Accounting for Symptomatic and Asymptomatic in a SEIR-type model of COVID-19

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Abstract

A mathematical model was developed describing the dynamic of the COVID-19 virus over a population considering that the infected can either be symptomatic or not. The model was calibrated using data on the confirmed cases and death from several countries like France, Philippines, Italy, Spain, United Kingdom, China, and the USA. First, we derived the basic reproduction number, $\mathcal{R}_0$ and estimated the effective reproduction $\mathcal{R}_{\text{eff}}$ for each countries. Second, we were interested in the merits of containment. Results revealed that total and partial containment is effective in reducing the transmission. However, its duration may be long to eradicate the disease (185 days for France). By setting the end of containment as the day when hospital capacity is reached, numerical simulations showed that the duration can be reduced (up to only 84 days for France if the capacity is 1000 patients). Further, results pointed out that the effective reproduction number remains large after containment. Therefore, testing and isolation are necessary to stop the disease.

1. Introduction

In late 2019, a disease outbreak emerged in a city of Wuhan, China. The culprit was a certain strain called Coronavirus Disease 2019 or COVID-19 in brief Wor (2020a). This virus has been identified to cause fever, cough, shortness of breath, muscle ache, confusion, headache, sore throat, rhinorrhoea, chest pain, diarrhea, and nausea and vomiting Hui et al. (2020); Chen et al. (2020).

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COVID-19 belongs to the *Coronaviridae* family. A family of coronaviruses that cause diseases in humans and animals, ranging from the common cold to more severe diseases. Although only seven coronaviruses are known to cause disease in humans, three of these, COVID-19 included, can cause a much severe infection, and sometimes fatal to humans. The other two to complete the list were the severe acute respiratory syndrome (SARS) identified in 2002 in China, and the Middle East respiratory syndrome (MERS) originated decade after in Saudi Arabia.

Like MERS and SARS, COVID-19 is a zoonotic virus and believed to be originated from bats transmitted to humans Zhou et al. (2020). In comparison with SARS, MERS, the COVID-19 appears to be less deadly. However, the World Health Organization (WHO) reported that it has already infected and killed more people than its predecessors combined. Also, COVID-19 spreads much faster than SARS and MERS. It only took over a month before it surpassed the number of cases recorded by the SARS outbreak in 2012. According to WHO, it only took 67 days from the beginning of the outbreak in China last December 2019 for the virus to infect the first 100,000 people worldwide Wor (2020b). As of 25 March 2020, a cumulative total of 372,757 confirmed cases while 16,231 deaths have been recorded for COVID-19 by World Health Organization Wor (2020c).

Last 30th of January WHO characterized COVID-19 as Public Health Emergency of International Concern (PHEI) and urge countries to put in place strong measures to detect disease early, isolate and treat cases, trace contacts, and promote social distancing measures commensurate with the risk Wor (2020d). In response, the world implemented its measures to reduce the spread of the virus. Limitations on mobility, social distancing and self-quarantine have been implemented. Moreover, health institutions advise people to practice good hygiene to keep from being infected. All these efforts have been made to reduce the transmission rate of the virus.

For the time being, COVID-19 infection is still on the rise. Government and research institutions scramble to seek antiviral treatment and vaccines to combat the disease. Several reports list possible drugs combination to apply, yet it is still unclear which drugs could combat the viral disease and which won't.

Several mathematical models have been proposed from various epidemiological groups. These models help government as an early warning device about the size of the outbreak, how quickly it will spread, and how effective control measures may be. However, due to the limited emerging understanding of the new virus and its transmission mechanisms, the results are largely inconsistent across studies.

In this paper, we will mention a few models and at the end to propose one. Gardner and his team Gardner (2020) at Center for Systems Science and Engineering, Johns Hopkins University implemented on a previously published model applied for COVID-19. It is a metapopulation network model represented by a graph with each nodes follows a discrete-time Susceptible-Exposed-Infected-Recovered (SEIR) compartmental model. The model gives an estimate the expected number of cases in mainland China at the end of January 2020, as well as

the global distribution of the infected travelers. They believe that the outbreak began in November 2019 with hundreds of infecteds already present in Wuhan last early December 2019. Wu et al.Wu et al. (2020) from WHO Collaborating Centre for Infectious Disease Epidemiology and Control at University of Hong Kong presented a modelling study on the nowcast and forecast of the 2019-nCoV outbreak at Wuhan. The group used an SEIR metapopulation model to simulate epidemic and found reproductive number R_0 around 2.68 (with 95% credible interval 2.47-2.86). Imai et al. Imai et al. (2020) estimates R_0 around 2.6 with uncertainty range of 1.5-3.5. Zhao et al.Zhao et al. (2020a,b) found R_0 to range from 2.24 to 5.71 based on the reporting rate of cases. If the reporting rate increase 2-fold, $R_0 = 3.58$, if it increase 8-fold, $R_0 = 2.24$. If there is no change in the reporting rate, the estimated R_0 is 5.71. Similar with the above authors, Wang et al.Wang et al. (2020) employed an infectious disease dynamics model (SEIR model) for the purpose of modeling and predicting the number of COVID-19 cases in Wuhan. They opined that in order to significantly reduce R_0 the government should continue implementing strict measures on containment and public health issues. In the same tune as the latter, the model of Danchin et al. Danchin et al. (2020) also suggests to continue implementing effective quarantine measures as to avoid resurgence of infection. The model consist of five (5) compartments: susceptible, infected, alternative infection, detected and removed.

Here, we proposed an extension from the classical SEIR model by adding a compartment of asymptomatic infected. We address the challenge of predicting the spread of COVID-19 by giving our estimates for the basic reproductive numbers $\mathcal{R}_0$ and its effective reproductive number $\mathcal{R}_{\text{eff}}$. Afterwards, we also assess risks and interventions via containment strategy.

The rest of the paper is organized as follows. Section 2, outlines our methodology. Here the model was explain, where the data was taken and its parameter estimates. Section 3 contained the qualitative analysis for the model. Here, we give the closed form equation of reproductive number R_0 then tackling best strategy to reduce transmission rates. Finally, section 4 outlines our brief discussion on some measures to limit the outbreak.

2. Materials and methods

2.1. Confirmed and death data

In this study, we used the publicly available dataset of COVID-19 provided by the Johns Hopkins University Dong et al. (2020). This dataset includes many countries daily count of confirmed cases, recovered cases and deaths. Data can be downloaded from https://github.com/CSSEGISandData/COVID-19/tree/master/csse_covid_19_data. These data are collected through public health authorities' announcements and are directly reported public and unidentified patient data, so ethical approval is not required.
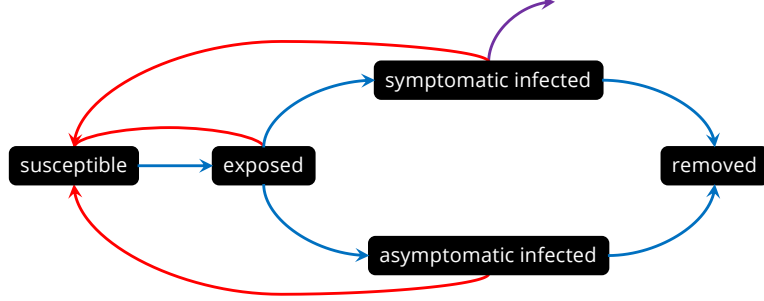
Figure 1: Compartmental representation of the SEI_aI_sR-model. Blue arrows represent the infection flow. Red arrows denote for the infectivity. Purple arrow is the death.

2.2. Mathematical model

We focus our study on five components of the epidemic flow (Figure 1), *i.e.* Susceptible individual (S), Exposed individual (E), Symptomatic Infected individual (I_s), Asymptomatic Infected individual (I_a) and Removed individual (R). To build the mathematical model, we followed the standard strategy developed in the literature concerning SIR model. We assumed that susceptible can be infected by exposed, symptomatic infected as well as asymptomatic infected individuals. The dynamics is governed by a system of five ordinary differential equations (ODE) as follows

$$
\begin{aligned}
S'(t) &= -\left(\beta_e E + \beta_s I_s + \beta_a I_a\right)\frac{S}{N} \\
E'(t) &= \left(\beta_e E + \beta_s I_s + \beta_a I_a\right)\frac{S}{N} - \delta_e E \\
I_s'(t) &= f\delta_e E - (\gamma + \mu_s)I_s \\
I_a'(t) &= (1-f)\delta_e E - \gamma I_a \\
R'(t) &= \gamma(I_s + I_a)
\end{aligned}
$$

Note that the total living population follows $N'(t) = -\mu_s I_s$, while death is computed by $D'(t) = \mu_s I_s$. We assume that there is no new recruit. The parameters are described in Table A of Figure 2.

2.3. Parameters estimation

The model is made up of seven (7) parameters $\theta = (\beta_e, \beta_s, \beta_a, \delta_e, f, \gamma, \mu_s)$ that need to be determined. Given, for N days, the observations $I_{s,obs}(t_i)$ and $D_{obs}(t_i)$, the cost function consist of a nonlinear least square function

$$
J(\theta) = \sum_{i=1}^{N}(I_{s,obs}(t_i) - I_s(t_i,\theta))^2 + (D_{obs}(t_i) - D(t_i,\theta))^2,
$$

with constraint $\theta \geq 0$, and $0 \leq f \leq 1$. Here $I_s(t_i,\theta)$ and $D(t_i,\theta)$ denote output of the mathematical model at time t_i computed with the parameters θ. The optimization problem is solved using Approximate Bayesian Computation combined with a quasi-Newton method Csilléry et al. (2010).

3. Results

3.1. Basic and effective reproduction numbers

It is standard to check that the domain

$$\Omega = \{(S, E, I_s, I_a, R) \in \mathbb{R}_+^5; 0 \leq S + E + I_s + I_a + R \leq N(0)\}$$

is positively invariant. In particular, there exists a unique global in time solution (S, E, I_s, I_a, R) in $\mathcal{C}(\mathbb{R}_+; \Omega)$ as soon as the initial condition lives in Ω.

Since the infected individuals are in E, I_a and I_s, the rate of appearance of new infections in each compartment $\mathcal{F}$ and the rate of other transitions between all compartments $\mathcal{V}$ can be rewritten as

$$\mathcal{F} = \begin{pmatrix} (\beta_e E + \beta_s I_s + \beta_a I_a)\frac{S}{N} \\ 0 \\ 0 \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} \delta_e E \\ (\gamma + \mu_s)I_s - f\delta_e E \\ \gamma I_a - (1-f)\delta_e E \end{pmatrix}.$$

Thus,

$$F = \begin{pmatrix} \frac{\beta_e S}{N} & \frac{\beta_s S}{N} & \frac{\beta_a S}{N} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

and

$$V = \begin{pmatrix} \delta_e & 0 & 0 \\ -f\delta_e & \gamma + \mu_s & 0 \\ -(1-f)\delta_e & 0 & \gamma \end{pmatrix}, \quad V^{-1} = \begin{pmatrix} \frac{1}{\delta_e} & 0 & 0 \\ \frac{f}{\gamma+\mu_s} & \frac{1}{\gamma+\mu_s} & 0 \\ \frac{1-f}{\gamma} & 0 & \frac{1}{\gamma} \end{pmatrix}.$$

Therefore, the next generation matrix is

$$FV^{-1} = \begin{pmatrix} \frac{\beta_e S}{\delta_e N} + \frac{f\beta_s S}{(\gamma+\mu_s)N} + \frac{(1-f)\beta_a S}{\gamma N} & \frac{\beta_s S}{(\gamma+\mu_s)N} & \frac{\beta_a S}{\gamma N} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

We deduce that the basic reproduction number $\mathcal{R}_0$ for the Disease Free Equilibrium $(S^*, 0, 0, 0, R^*)$, with $N^* = S^* + R^*$, is

$$\mathcal{R}_0 := \left(\frac{\beta_e}{\delta_e} + \frac{f\beta_s}{(\gamma + \mu_s)} + \frac{(1-f)\beta_a}{\gamma} \right) \frac{S^*}{N^*}.$$

In the subsequent, we write *DFE* when we mean by Disease Free Equilibrium.

Theorem 3.1. *The DFE $(S^*, 0, 0, 0, R^*)$ is globally asymptotically stable.*

Proof. By computing the eigenvalues of the Jacobian matrix, we deduce that if $\mathcal{R}_0 < 1$, then DFE is locally asymptotically stable.

Reciprocally, local asymptotic stability of DFE does not necessarily imply $\mathcal{R}_0 < 1$. Here we will prove that global asymptotic stability is independent that of $\mathcal{R}_0$. Indeed, from the last differential equation in our system of ODE, we can

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deduce that R is an increasing function bounded by $N(0)$. Thus $R(t)$ converges to R^* as t goes to $+\infty$. Then integrating over time this equation provides

$$R(t) - R(0) = \gamma \int_0^t I_s(s) + I_a(s)\,ds$$

and

$$R^* - R(0) = \gamma \int_0^{+\infty} I_s(s) + I_a(s)\,ds,$$

which is finite. Furthermore, $I_s(t), I_a(t)$ also go to 0 as $t \to +\infty$ thanks to the positivity of the solution. Similarly, integrating the third or the fourth equation in the system gives $E(t) \to_{t\to+\infty} 0$. Finally, the first equation points to the decreasing of S that is bounded by below by 0 and $S(t) \to_{t\to+\infty} S^*$. $\qquad \square$

This theorem means that the asymptotic behavior does not depend on $\mathcal{R}_0$. For all initial data in Ω, the solution converges to the DFE when time goes to infinity.

Here $\mathcal{R}_0$ is not a relevant indicator, and it is better to consider the effective reproduction number

$$\mathcal{R}_{\text{eff}}(t) = \left(\frac{\beta_e}{\delta_e} + \frac{f\beta_s}{(\gamma + \mu_s)} + \frac{(1-f)\beta_a}{\gamma} \right) \frac{S(t)}{N(t)} =: \mathcal{R}\frac{S(t)}{N(t)}.$$

The effective reproduction number $\mathcal{R}_{\text{eff}}(t)$ is represented in Figure 2D and values of $\mathcal{R}$ are computed in Table A of Figure 2.

3.2. Model resolution

To calibrate the model, simulations start the day of first confirmed infection and finish before interventions to reduce the disease. We assume that the whole population of the country is susceptible to the infection. Seven states with comparable population are chosen. The objective function J is computed to provide a relative error of order less than 10^{-2}. In Figure 2, Table A. shows estimated parameters. The rest of the Figure presents the solution and data.

A.

| country/state	description	France	Philippines	Italy	Spain	United Kingdom	Hubei	New York
J	relative cost function	4.70E-03	2.02E-02	3.07E-02	5.94E-03	4.27E-03	7.25E-02	7.96E-04
β_e	transmission rate from S to E from contact with E	0.057899	0.223627	0.251883	0.273061	0.237637	0.000000	0.277300
β_s	transmission rate from S to E from contact with I_s	0.368146	0.399019	0.675281	0.503112	0.979359	0.507331	0.002878
β_a	transmission rate from S to E from contact with I_a	0.471628	0.186464	0.000000	0.000432	0.598530	0.000000	4.263184
δ_e	latency rate	0.269925	0.143335	2.872143	0.572886	0.183673	3.445527	0.135697
f	probability of be symptomatic	0.387702	0.084839	0.426419	0.490394	0.051886	0.598110	0.159819
1-f	**probability of be asymptomatic**	**0.612298**	**0.915161**	**0.573581**	**0.509606**	**0.948114**	**0.401890**	**0.840181**
γ	recovery rate	0.103809	0.122703	0.063046	0.044322	0.383395	0.033333	1.787245
μ_s	death rate	0.007093	0.015090	0.020413	0.013520	0.012764	0.009753	0.005178
$\mathcal{R}$	**effective reproduction number**	**4.283329**	**3.196568**	**3.537937**	**4.747048**	**2.902201**	**7.042639**	**4.047891**
median $\mathcal{R}$	median effective reproduction number	3.096738	3.155502	2.089169	2.902238	2.868001	7.405132	3.781538
mean $\mathcal{R}$	mean effective reproduction number	3.474858	3.779976	3.542378	3.439437	3.939954	7.633783	4.111556

B.

C.

D.
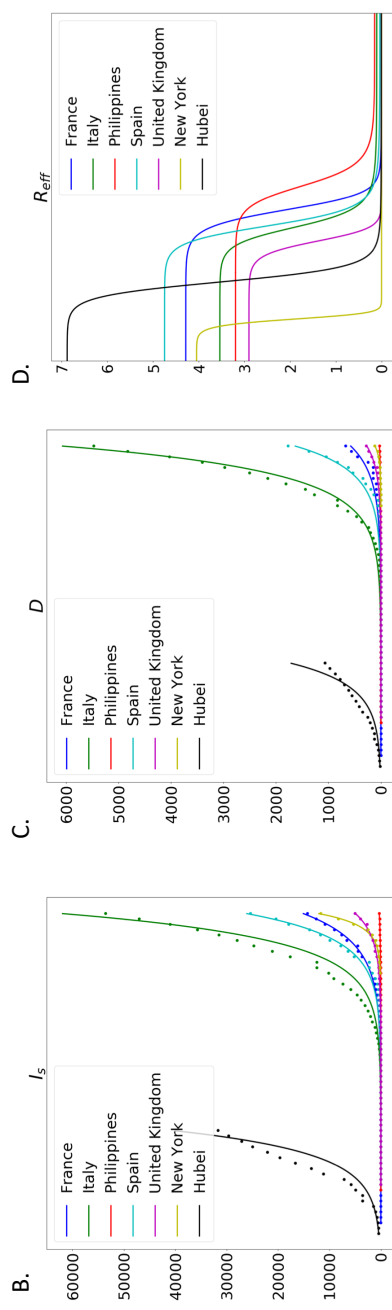
Figure 2: A. Parameters calibrated according to data issues from France, Philippines, Italy, Spain, United Kingdom, Hubei and New York. B. and C. Calibrated solution (straight line) and data (dots) with respect to day for France, Philippines, Italy, Spain, United Kingdom, Hubei and New York. First is the infected I_s (B.), and the second one is the death D (C.). D. Effective reproduction number with respect to day.
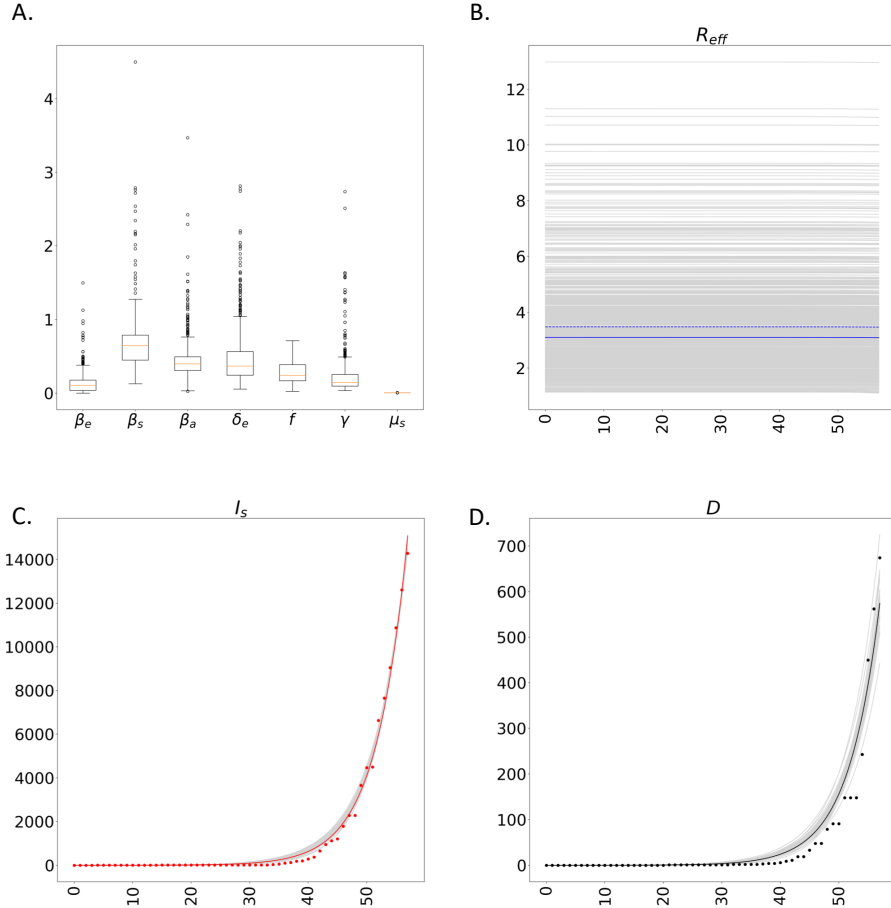
Figure 3: A. Boxplot of the posterior distribution computed from France data. B. Effective reproduction number in grey of the posterior distribution, median (= 3.096738) in blue straight line, mean (= 3.474858) is dotted line. C. Fitted symptomatic infected in grey of the posterior distribution, median in red straight line, mean is dotted line. D. Fitted death in grey of the posterior distribution, median in black straight line, mean is dotted line.

3.3. A strategy to reduce disease to a given threshold

To temporarily reduce the value of $\mathcal{R}_{\text{eff}}$, the best strategy consists in reducing the transmission rates $\beta_e, \beta_s, \beta_a$. Let t_1, t_2 be the beginning and end of the intervention, we set

$$\beta_e \leftarrow \omega\beta_e, \beta_s \leftarrow \omega\beta_s, \beta_a \leftarrow \omega\beta_a \text{ for } t_1 \leq t \leq t_2. \tag{1}$$

Given a critical infection threshold $\mathcal{T}_c$, the intervention duration is provided by the difference of time $t_2 - t_1$ such that

$$E(t_2) + I_s(t_2) + I_a(t_2) \leq \mathcal{T}_c.$$

Full containment is translated by $\omega = 0$, and a 90% of containment by $\omega = 0.1$.

To juxtapose the benefit of the intervention, we assume that containment starts 53 days after the first confirmed infection. We remind that the first infection in France was confirmed on January 24th, 2020 and containment begins on March 17th, 2020. Comparison between three strategies can be found in Table 1 and Figures 4-5. Without intervention to control the disease, the maximum number of symptomatic infecteds varies from 3.49×10^5 to 2.02×10^7. The maximum number of deaths totals from 8.85×10^3 to 7.92×10^6. Concerning France, Philippines, Italy, Spain and Unitedd Kingddom, when containment is fully respected and when the sum of infecteds is reduced to 1, the maximum number of symptomatic infecteds and deaths has been cut sharply, of an order 10^3. It varies now from 5.75×10^2 to 7.04×10^4 and 1.94×10^2 to 2.52×10^4 respectively. To wait from 131 to 315 days is the price to paid. On the contrary, for the states of Hubei and New York, 53 days to intervene seems to be already too late.

Note that when the intervention is done at time t_2, the number of susceptible $S(t_2)$ is large so that the effective reproduction number $\mathcal{R}_{\text{eff}}$ is larger than 1.

country/state	France	Philippines	Italy	Spain	United Kingdom	Hubei	New York
population	6.67E+07	1.03E+08	6.03E+07	4.63E+07	6.49E+07	5.84E+07	1.95E+07
maximum number of symptomatic infected without control	7.74E+06	1.75E+06	9.43E+06	1.05E+07	3.49E+05	2.02E+07	9.25E+04
maximum number of dead without control	1.64E+06	9.15E+05	6.15E+06	5.29E+06	1.02E+05	7.92E+06	8.85E+03
containment duration if $\mathcal{T}_c = 1$ starting 53 days after the first confirmed infection	157	146	247	315	131	550	172
maximum number of symptomatic infected with full containment	8.78E+03	5.75E+02	1.19E+05	7.40E+04	1.16E+04	2.00E+07	9.22E+04
maximum number of dead with full containment	1.24E+03	1.94E+02	4.24E+04	2.52E+04	1.96E+03	6.53E+06	6.82E+03
containment duration if $\mathcal{T}_c = 1000$ starting 53 days after the first confirmed infection	92	86	139	163	93	345	121

Table 1: Comparison between the maximum number of symptomatic infected and death without control and the solution reducing transmission rates to 0 to reach $\mathcal{T}_c = 1$ and $\mathcal{T}_c = 1000$.
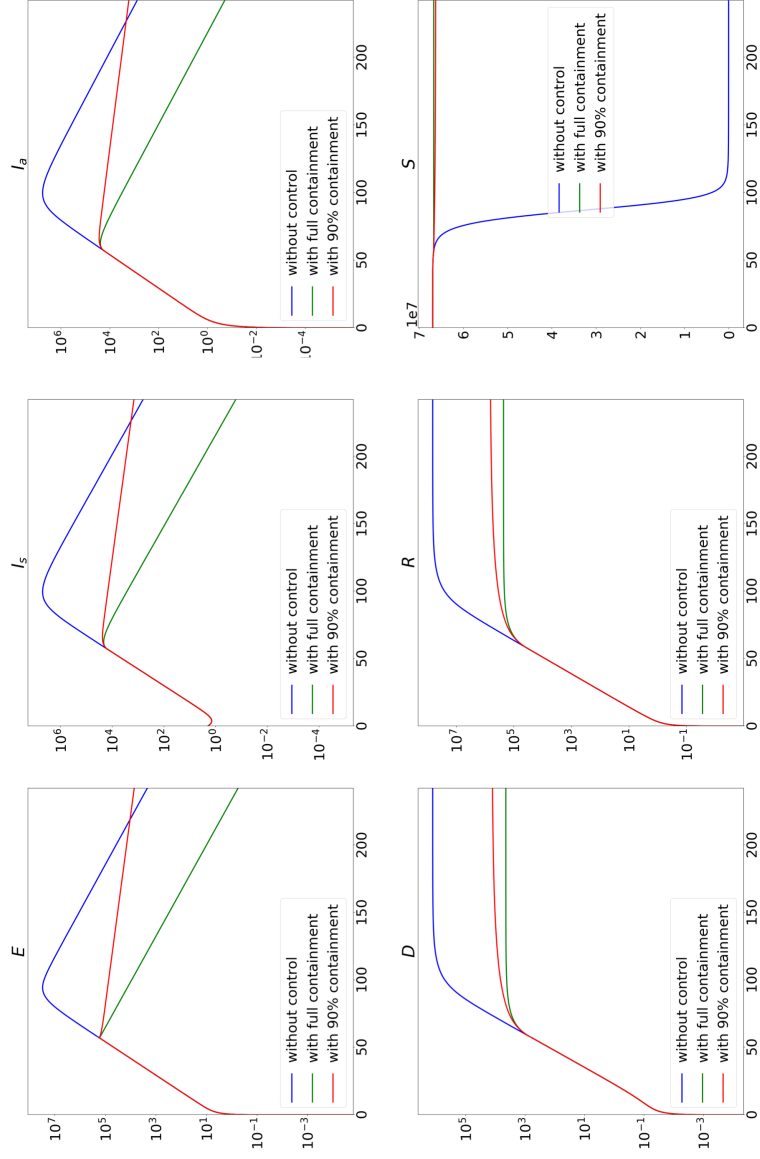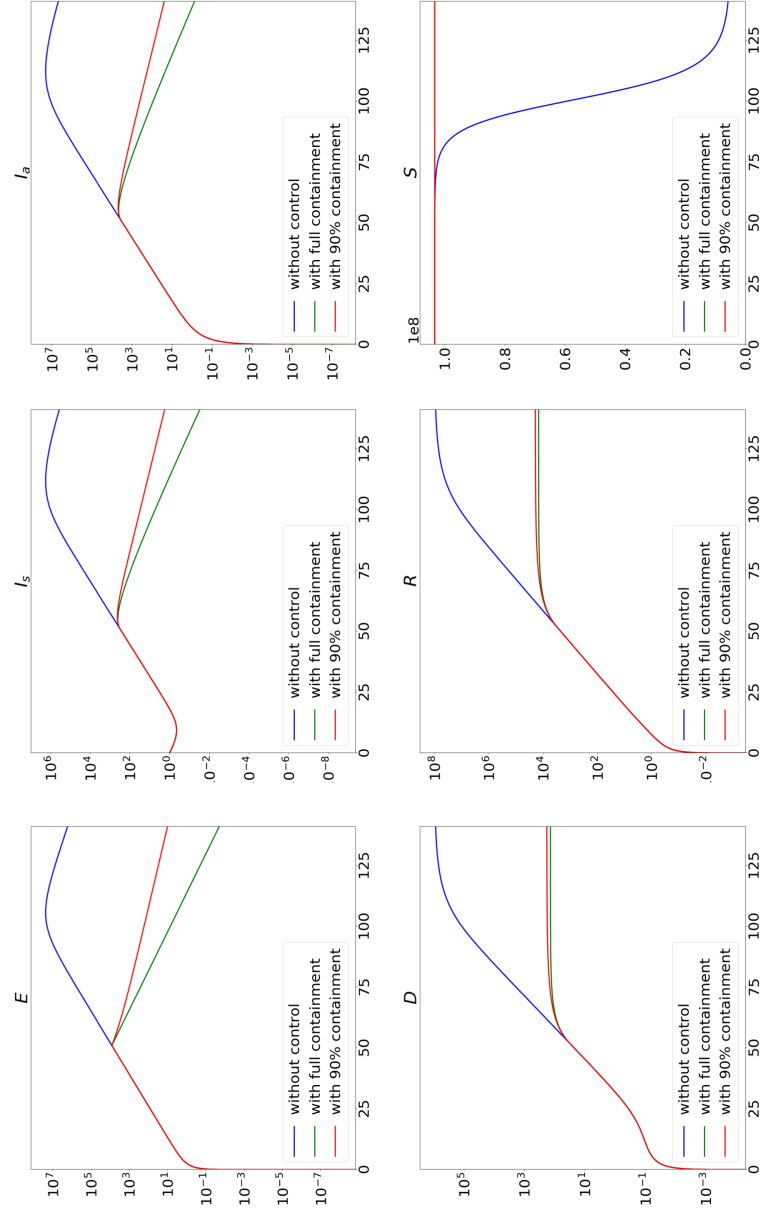
Figure 4: Comparison of solutions S, E, I_s, I_a, R, D without control in blue, with full containment in green ($\omega = 0$), with 90% containment in red ($\omega = 0.1$) for France. Ordinate axis is expressed in log.

Figure 5: Comparison of solutions S, E, I_s, I_a, R, D without control in blue, with full containment in green ($\omega = 0$), with 90% containment in red ($\omega = 0.1$) for the Philippines. Ordinate axis is expressed in log.

4. Discussion

Without intervention, we observe in Figures 4-5 that the number of susceptible S is decreasing, most of the individuals are recovering, which generates population immunity. It translates that the disease free equilibrium is globally asymptotically stable. Nevertheless, the price to pay is high, the number of deaths being excessive. As presented in Figure 2, the effective reproduction is decreasing and points out that control has been done as fast as possible.

The second important information is that, as discovered by Danchin et al. (2020), an alternative transmission way may occur. Here, it is due to the proportion of asymptomatic infected individuals that is not negligible as shown in Table 1.

Finally, with the little knowledge about COVID-19 nowadays, decreasing transmission, *i.e.* $\beta_e, \beta_s, \beta_a$, is the preferred option. The simplest choice consists in reducing contact between individuals. Table 1 and Figures 4-5 show that total and partial containment do indeed drastically reduce the disease. However the duration of containment may be too long, and then impracticable especially if we aim at totally eradicating the infection ($\mathcal{T}_c = 1$). Instead, to stop the containment as soon as the capacity of the hospitals has been reached could be privileged. When this criterion is set to 1000 patients ($\mathcal{T}_c = 1000$), the duration goes from 157 to 92 days for France. Similar reduction in duration is also obtained for other countries. Again, we see that the earlier the intervention, the more effective it is. Due to the high number of susceptible, it is worth noting that the effective reproduction number remains large after containment. Screening tests are then necessary to be carried out and the positive individuals be quarantined.

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