

Prediction of the time evolution of the COVID-19 disease in Guadeloupe with a stochastic evolutionary model

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Predictions on the time-evolution of the number of severe and critical cases of COVID-19 patients in Guadeloupe are presented. A stochastic model is purposely developed to explicitly account for the entire population (≈ 400000 inhabitants) of Guadeloupe. The available data for Guadeloupe are analysed and combined with general characteristics of the COVID-19 to constrain the parameters of the model. The time-evolution of the number of cases follows the well-known exponential-like model observed at the very beginning of a pandemic outbreak. The exponential growth of the number of infected individuals is controlled by the so-called basic reproductive number, R_0 , defined as the likely number of additional cases generated by a single infectious case during its infectious period T_I . Because of the rather long duration of infectious period (≈ 14 days) a high rate of contamination is sustained during several weeks after the beginning of the containment period. This may constitute a source of discouragement for people restrained to respect strict containment rules. It is then unlikely that, during the containment period, R_0 falls to zero. Fortunately, our models shows that the containment effects are not much sensitive to the exact value of R_0 provided we have $R_0 < 0.6$. For such conditions, we show that the number of severe and critical cases is highly tempered about 4 to 6 weeks after the beginning of the containment. Also, the maximum number of critical cases (i.e. the cases that may exceed the hospital's intensive care capacity) remains near 30 when $R_0 < 0.6$. For a larger $R_0 = 0.8$ a slower decrease of the number of critical cases occurs, leading to a larger number of deceased patients. This last example illustrates the great importance to maintain an as low as possible R_0 during and after the containment period. The rather long delay between the beginning of the containment and the appearance of the slowing-down of the rate of contamination puts a particular strength on the communication and sanitary education of people. To be mostly efficient, this communication must be done by a locally recognised medical staff. We believe that this point is a crucial matter of success.

COVID-19 | Time-evolution | Guadeloupe | SAMU | Critical care | Monte Carlo Model
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Introduction

The most recent evolution of the pandemic COVID-19 disease in Western Europe indicates that this region is, together with the United States, the new centre of the pandemic spread (e.g. (1) and other reports issued by the World Health Organization). Italy and Spain are confronted with large outbreaks of SARS-CoV-2 infection. In France, the rate of new infections daily increases and measures have already been taken to increase the intensive care capacity of the main hospitals of the country. Also, in order to face with the strong heterogeneity of case number among the different regions in France, medevacs (either by air or railway) have been undertaken to optimally redistribute the most critical patients in the country's intensive care facilities. In this context, the situation of remote French territories like Guadeloupe is particularly critical since, although possible, medevacs should be anticipated with a longer delay because of the distance and the duration of the travels.

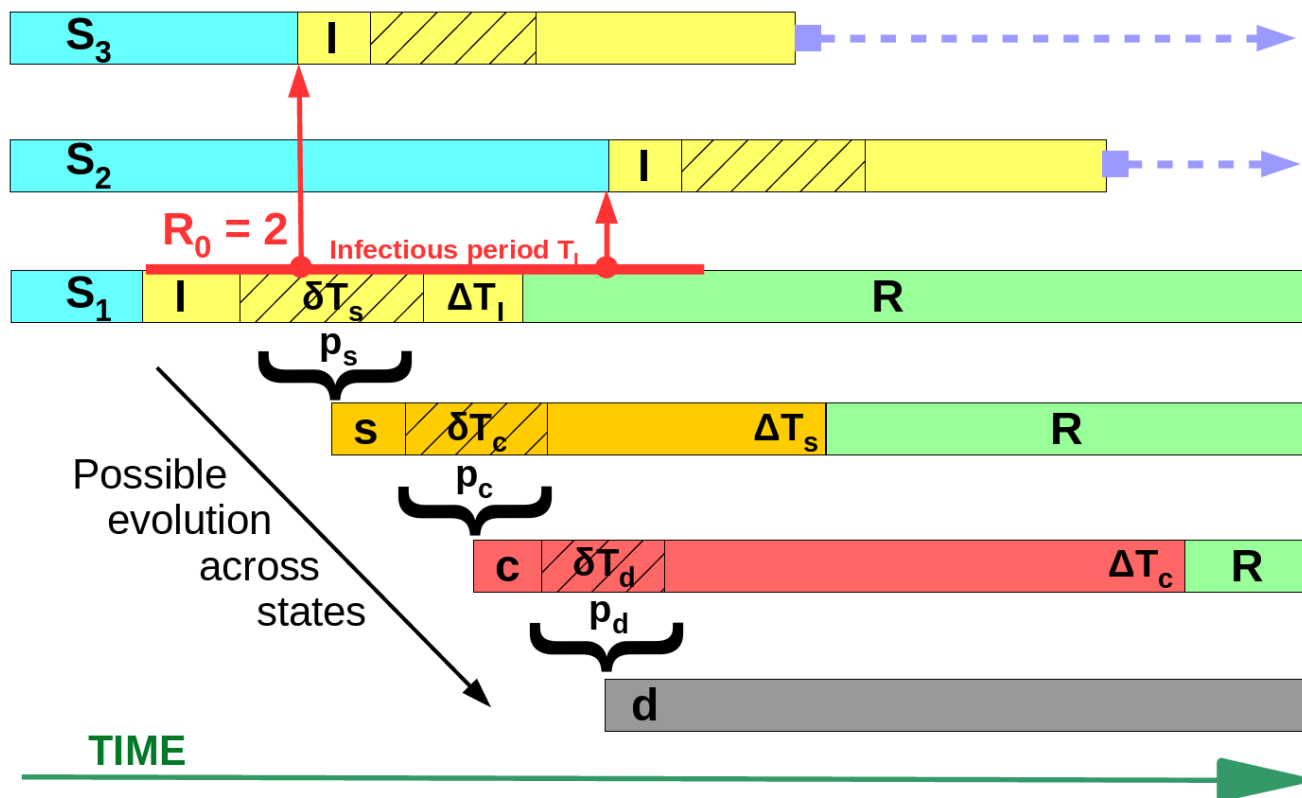
Numerical models of the spread of epidemic diseases may be of some help to anticipate the evolution of the situation in a near-future of several weeks and, eventually, may reveal a likely disruption of the local intensive care capacity. In short, mathematical models may be ranked in two main categories, namely semi-analytical models and numerical stochastic and Monte Carlo models (see (2) for a review). In the former category, the spread of the disease is modelled by a set of coupled differential equations that account for the most important characteristics of the disease. This approach is largely followed (3–5). The second category of models is, in some sense, more straightforward and relies on network models to explicitly considers the individuals constituting the population. Such an approach offers a great versatility to tackle with complicated features, like social interaction matrices, that are difficult to introduce in semi-analytical models. The main drawback of numerical stochastic models is their computer-intensive demand that, for large populations, necessitates the use of multi-scale or coarse-grained algorithms. Thanks to the moderate size of the population of Guadeloupe, no such difficulties are encountered and a straightforward approach is possible.

Method

The technical details of the model are explained in the appendix [Stochastic Monte Carlo model](#), and we here recall its main characteristics. A flowchart of the model is shown in Figure 1. As stated above, all individuals forming the population are considered as nodes in a fully connected network where everyone is able to meet anyone. By using social contact matrices, this full connection could be modified to account for demographic and social heterogeneity. Also, we have not considered the age-dependence of the COVID-19 effects.

Each individual of the network may, temporarily or definitely, be in the following state (Fig. 1): non-infected, infected with minor symptoms ("infectious"), infected with severe symptoms ("severe"), infected critical ("critical"), dead or recovered. In the vocabulary of epidemic modelling, non-infected correspond to the so-called "Susceptibles" and minor infected are "Infectious". In our model, both the severe and critical infected are not considered as infectious because they are isolated in hospital facilities and unable to significantly contaminate others. Although this is statistically justified in our model, actually this assumption is contradicted by the sad death of several French medics.

According to the classical nomenclature, our model is a S_IscR_d model where the lowercase "sc" indicate the transient and non-contaminating nature of these states. To the best of our knowledge at the time of writing this paper, it does not seem that recovered "R" patients are able to again become infectious "I" (6). The deceased "d" patients may remain infectious several days (7) and we assume that they are safely isolated to prevent any contamination.



 Switching period

S = susceptible, I = infectious (minor or no symptoms), s = severe, c = critical, d = deceased, R = recovered (not susceptible again)

Fig. 1. Flowchart of the stochastic modelling procedure. In the general case, a susceptible non-infected person S_1 becomes infected. This new infected I may contaminate a number R_0 of other susceptibles (here S_2 and S_3) during his infected period T_I (red line) which may run beyond the recovery period ΔT_I (in yellow). During the sub-period δT_s (shaded rectangle), the infectious "I" may switch to state "severe" with a probability p_s . If the patient remains in state "I" until the end of the recovery period ΔT_I , he becomes definitively recovered "R". Instead, if the patient switches to state "s", he may either recover at the end of the recovery period ΔT_s or switch with a probability p_c to state critical "c" during the switching period δT_c . The same procedure applies to state critical "c".

Each individual may switch from one state to another with given probabilities. For instance, a "susceptible" may become "infectious", then "severe" and finally "recovered". This example corresponds to the sequence:

$$S \rightarrow I \rightarrow s \rightarrow R. \quad (1)$$

The duration of stay in a given class is variable, depending on the initial health status of each patient. Clinical data collected world-wide put constrains on the possible range of each parameter.

The model is based on an evolutionary scheme where the initial conditions correspond to a non-infected population excepted a small (typically several tens) number of "infectious". Once initialised, the algorithm proceeds by time-steps and, for each time-step, the sequence of evolutionary operations is applied. For instance, for the time-step corresponding to day k of the simulation process:

1. All infectious, severe and critical patients that reached their respective recovery duration (i.e. ΔT_I , ΔT_s , ΔT_c) are definitively switched to the state recovered "R" (Fig. 1).
2. The ensemble of infectious at day k may contaminate susceptibles "S" with a probability given by the R_0 value at day k . By this way, the model is able to account for rapid time-changes of R_0 .
3. All infectious, severe and critical patients that are in their switching period (i.e. δt_s , δt_c and δt_d in Fig. 1) may switch to the next stage with a given probability. This corresponds to the following possible transitions: $I \rightarrow s$, $s \rightarrow c$, $c \rightarrow d$.

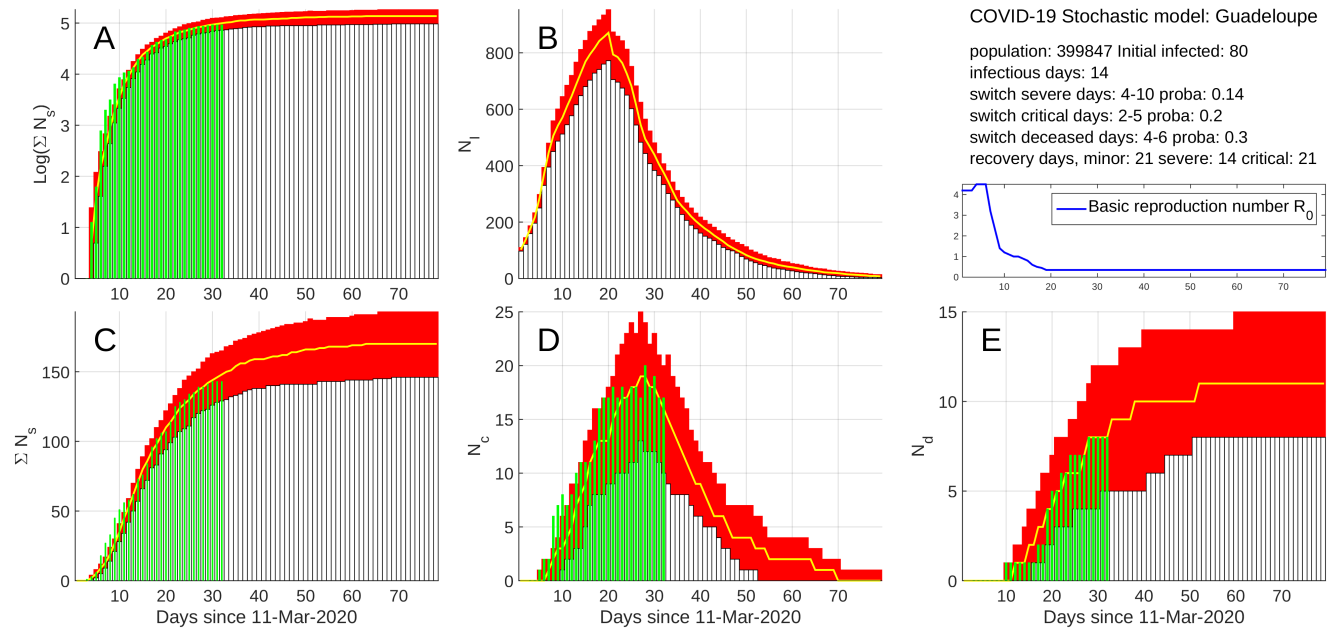


Fig. 2. Model 1 results. A) semi-logarithmic (Natural logarithm) plot of the cumulative number ΣN_s of severe cases. Green bars = data and red bars = model. B) Instantaneous number N_I of infectious. C) same as (A) in linear axis. D) Instantaneous number N_c of critical cases. Green bars = data and red bars = model. E) Cumulative number ΣN_d of deceased patients. Green bars = data and red bars = model. The parameter values used in the model are shown in the upper-right part of the figure together with the time-variation of the basic reproductive number R_0 . The red rectangles represent the 80% confidence interval centred on the median.

Data

The data used in the present study, are daily communicated by the University Hospital to the local authorities, i.e. the Regional Health Agency (Agence Régionale de Santé in French). They correspond to the cumulative number of persons with COVID-19, the cumulative number of deceased patients and the number of patients presently in intensive care units. Detailed data for France are made available by Santé Publique France (8).

Both the cumulative number of deceased patients and the number of patients presently in intensive care units respectively correspond to ΣN_d and N_c in the model. The cumulative number of persons with COVID-19 could be something between ΣN_I and ΣN_s , depending on the screening procedure. In France, a majority of the persons tested for COVID-19 are patients with severe symptoms and admitted in specialised COVID-19 units. Such is the case in Guadeloupe and, consequently, the cumulative number of persons with COVID-19 as announced by ARS correspond to the ΣN_s of the model.

In the present study, we use the data going from March 13 2020 to April 11 2020 shown in Figure 8 of appendix [Bootstrapping method of data analysis](#).

Results

The model derived in the present study is highly non-linear with respect to most parameters, and it is expected that non-unique and significantly different solutions fitting the data might be obtained. This could be performed by the means of non-linear inverse methods like simulated annealing (9, 10) and will be presented in a forthcoming study. In the present study, the Z_I and time-varying R_0 parameters are adjusted with the Nelder-Mead downhill simplex (11, 12). The other parameters are determined with clinical observations in the Guadeloupe hospital and data published in the abundant literature concerning COVID-19.

Figure 2 shows the results for model 1. The time variations of R_0 have been adjusted to reproduce the flattening visible in the ΣN_s data from day 21. The corresponding values of the parameters are recalled in the upper-right panel of the Figure. This model provides a good fit to all data ΣN_s , N_c and ΣN_d . In order to reproduce the initial rapid exponential increase observed before day 10, quite large values of $R_0 = [4.2 \ 4.2 \ 4.2 \ 4.5 \ 4.5 \ 4.5]$ are found for the first 6 days of the simulation (day 1 corresponds to March 11, 2020). These high R_0 are obtained during the week before municipal elections when meetings occurred and were probably places of high contamination rates (13, 14). This could explain the high R_0 values found with the model.

Interestingly, the large R_0 from days 1 to 6 must be combined with a large $Z_I = 80$ to fit the sharp onset of the ΣN_s curve (Fig. 2A). The reasons for such a large number of initial infectious remain unknown, but we may suspect either a massive arrival of infected aircraft or ship passengers or the existence of several infectious spots like funeral wakes or election meetings as mentioned above.

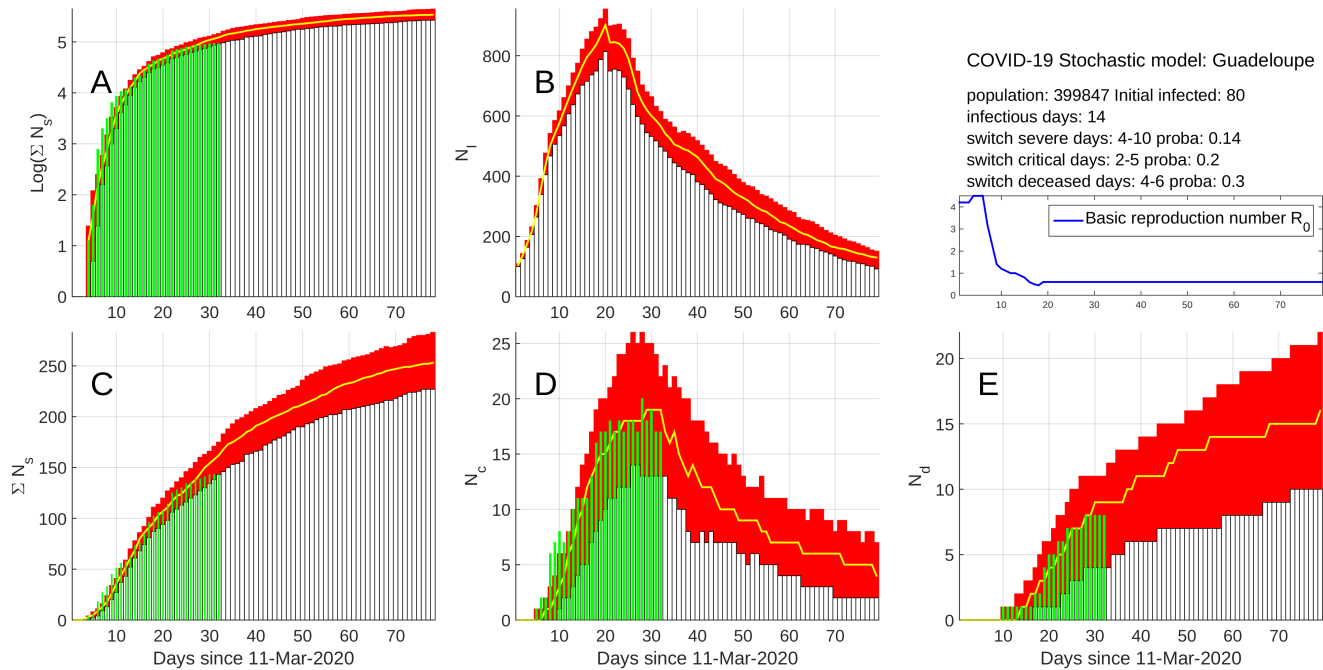


Fig. 3. Results for model 2 with the same parameters as model 1 (Fig. 2) excepted for the containment $R_0 = 0.6$ from day 19 (March 29).

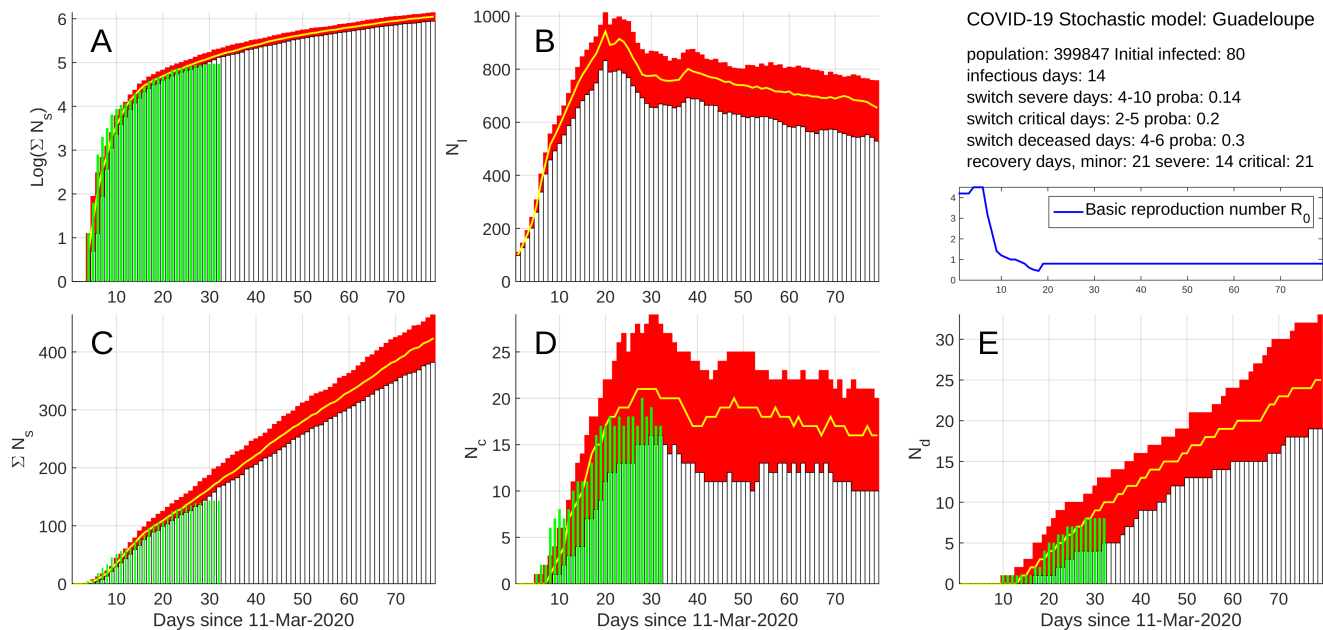


Fig. 4. Results for model 3 with the same parameters as model 1 (Fig. 2) excepted for the containment $R_0 = 0.8$ from day 19 (March 29).

In order to fit the strong inverse curvature of the $\log(\Sigma N_s)$ curve between days 10 to 20, R_0 must be gradually decreased from day 7 (March 17) to day 18 (March 28): $R_0 = [3.2 \ 2.3 \ 1.4 \ 1.2 \ 1.1 \ 1.0 \ 1.0 \ 0.9 \ 0.8 \ 0.6 \ 0.5 \ 0.45]$. The starting date of the decrease corresponds to the beginning following the second speech of the President of the French Republic, Mr Macron, on March 16. To reproduce the flat almost horizontal end of the data from day 21 (March 31), it is necessary to reduce $R_0 = 0.35$ from day 19 (March 29) until the end of the process (i.e. day 80).

With this model, the maximum $N_c = 25 \pm 3$ is reached near day 28 (April 7) about 3 weeks after the beginning of the containment. After this date, the number of critical cases sharply decreases to reach a low base level about 4 weeks later, i.e. during the first week of May.

To illustrate the role played by R_0 during the containment period, we present the results for 2 models with the same parameter values as for model 1 excepted during the containment. We set $R_0 = 0.6$ in model 2 (Fig. 3) and $R_0 = 0.8$ for model 3 (Fig.

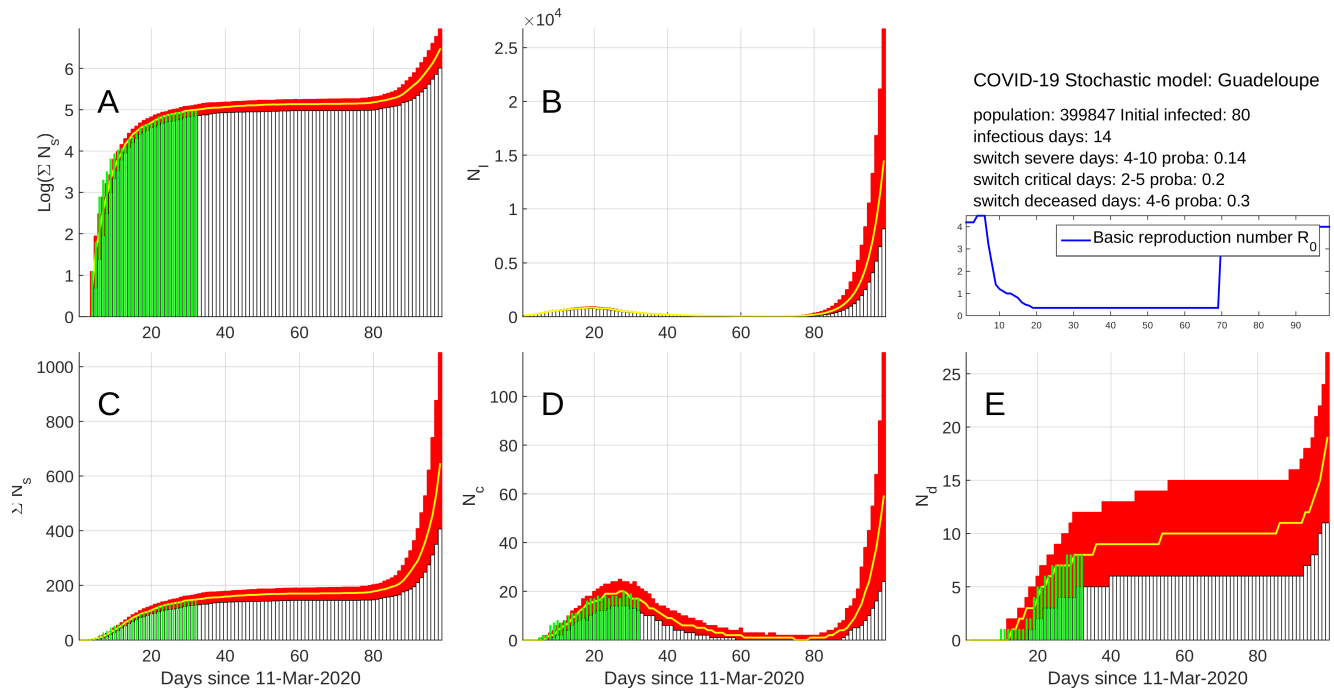


Fig. 5. Effects of uncontrolled end of containment. Same model as in Figure 2 but with an uncontrolled end of containment at day 70 with a sudden resetting of $R_0 = 4.0$. The model indicates that, after approximately one month of low-level infectious spread, the number of cases again dramatically increases after day 90.

4). Both models 2 and 3 fit the data as well as model 1 excepted for the flattening part of the ΣN_s data after day 21 (March 31). This indicates that the determination of R_0 during the containment is constrained only by the most recent data values. The quality and reliability of these data is then of a great importance to derive models able to predict an eventual decrease of critical cases.

Model 2 (Fig. 3) corresponds to the containment $R_0 = 0.6$ and gives a maximum number of critical cases of the same order and at the same date as the one obtained with model 1 (Fig. 2). However, the decrease following the maximum is less steep and low values are reached about 3 weeks later with respect to what is observed with model 1. This translates into a larger cumulative number of critical case and, consequently, in a larger number of deceased patients (compare Fig. 2E and Fig. 3E).

Model 3 (Fig. 4) corresponds to $R_0 = 0.8$. As can be verified in Figure 4A,C, the flattening of the data after day 21 (March 31) is poorly reproduced making this model less likely than models 1 and 2. However, owing that the flattening of the ΣN_s curve relies on a small part of the most recent data, this pessimistic model cannot be totally excluded at the time of writing this article. The maximum values of critical cases may reach a maximum of 30 critical patient followed by a plateau with a small slope during which the instantaneous number N_c remains around 15-20 one month after the date of the maximum. This correspond to a situation where the treatment of numerous critical patients must be sustained during a long period, implying the disposal of a sufficiently large medical staff and amount of equipment. As can be observed in Figure 4E the number of deceased patients increases steadily.

Concluding remarks

A common characteristic to all 3 models presented above, is the need of a quite large number $Z_I = 80$ of initial infectious persons coupled with a large $R_0 \simeq 4$ at the beginning of the epidemic spread. This large Z_I could be explained either by the importation of a large number of infected persons or the presence of several super contaminators able to contaminate tens of persons during meetings in a short period of time (see (13, 14) for the effects of mass gathering). Let us remark that large R_0 are reported by others; for instance, Tang et al. report values as high as 6.47 for data from China. These authors mention that this high R_0 corresponds to data collected during a period of intensive social contacts (i.e. before the Chinese New Year). Mizumoto and Chowell report R_0 values as high as 10 for the case of Diamond Princess, and for the same data Rocklöv et al. find a maximum $R_0 = 14.8$ and a 8-fold reduction to $R_0 = 1.78$ during isolation and quarantine.

Another characteristic of the model is the need to significantly reduce R_0 to fit the decelerating curvature of the ΣN_I data curve (e.g. Fig. 2A). This reduction is delayed by about 3 days with respect to the beginning of the containment and confirms an overall good respect of the social distancing rules by the population of Guadeloupe. Several French national media published articles stating that Guadeloupe was relatively spared from the disease (18). Such a claim could have triggered a common sense reflex of protection applied through social distancing and usage of rules of hygiene. To fit the most recent ΣN_I data, a low

$R_0 = 0.35$ must be applied. If true, this would indicate that people of Guadeloupe continued to improve their social behaviour during the 3 weeks after the beginning of the containment.

The models allows to get an estimate of the number of instantaneous infectious N_I and cumulative recovered patients ΣN_R . In absence of systematic detection of COVID-19 among the population, no N_I data are available and the N_I curve is actually indirectly constrained by the fit to the ΣN_s and N_c data and by the switching probabilities p_s and p_c . However, the values given to these probabilities fall in ranges widely recognised by the medical community and we may safely consider them sufficiently reliable to give credit to the modelled N_I and ΣN_R curves. A simple assessment may be done by dividing ΣN_d by the observed number of deceased patients. On day 28 (April 7), this ratio equals 0.7%, a value slightly lower than the generally recognised ratio of $1 - 2\%$ (19).

For the models discussed above, several thousands of persons have been infected and a large fraction of them have recovered and are supposed protected against another infection. However, these supposedly protected persons represent a relatively small part of the total population and the number of susceptible persons remains sufficiently large to ensure a restart of a second epidemic spread of the disease. This is shown in Figure 5 which represents a long-term simulation with model 1 as in Figure 2 but with an abrupt resetting of $R_0 = 4.0$ at day 70 (mid-May), about 2 months after the beginning of the containment. This corresponds to a situation of uncontrolled end of containment. Because of the existence of only several infectious cases, the spread of the virus proceed at a low-level during approximately 3 weeks (i.e. until day 90) before exponentially exploding again into a second epidemic crisis. These results illustrate the future difficulty to control such a restart of the virus propagation and the necessity to maintain a low R_0 for a long period of time. The simulation shown in Figure 5 assumes that the patient who recovered during the first epidemic crisis cannot be infected during the second crisis, a medical assumption that remains to be confirmed.

ACKNOWLEDGEMENTS

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Supplementary Note 1: Stochastic Monte Carlo model

A. Description of the model structure. The stochastic model used in the present study falls in the class of exact stochastic Monte Carlo models (2). In such models, the entire population exposed to an infection is represented by a network where each node represents a person. This type of models offers very large possibilities to design epidemic processes that agree with the medical knowledge of the virus dissemination and its possible pathological issues.

A set of stochastic rules determines the evolution of each node at each time iteration. The infection simulation process begins by choosing a given number of nodes initialised as "infected" to put the pathogen in the network. Other initial conditions can be imposed to fix the state of the population. For instance, we may fix the number of vaccinated persons. In the case of COVID-19 for which no vaccine exists at the present time, the main initial condition is to fix the number Z_I of initially infected individuals. The next phase of the modelling is a loop over the time steps covering the period to simulate. For each time-step, the state of each node is eventually modified depending on its present stage and according to the set of stochastic rules that define the way by which the infectious disease propagates (a flowchart of the stochastic process is shown in Figure 1).

In our model, we define 6 different possible states for the nodes (Fig. 1):

1. **state "S" for susceptible** corresponds to non-infected people and likely to become infected. In this sense, state "S" is also equivalent to the commonly defined "exposed" state. We note N_S instantaneous number of "S" persons.
2. **state "I" for infectious** is for people infected by COVID-19 and presenting either no or only minor symptoms. These persons are likely to remain undetected by the medical services and expected to pursue their daily activities and maintain contacts with other people. By this way, they are the primary cause of infection diffusion among the set of "S" nodes. The basic reproductive number, R_0 , defined as the likely number of "S" infected by a single "I" case during his infectious period T_I . We note N_I the instantaneous number of "I" patients.
3. **state "s" for severe** is for people with enough severe symptoms to either see an urban doctor or be admitted in a hospital. These patients are considered to be isolated either at home or at the hospital and are no more able to infect other people. We note N_s the instantaneous number of "s" patients and ΣN_s the corresponding cumulative number.
4. **state "c" for critical** is for patients in a critical state and necessitating intensive care in hospitals. As for "c", these patients are considered isolated from the "S" population and unable to infect others. We note N_c the instantaneous number of "c" patients.
5. **state "R" for recovered** is for patients "I", "s" or "c" that recovered after a period of time that depends on the considered state. The recovery periods will be respectively written ΔT_I , ΔT_s and ΔT_c for states "I", "s" and "c". In the specific case of COVID-19, the main medical opinion is that "R" persons are protected against a new infection by the virus. We note ΣN_R the cumulative number of "R" patients.
6. **state "d" for deceased** patients. We note ΣN_d the cumulative number of "d" patients.

A stochastic set of rules determines the probability to switch from one state to another. In our model, these rules are (Fig. 1):

1. **rule $S \rightarrow I$ determines the condition to switch from non-infected to infected.** The main parameters of this rule are the infectious period T_I and the basic reproductive number R_0 . In our model, this rule is applied to each new "I" node, i.e. nodes that were "S" one day before. For such new "I", an average number of R_0 are randomly taken among the "S" persons and are randomly set in state "I" in the next T_I days.
2. **rule $I \rightarrow s$ determines the conditions to switch from infectious to state severe.** This is controlled by a probability level p_s .
3. **rule $s \rightarrow c$ determines the conditions for a patient with severe symptoms to become critical** and will be admitted in a critical care unit. This is controlled by a probability level p_c .
4. **rule $c \rightarrow d$ determines the conditions for a critical patient to die.** This is controlled by a probability level p_d .
5. **rule $* \rightarrow R$ represents the switch to state "recovered".** This transition applies to "I", "s" and "c" states with probability 1 as long as the patients respectively remained in their state for a duration of ΔT_I , ΔT_s and ΔT_c .

The explicit definition of the rules and the fact that they apply to each node of the network provides a great flexibility to account for more or less sophisticated conditions. For instance, the switching probabilities may easily account for the age of each person. Also, and indeed the model does it, we may consider that a switch from one state to another takes place in a given time interval whose duration is constrained by clinical data. The model is also able to use a time-varying basic reproductive number $R_0(t_k)$ in order to account for the effects of containment and social isolation. The nodes may be assigned to different subsets in order to define regions with given populations. Rules may be defined to account for interactions between regions. In the present study, this possibility has not been implemented due to the lack of data to constrain the process.

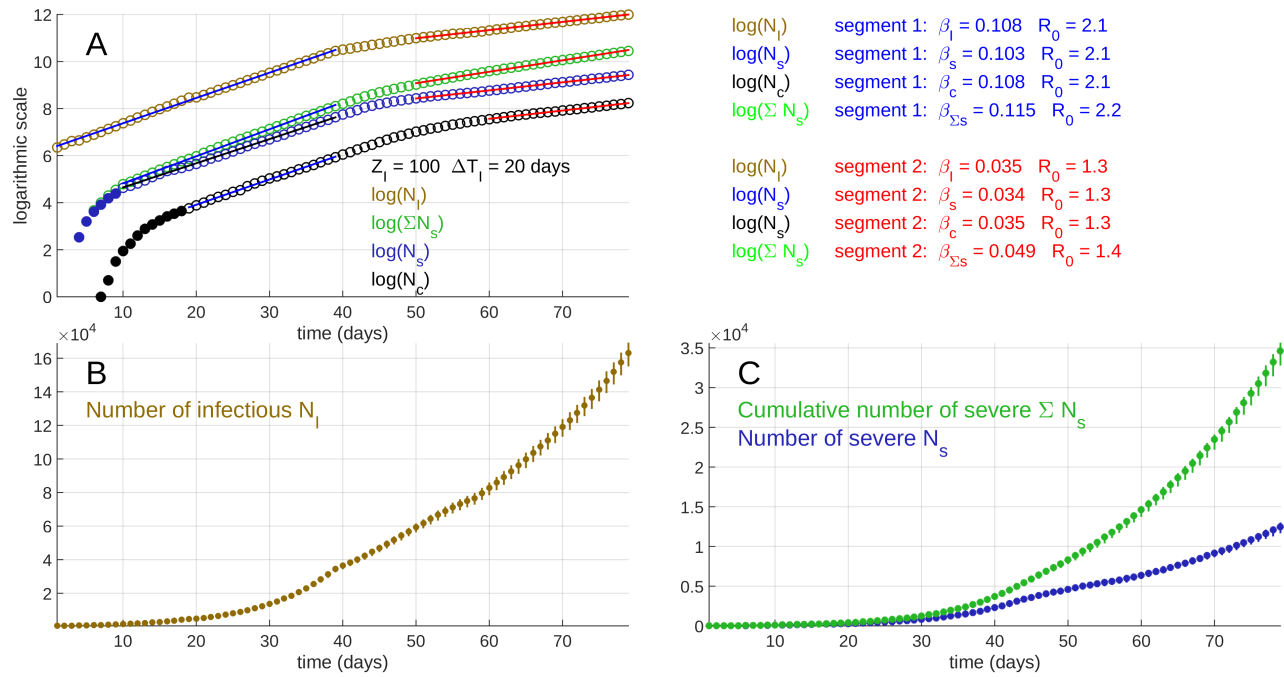


Fig. 6. Stochastic simulation for a period of 80 days. B) time variations of N_I . C) time variations of ΣN_s and N_s (D). A) The same curves together with N_c displayed in a semi-logarithmic graph (Natural logarithm). The upper right panel of the figure shows the parameters β obtained for the 8 linear segments identified in the curves shown in (A). The R_0 values have been derived from β using equation 2 with $T_I = 14$ days. The model started with $Z_I = 500$ initial infectious "I" and $R_0 = 2.0$. At day 40, the basic reproduction number changed to $R_0 = 1.2$. During all the process, the other parameters remained unchanged: $p_s = 0.14$, $p_c = 0.25$, $p_d = 0.20$, $\Delta T_I = 20$ days, $\Delta T_s = 14$ days, $\Delta T_c = 21$ days, and the switch periods $\delta T_s = [4 - 10]$ days, $\delta T_c = [3 - 9]$ days, $\delta T_d = [3 - 8]$ days.

B. Model parameters. The model is totally determined by the following parameters:

1. the total number N of nodes, i.e. of persons forming the population. For Guadeloupe, we set $N = 399847$ from the age distribution (20).
2. the number Z_I of initial "I".
3. the basic reproductive number, R_0 . This parameter may be time-varying in order to account for different social behaviours. It is generally assumed that R_0 is large for COVID-19, and the range of possible values is large (21). In the present study, we experimentally determine the value of R_0 that best reproduces the observed data. This point will be discussed in details in section [Bootstrapping method of data analysis](#).
4. the infectious period T_I is typically assumed to be of the order of 20 days with possible values as large as 37 days in some exceptional circumstances. In the present study, we determine a value for T_I that best matches with both the data and the prior assumptions taken other studies. This point is considered in section [Bootstrapping method of data analysis](#).
5. the recovery periods ΔT_I , ΔT_s and ΔT_c are constrained by clinical data.
6. the switching probabilities p_s , p_c , and p_d are constrained by clinical data. These probabilities are completed by switch periods, δT_s , δT_c and δT_d during which a given state "I", "s" and "c" may respectively switch to "s", "c" and "d".

C. Simulation examples. In this section we present several simulations to illustrate the effects of the key parameters of the model. This will help the reader to understand where information able to put constraints on the parameters can be obtained from the data processed in section [Bootstrapping method of data analysis](#). In order to quantify the random fluctuations due to the stochastic nature of the model, each simulation is performed 20 times to compute the median and the confidence intervals of the results.

The first simulation corresponds to a duration of 80 days with a basic reproduction number $R_0 = 2.0$ from day 1 to day 39, and $R_0 = 1.2$ afterwards. The model started with $Z_I = 500$ initial infected "I" and the duration of the infectious period is fixed to $T_I = 14$ days. All other parameters are kept fixed during the process: $p_s = 0.14$, $p_c = 0.25$, $p_d = 0.20$, $\Delta T_I = 20$ days, $\Delta T_s = 14$ days, $\Delta T_c = 21$ days, and the switch periods $\delta T_s = [4 - 10]$ days, $\delta T_c = [3 - 9]$ days, $\delta T_d = [3 - 8]$ days.

Figure 6 shows the results of the simulation for the time variations of N_I (Fig. 6B), and of ΣN_s and N_s (Fig. 6C). The three curves together with N_c are represented in a common semi-logarithmic graph in Figure 6A. The Natural logarithm is used throughout the present paper.

As a starting point for the discussion, it can first be observed that the time variations of N_I and N_s significantly depart from a pure exponential pattern, particularly because of the presence of smooth bumps in the curves around day 55. These bumps can be better understood in the semi-logarithmic plot of Figure 6A where the N_I and N_s curves appear partly linear in two segments. The same linear segments are also visible in the N_c curve. The presence of linear segment in the curves indicates an exponential time variation. In the N_I curve (orange symbols), a first linear segment goes from day 1 until day 39 with slope $\beta = 0.108$. A second linear segment with slope $\beta = 0.035$ starts at day 50. A curved segment locates in between the two linear segments, from day 40 to day 50.

The slopes β of the linear segments are related to the basic reproductive number through,

$$R_0 = \exp\left(\beta \times \frac{T_I}{2}\right). \quad (2)$$

In the present example, taking $T_I = 14$ days, we have $R_0 \simeq 2.1$ for the first segment of N_I , N_s and N_c . For the second segment, $R_0 \simeq 1.3$ for curves N_I , N_s and N_c . These values agree very well with the input values used in the model. The linear segment of N_I is slightly biased by the presence of a small jump at day 20 that corresponds to the recovery time $\Delta T_I = 20$ days of the initially infected patients (i.e. 500 persons in this simulation). This cohort of initial infectious massively contaminates $Z_I \times R_0 = 1000$ persons, some of these initials switched to state "s" but most of them (i.e. $\simeq Z_I \times (1 - p_s) = 430$) recovered and suddenly switched to state "R" at day 20. This produces a sharp decrease of the instantaneous number of infectious patients in the N_I curve. This jump is transmitted in the other curves but highly blurred by the switching process (through stochastic causal convolutions).

The parameter β is a primary quantum of information that can be obtained from the data, and equation 2 shows that the parameters R_0 and T_I are linked:

$$\log(R_0) \times \frac{2}{T_I} = \beta. \quad (3)$$

This equation shows that, if β is the only information available, the pairs of parameters $[R_0, T_I]$ cannot be determined uniquely unless additional information is available through the knowledge of either R_0 or T_I .

Information about T_I can be obtained by recognising that this period of time corresponds to the duration of the smooth curved segment that separates the two linear segments discussed above. In curve N_I , the curved segment starts at day 40 when the change of R_0 occurs. However, because new infectious patients do not immediately contaminate others but instead do that during the period of time T_I , an abrupt change of R_0 appears smoothed. Consequently, this is only after day 50, that a linear segment corresponding to the new value of R_0 appears in the N_I curve. This phenomena is of a considerable practical importance because it represents a latency (or an inertia) of the control measures taken by the authorities to reduce and extinct the epidemic process. Such a latency has to be clearly explained to the population in order to encourage people to maintain their efforts to remain in containment.

The two linear segments of the N_s curve (Fig. 6A) are delayed by 9 days with respect to the segments of the N_I curve. This duration of 9 days corresponds to the onset period of the N_s curve during days 1 to 9 of the process (filled blue dots in Fig. 6A). The delay of 9 days is caused by existence of the time period $\delta T_s = [ts_1, ts_2]$ during which a "I" patient is able to become "s" (in this simulation, δT_s runs from day 4 to day 10). Consequently, the first "s" patients begin to appear after a delay of ts_1 days (i.e. 4 days in this example) and all "s" patients are created at day $ts_2 + 10$. This explains the duration and the shape of the onset period visible at the beginning of the N_s curve. Consequently, the onset period of the N_s curve may provide information about the switch period δT_s . The same onset phenomena is observed in the N_c curve but with a delay equals to the sum of $ts_1 + tc_1 = 7$ days. The end of the onset period falls at day $ts_2 + tc_2 = 19$.

We now turn to the case of the ΣN_s curve (green circles in Fig. 6A) which is particularly important because it generally corresponds to the available data. Contrarily to the instantaneous quantities N_I and N_s which give the number of either "I" or "s" patient at a given time, ΣN_s is a cumulative quantity which gives the total number of patients who passed by stage "s" anytime before present. We emphasise that this quantity is NOT the integral of N_s and, as a consequence, the slopes of the linear segments present in the ΣN_s curve are not simply related to those of the N_s curve. Indeed, a careful examination of the ΣN_s reveals that the segments are not strictly linear. At the beginning of the process, we have $\Sigma N_s = N_s$ until the end of the time periods where first "s" patients begin to switch either to the state "R" or "c". At that time, the two curves begin to diverge. The slopes of the linear segments in ΣN_s are always slightly larger than the slopes of N_s and the formula 2 and 3 are no more exact for the ΣN_s case. Indeed, the R_0 values derived for ΣN_s in the example (upper right part of Fig. 6) are significantly biased, and to obtain reliable R_0 estimates, it is necessary to use data at the very end of the process, in the narrow time-window comprised between the end of the onset period and the beginning of the switching from "s" to "c".

The size of the confidence intervals appears constant in the semi-logarithmic plots (Fig. 6A). This is typical of a multiplicative noise where the amplitude of the statistical fluctuations is proportional to the data amplitude as can be checked in Figure 6B,D.

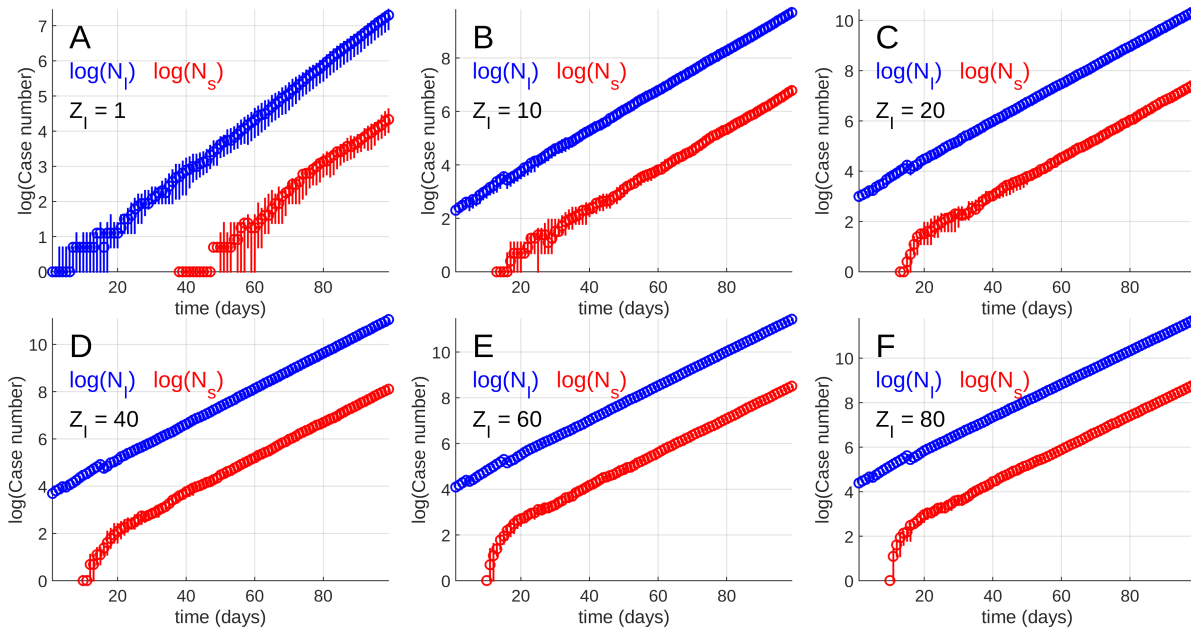


Fig. 7. Stochastic simulations obtained for 6 different values of the initial number of infectious. The value of Z_I is indicated on each subplot.

In our case, this multiplicative noise may be explained by the growing Brownian divergence of some random walks in the network. Practically, this conducts to the appearance of some outlier simulations and justifies the use of the median.

We now address another important characteristic of the epidemic process through the random variations occurring at the very beginning of the process. The features we want to discuss are illustrated in Figure 7 where the plots have been obtained by running the model with a different number on initial infected Z_I . In the case of rather small values of Z_I (i.e. 1, 10 or 20 in Fig. 7A,B,C), random fluctuations perturb the beginning of the curves, with a longer persistence for the N_s curve. For larger values of Z_I (i.e. 40, 60 or 80 in Fig. 7D,E,F), the random fluctuations almost disappear while the starting sequence becomes steeper. Consequently, a careful observation of the starting sequence may provide some information about the number Z_I of initial infectious persons. Let us remark that these features can only be obtained with a stochastic model as the one developed in the present study.

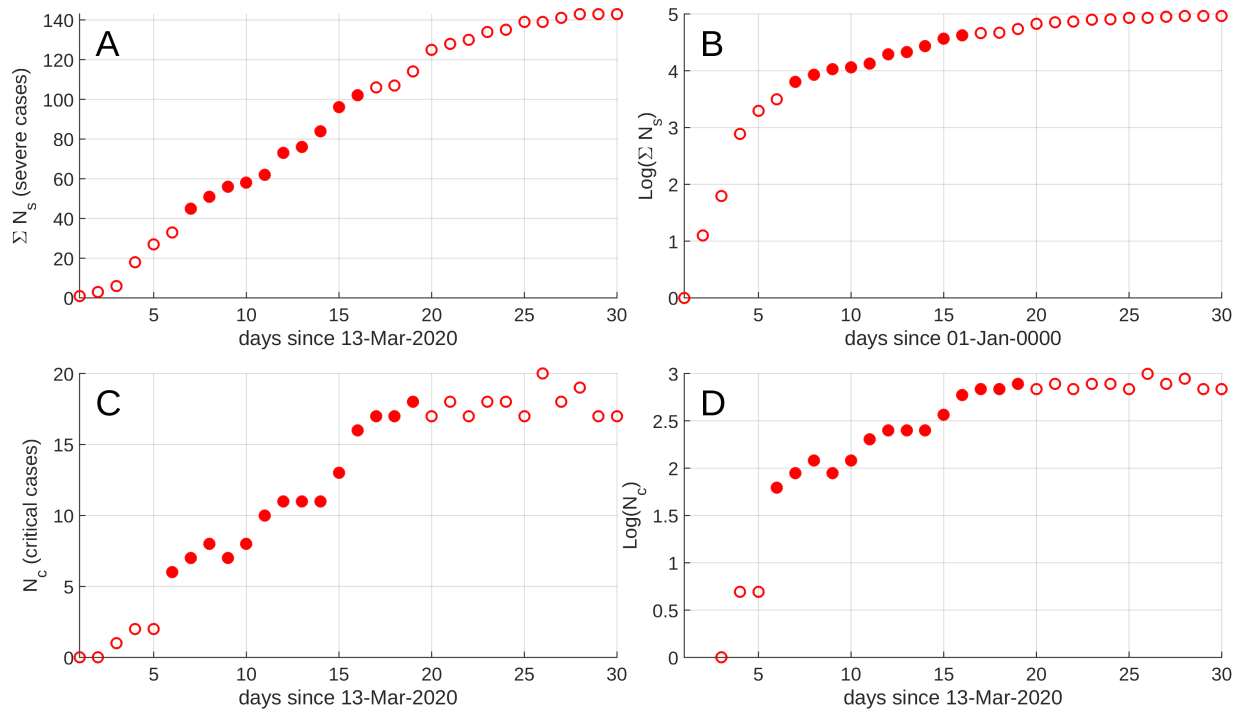


Fig. 8. Data used in the present study. Number ΣN_s of severe cases (A) and N_c of critical cases (C) as a function of time. In plots B and D, the data are presented in a semi-logarithmic graph. The filled symbols represent the data points used in the bootstrap computations and are assumed to belong to the linear part of the semi-logarithmic curves when an exponential regime is established.

Supplementary Note 2: Bootstrapping method of data analysis

A. Presentation of the data. This section gives details on the method used for the data analysis and presents the results used to constrain the stochastic model presented in section [Stochastic Monte Carlo model](#).

Figure 8 shows the data used in the present study, namely the numbers N_s and N_c of severe and critical case observed in Guadeloupe from March 13th until April 2 of year 2020. These data are presented in both linear and semi-logarithmic plots in order to better emphasise a possible exponential-like pattern. Because of the small number of data available at the time of writing the present paper, the exponential increase of either N_s or N_c is not as conspicuous as for the synthetic case presented in Figure 6. However, for the data set N_s , the semi-logarithmic plot (Fig. 8B) is reasonably linear in the [day 7 – day 16] period. The first 6 points are expected to correspond to the δT_s onset period of 6 to 7 days. For the N_c data, a linear segment may be identified in the [day 6 – day 19] period.

By comparing the onset period in the data with the simulation results of Figure 7, we may claim that the onset sequence of the data curve N_s corresponds to a rather large number of at least 80 initial infectious persons. These persons could for instance be passengers of an aircraft or members of a group infected by a single infectious during a meeting.

B. Data bootstrapping and parameter determination. In order to determine the β parameter and its uncertainty limits from the small-size data sets of Figure 8, we use a bootstrapping approach (22). Let us recall that this method relies on a statistical resampling of the data sets in order to reconstitute the statistical variability of the estimated parameter β . In the present study, we performed 1000 bootstrap resamplings for each data set N_s and N_c , and the so-obtained 1000 estimates of β may be used to compute the probability density kernels shown in Figure 9A. The two probability distributions are poorly statistically coherent with a small overlap of the two curves. Equation 2 may be used to compute R_0 (using $T_I = 14$ days) from the β probability curves. The estimate for $R_0 \simeq 1.85 \pm 0.03$ is coherent with the values published by Li et al. (21) who found $R_0 = 2.2$ with a 95% confidence interval [1.4 – 3.9].

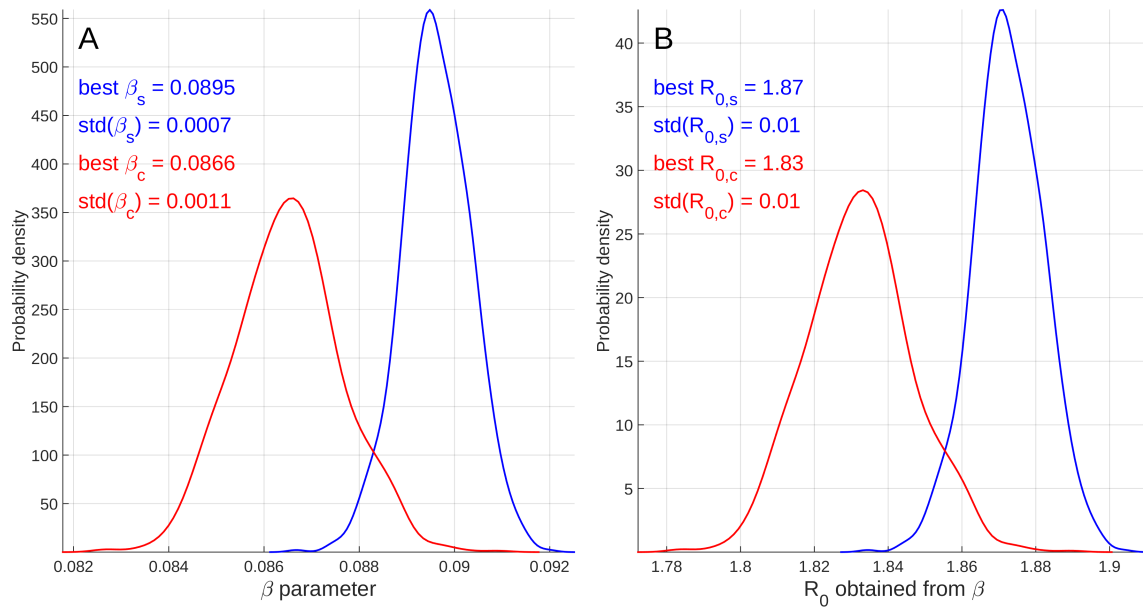


Fig. 9. A) Probability density kernels for the parameters β_s and β_c obtained by respectively bootstrapping the data ΣN_s and N_c . The data used are represented as filled symbols in Figure 8. B) Kernels for R_0 obtained by applying equation 2 to the bootstrapped parameters β_s and β_c .