital variables: hospitalized people H, people in intensive care unit ICU, hospital discharge G, and deaths at hospital D. We have also introduced Erlang-distributed stage durations (with a shape parameter equal to 2) for the E, I, A and H compartments to mimic a gamma distribution for stage duration in these compartments discounting inappropriate exponential stage durations (eqs A1). The parameters are defined in the Figure caption and in Table 1.

Table 1

Defnition of the different parameters and their priors and posteriors based on current literature [8–11] (see Fig. A1). U is for uniform distribution and tN for truncated normal distribution (tN[mean,std,limit inf,limit sup]).

Parameters	Definitions	Prior or constant value	Posterior Median, [95%CI]
$I_1(0)$	Initial condition	U[5,100]	14, [7–34]
$S(0)$	Initial condition	N = 5176000	
$E_1(0)$, $E_2(0)$, $I_2(0)$, $A_1(0)$, $A_2(0)$,	Initial conditions	Use of steady-state conditions *	
Other Initial Conditions	Initials conditions	0	
$\beta(0)$	Initial condition of the transmission rate	0.70	
v	Volatility of the Brownian process	U[0.05,015]	0.132, [0.101–0.149]
$1/\sigma$	average duration of the incubation	tN[4,0.1,3,5]	4.01, [3.83–4.21]
$1/\gamma$	average duration of infectious period	tN[6,0.2,4.5,7.5]	6.01, [5.61–6.41]
$1/\kappa$	average hospitalized period	U[8, 20]	12.01, [10.49–13.55]
$1/\delta$	average time in ICU	U[8, 20]	17.07, [14.20-19.33]
τ_A	fraction of asymptomatics	U[0.20,0.80]	0.497, [0.218–0.789]
τ_H	fraction of hospitalization	U[0.025,20]	0.091, [0.068–0.136]
τ_I	fraction of ICU admission	U[0.025,20]	0.029, [0.025–0.039]
τ_Δ	death rate	U[0.10,070]	0.461, [0.416–0.510]
q_1	reduction of transmissibility	$1.5*q_2$ but ≤ 1	
q_2	reduction of transmissibility	0.55 (Li et al, 2020)	
q_I	reduction of ICU admission fraction	0.30	
q_D	reduction of death rate	0.40	
ρ_I	reporting rate for symptomatic infectious	U[0.05, 0.40]	0.286, [0.196–0.390]
ρ_H	reporting rate for hospitalized people	U[0.95,1]	0.967, [0.950–0.997]
ρ_{ICU}	reporting rate for the ICU admission	0.96	

* steady-state conditions are defined by : $\frac{dE_1}{dt}=\frac{dE_2}{dt}=\frac{dI_1}{dt}=\frac{dI_2}{dt}=\frac{dA_1}{dt}=\frac{dA_2}{dt}=0$

Parameters	Definitions	Prior or constant value	Posterior Median, [95%CI]
ρ_G	reporting rate for hospital discharge	0.96	
ρ_D	reporting rate for death	0.98	
* steady-state conditions are defined by : $\frac{dE_1}{dt}=\frac{dE_2}{dt}=\frac{dI_1}{dt}=\frac{dI_2}{dt}=\frac{dA_1}{dt}=\frac{dA_2}{dt}=0$			

As the peaks of those hospitalized and those admitted to ICU are concomitant we consider that a weak fraction, $q_I.\tau_I$ of infectious with severe symptoms goes directly to ICU. Even if the majority of deaths occur in the ICU, a small fraction, $q_D.\tau_D$, can occur in hospital but not in intensive care.

An interesting sub-product of our framework is the possibility of estimating the time evolution of the effective reproduction number, R_{eff}. R_{eff} is defined as the mean number of infections generated during the infectious period of a single infectious case at time t. When considering different transmission capacity for different infectious, its value is a function of both the fraction of asymptomatic infectious $A_i(t)$, τ_A, and of symptomatic infectious $I_i(t)$, 1-τ_A:

$$R_{eff}(t) = ((1-\tau_A).(1+q_1)/2 + q_2.\tau_A) \; . \; (\beta(t)/\gamma) \; . \; (S(t)/N)$$

$$R_{eff}(t)=\left(\frac{(1+q_1)}{2}.(1-\tau_A)+q_2.\tau_A\right).\frac{\beta(t)}{\gamma}.\frac{S(t)}{N}$$

Inference

As we used Brownian process for modeling the time-varying transmission rate our model is stochastic, the likelihood is intractable and it is estimated with particle filtering methods (Sequential Monte Carlo, SMC). Then the particle filter is embedded in a Markov Chain Monte Carlo framework, leading to the PMCMC algorithm [12]. More precisely, the likelihood estimated by SMC is used in a Metropolis Hasting scheme (particle marginal Metropolis Hastings) (see Appendix). The priors of the inferred parameters are in Table 1.

For the inference the observations considered are daily incident infectious at the beginning of the epidemic, new hospitalized patients, new ICU admission, new deaths and hospitalized discharges. Hospital observations are only available after the lockdown (25th of March). Because these are count processes, we model their observations with Negative Binomial likelihoods (see Appendix). Current hospital data, observed, hospitalized patients ($H_1 + H_2 + ICU$) and ICU beds used (ICU) have also been used in the inference process and we make the assumption that these variables follow a normal distribution (see Supplementary information).

Results

Figures 2 and 3 present our main results, Fig. 2 displays the fit of the model and Fig. 3 shows the dynamic of the model. The posteriors of the fitted parameters are in Table 1 and in Fig. A1.

Figure 2 illustrates the potential of the framework to effectively describe the numerous observations of this complex epidemic. The main characteristic this framework offers is the ability to reconstruct the time variation of the transmission rate $\beta(t)$ (Fig. 2A) that is needed to fit the observations. We can then compute the time-variation of R_{eff} (Fig. 2A). The initial value of R_{eff} is around 3.2 in accordance with numerous published papers (*e.g.* Flaxman et al, 2020). The peak of R_{eff} around the time of the first hospital observations is presumably a compensation effect of the model to accommodate

diverging trends between reported case data and hospital data. Then one can note a decrease of 79% of R_{eff} between the 1st of March and the 1st of May and a decrease of 82% between the 12th of March (school closure and lock down of offices, restrictions on travel etc) and the 1st of May (Fig. 2A). This sharp decrease is clearly as a result of the mitigation measures taken.

Taking account of the large variability of the daily observations, since the 1st of June we have only used a weekly average of the daily values observed (Figs. 2C-2F).

Another important characteristic of this epidemic is the fact that the peak of daily hospital admission and daily ICU admission are concomitant (Figs. 2G-2H), this concomitance has influenced the structure of the model we developed.

A final important point concerns the observed daily incident infectious. It is a source of data that the model has not taken into account in the inference process (Fig. 2B). We fit the model to the daily incident infectious up to March 25th only (black points on Fig. 2B), and plot our daily incident infectious estimates with the corresponding estimate of the reporting rate, with a median of 0.28 (95% CI: 0.19–0.39). These data highlight that the peak in observed incident infectious comes 2–3 weeks late, and is higher than expected. This shows that it is important to take into account a delay in reporting, for instance using models for now-casting [13–14]. This also clearly illustrates that the reporting rate has greatly evolved during the course of the epidemic, with part of the increase maybe explained by a greater proportion of asymptomatics tested as time went on, whereas in the model the people tested are considered symptomatic.

Figure 3 displays the dynamic of the model. Figures 3C-3D show that the asymptomatic infectious are as important as symptomatics but with a larger uncertainty due to lack of information available in the data. We are also able to estimate the % of asymptomatic transmission, the mean of this estimation equals 40% and is associated with a large uncertainty (95% CI: 14%-73%).

One can also note that the removed are few in number and the computed seroprevalence on mid October was estimated to be 0.85% (95% CI: 0.44–2.14%) showing that the country is very far from the herd immunity. Focusing on cumulative deaths at hospital our framework underestimates the cumulative deaths (Fig. 3G) even if it describes correctly the incidence of deaths (Fig. 2E).

It is worth noting that the previous remark on the difficulty using observed incident infectious can be illustrated on Fig. 3C when the reporting rate is not used for plotting.

Discussion

The need globally to accurately model COVID-19 mitigation strategies and asymptomatic transmission in order to plan for the burden on hospital admissions was identified early in the pandemic [15]. The impact of mitigation scenarios on asymptomatic cases is also clearly articulated in the modeling work of Davies et al [16]. Within their models in the United Kingdom they have predicted that extreme measures are probably required to prevent an excess of demand on hospital beds, especially those in ICUs during 2021. Similarly in France, Di Domenico et al [9] have used modeling techniques calibrated with hospital admission data to model the impact of mitigation strategies to predict the scale of the epidemic within the Ile-de-France region. In the same way, we provide estimations of the dynamics of the COVID-19 epidemic in Ireland and its key parameters. We also quantify the effects of mitigation measures on the virus transmission before, during and after the lockdown. The main characteristics of our approach is accounting for non-stationarity by embedding time-varying parameters in a stochastic model coupled with Bayesian inference. This allows us to describe the time evolving COVID-19 epidemic based purely on the available data without specific hypothesis on this time evolution.

Using known reported hospitalized cases, we present the first Irish modeling estimates of hidden asymptomatic cases and resulting short term predictions of numbers hospitalized and number recovering. The model presented predicted that in

Ireland as of the 31st July between 0.44% and 2.14% of the population had been infected either as a symptomatic or asymptomatic case. Within the population of 5.1 million this equates to over 55,000 individuals. The number known through testing at the same time was 26,027 individuals [3, 5] and we can see from these figures that the number of predicted cases from the model in Ireland at the end of July may have been almost 2 times the number identified through testing centers.

This disparity between estimated number infected and known is not to be unexpected and similar percentages of infections have been observed in other countries in these early stages of the epidemic. Li et al [8] estimate that in the early stages of the epidemic in China before the 23 January 2020 travel restrictions 86% of all infections were undocumented (95% CI: 82–90%). Similar proportions are evident when we compare the known and estimated cases in Ireland. However perhaps what is more important according to Li et al [8] was that the transmission rate of undocumented infections per person was 55% the transmission rate of documented infections (95% CI: 46–62%), yet, because of their greater numbers, undocumented infections were the source of 79% of the documented cases.

Undocumented infections particularly asymptomatic infections are known to be the silent drivers of infection. The European Centre for Disease Prevention and Control [17] in their update on the role of asymptomatic and pre-symptomatic individuals on August 10th 2020 report that similar viral loads in asymptomatic versus symptomatic cases have been reported, indicating the potential of virus transmission from asymptomatic patients. Furthermore viral loads in asymptomatic patients from diagnosis to discharge tended to decrease more slowly than those in symptomatic (including pre-symptomatic) patients.

Aguilar et al [18] in their study investigating the impact of asymptomatic carriers on COVID-19 transmission state that, in a public health context, the silent threat posed by the presence of asymptomatic carriers in the population results in the COVID-19 pandemic being much more difficult to control. Their study shows that the population of individuals with asymptomatic COVID-19 infections is driving the growth of the pandemic.

Given the increases in cases in Ireland in September 2020 (see Fig. 2B) where a second wave is emerging from the capital city and in some rural regions in spite of a period of increased testing and sustained mitigation strategies the role of the silent asymptomatic cases is now more important than ever. One rural region in the Republic of Ireland currently experiencing a second wave is in close proximity to a border city in Northern Ireland where different mitigation strategies, different testing protocols and different definitions are in operation, These emerging second waves highlight the unique challenges facing policy makers within and across borders.

It is interesting to compare this model prediction with recent preliminary national serological results, which found that among 12 to 69 year olds living in Ireland the sero-prevalence rate was estimated in July 2020 at 1.7% (95% CI: 1.1–2.4%) but at 0.6% (95% CI: 0.2–1.4%) in the Sligo province (a lower incidence of COVID-19 cases area) [19]. Despite the fact that the HSE model is age and region limited and given that the majority of cases in Ireland in the early stage of the epidemic were in those over the age of 65 years, the observed range is in the same order of magnitude as our predictions.

Our seroprevalence predictions contrast with those of more densely populated areas. Comparing the Davies et al [16] model predictions on serological prevalence with recent serological study results in the UK we see that Davies et al [16] assumes that subclinical or asymptomatic rates are 50% of all infections and under this and other model assumptions authors find in their scenario analyses that extreme measures are probably required to bring the epidemic under control and to prevent very large numbers of deaths and an excess of demand on hospital beds, especially those in ICUs. To date estimated serological prevalence in the United Kingdom based on a random sample of home based testing has found that 6.0% (95% CI: 5.8–6.1%) of individuals tested positive, of these one third (32.2%, (95% CI, 31.0–33.4%)) reported no symptoms and were asymptomatic [20]. Overall the authors estimated that 3.36 million (3.21 million to 3.51 million) people had been infected with SARS-CoV-2 in England by the end of June 2020. This estimate was substantially higher than the recorded numbers in the UK of 315 000 cases. This is in accordance with observations from Spain where between

April and May 2020, seroprevalence was 5,0% and only few cases of these people had a PCR test [21]. Within the Di Domenico et al [9] study on the Ile-de-France authors estimate that the population infected by COVID-19 as of April 5 and prior to lockdown to be in the range 1–6%. This was predicted using two values of the probability of being asymptomatic, namely 20% and 50%.

Our study is not without limitations. Our model like all complex SEIR models developed for COVID-19 is non-identifiable which means that it is likely that several solutions exist and we only present one of the most likely. This point is always overlooked but see Li et al [8]. The major limitation is the use of the classical homogeneous mixing assumption in which all individuals are assumed to interact uniformly and ignores heterogeneity between groups by sex, age, geographical region. In all cases taking an age structure and mixing matrix appears insufficient and heterogeneity of contact is important (see [22]). However this kind of data is not easily available. Another weakness is perhaps the neglect of age-structure in the model to simulate age-based predictions as we enter the time of children returning to school. These weaknesses are however a future research development given the performance of the current model. Nevertheless in our opinion, these limitations are compensated for taking non-stationarity of this epidemic into account and by the fact that our results are mainly driven by hospital data, which is more accurate than the number of infected cases. Precise data from serological studies would significantly reduce the uncertainties of the model predictions [23–24].

The key strength of the current Irish study is the fit of the model to the current observed data on hospitalizations, deaths and ICU cases. The first estimate of the asymptomatic cases predicted by this model is also reflecting the emerging data from the Irish serological prevalence study. We also found a reduction of transmissibility of the SARS-CoV-2 of 78%-82% that is in accordance with the results published on the effects of mitigation measures in Europe [25–26]. For example, Garchitorena et al [26] by comparing 24 non-pharmaceutical interventions found that the median decrease in viral transmission was 74%, which is enough to suppress the epidemic and that a partial implementation of different measures resulted in lower than average response efficiency. Our results also highlighted that the observed confirmed cases are only a small fraction of the total number of cases, only the tip of the iceberg (see [27]. Then data from hospital system published by health authorities are crucial for understanding the course of this epidemic. These data are well measured, but are observed with a delay in relation to contamination. Nevertheless, these delays can be easily account for by mathematical models.

Conclusions

In this work we have used a stochastic framework that accounts for the time-varying nature of the COVID-19 epidemic by using time-varying parameters and hospital data to provide a description of this evolving epidemic. Our results demonstrate that Ireland has significantly reduced transmission by employing mitigation measures, physical distancing and lockdown. This has avoided the saturation of healthcare infrastructures, flattened the epidemic curve and likely reduced mortality. Our framework that accounts for the non-stationarity of the transmission also offers the possibility of computing the time varying $R_{eff}(t)$ and then to offer an interesting tool to follow the evolution of the COVID-19 epidemic. This tool could prove particularly useful in analyzing this new phase of worrying expansion of second waves, driven within and between borders by the so called silent and hidden asymptomatic yet powerful infections.

Declarations

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Authors' contributions

BC and CaC contributed to conception and design of the study. BC, BNVY and ClC constructed and ran the model. All authors analyzed the simulations. The first draft of the manuscript was written by BC and CaC, all authors provided comments, edited and approved the final manuscript.

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Availability of data and materials

All surveillance data are available at the site from Health Protection Surveillance Centre (HPSC): https://www.hpsc.ie/a-z/respiratory/coronavirus/novelcoronavirus/casesinireland/epidemiologyofcovid-19inireland/

or https://covid19ireland-geohive.hub.arcgis.com/

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests

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Figures

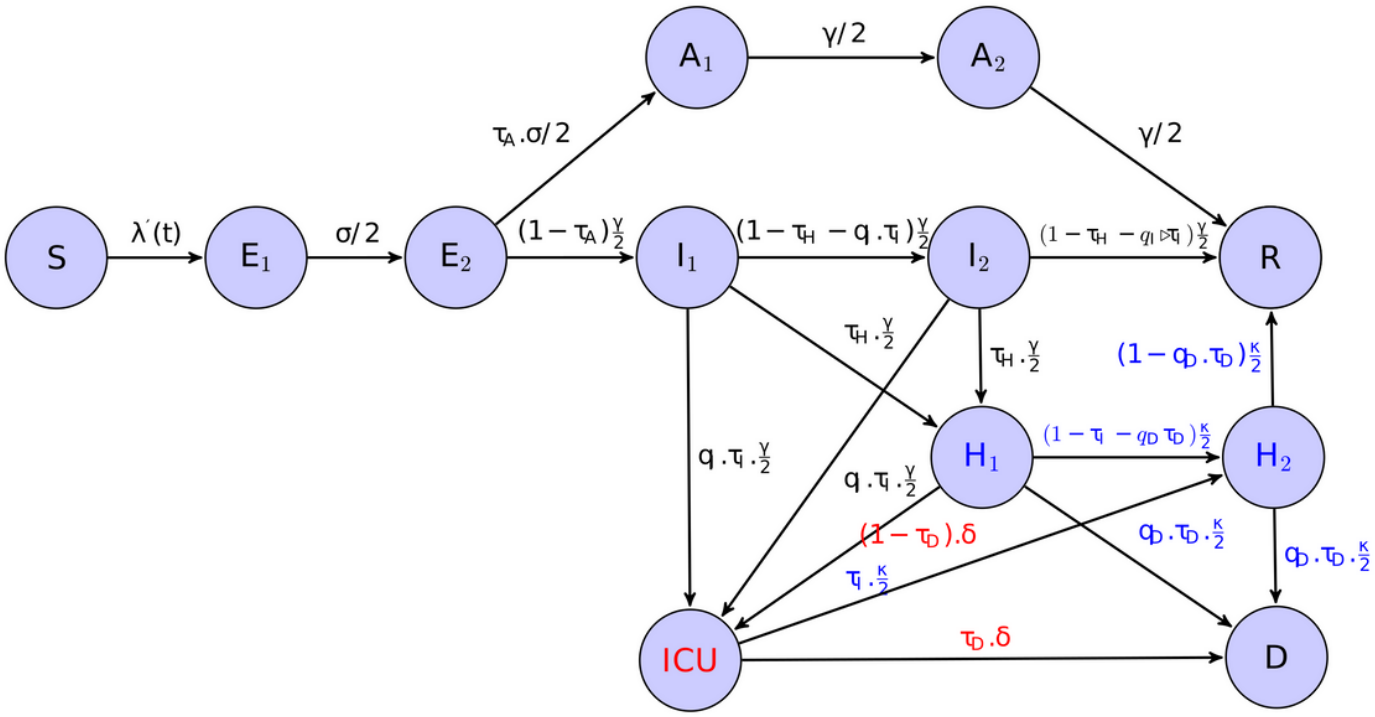

Figure 1

“See the Supplemental Files section for the complete figure caption”.

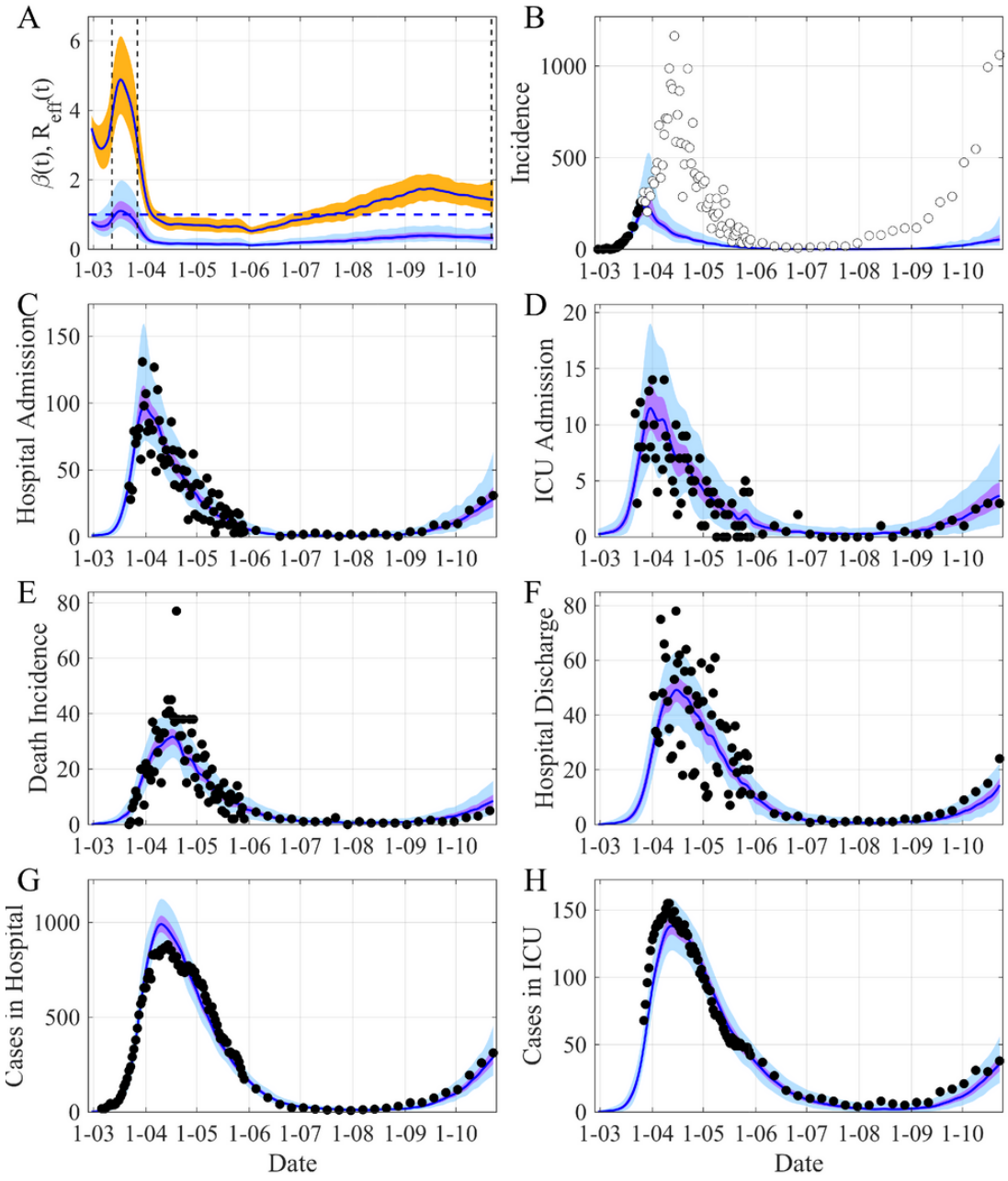

Figure 2

Reconstruction of the observed dynamics of COVID-19 in Ireland. (A) The time evolution of both β(t) and Reff (t). (B) Simulated observed daily incident infectious. (C-D) New daily admissions to hospital and to ICU. (E) Daily new deaths. (F) Hospital discharges. (G-H) Cases in Hospital and in ICU each day. The black points are observations used by the inference process, the white points are the observations not used. The blue lines are the median of the posterior of the simulated trajectories, the mauve areas are the 50% Credible Intervals (CI) and the light blue areas the 95% CI. In (A) the orange area is the 50% CI of Reff, the vertical dashed lines show the date of the main NPI measures and the horizontal dashed-line Reff = 1. For all the graphs, the reporting rates are applied to the model trajectories (Fig. 3) as during the inference process for comparison to the observations.

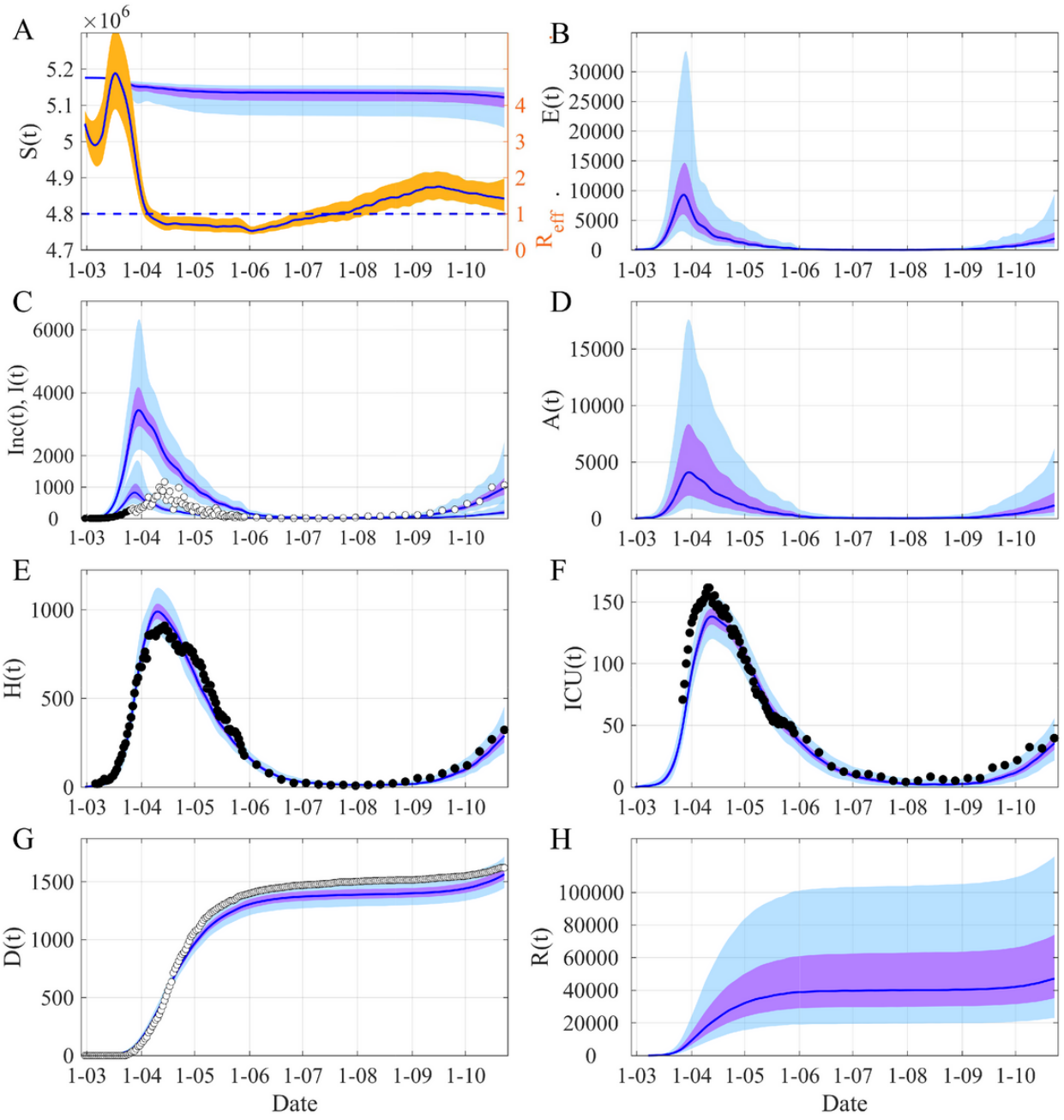

Figure 3

Dynamics of COVID-19 in Ireland. (A) Time evolution of both susceptibles S(t) and Reff (t). (B) Infected non infectious, E(t) = E1(t)+E2(t). (C) Symptomatic infectious I(t) = I1(t)+I2(t) and incidence of symptomatic infectious (the lower line). (D) Asymptomatic infectious A(t) = A1(t)+A2(t). (E) Hospitalized people H(t) = H1(t)+H2(t)+ICU(t). (F) People in ICU, ICU(t). (G) Cumulative death D(t). (H) Removed R(t). The blue lines are the median of the posterior of the simulated trajectories, the mauve areas are the 50% Credible Intervals (CI) and the light blue areas the 95% CI. In (A) the orange area is the 50% CI of Reff and the horizontal dashed-line indicates Reff = 1. The black points are observations used by the inference process, the white points are the observations not used.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- FigureA1.png
- CovidIrelandV5AdditionalFile.docx
- Figure1Caption.docx