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Abstract

We construct a new model for the comprehension of the Covid-19 dynamics in Cameroon. We present the basic reproduction number and perform some numerical analysis on the possible outcomes of the epidemic. The major results are the possibilities to have several peaks before the end of the first outbreak for a uniform strategy, and the danger to have a severe peak after the adoption of a careless strategy of barrier anti-Covid-19 measures that follow a good containment period.

Keywords: Cameroon; Covid-19; Barrier measures; Sensitivity analysis; Multi-peaks; Numerical simulation; Epidemiological model; Basic reproduction number; Stability; Equilibrium

MSC 2010 No.: 34A12, 92B05

1. Introduction

Covid-19 (Corona virus disease 2019) is a SARS-like virus that started in China in 2019 (Adamik et al. (2020); Magal et al. (2020); Liu et al. (2020a); Liu et al. (2020b); Ngonghala et al. (2020); Nneck and Ebangue (2020); Tang et al. (2020); Kwok et al. (2019); Shahid et al. (2020); Tappe (2020); Covid-19 (Cameroon) (2020); Zio et al. (2020)) and concerns almost all the countries in the world.

This disease is not well understood but several papers attempted to its dynamics using statistics or differential equations. Adam (2020) simulates the world's response to COVID-19 in terms of number of tests and number of new cases. Adamik et al. (2020) analyzes the data in some countries and show that the herd immunity strategy for COVID-19 is likely to fail. Then, it seems that Covid-19 is not an immunizing disease in the usual sense. AP News (2020) presents the fact that South Korea sees mass COVID-19 cases linked to night clubs. This reveals the danger to see a new outbreak of the disease after the containment period. Hasell et al. (2020) presents the world map of the total tests performed relative to the size of population. It is very interesting to see that even developed countries have difficulties to conduct massive test campaigns.

Kwok et al. (2019) studies the epidemic models of contact tracing through a systematic review of transmission studies of severe acute respiratory syndrome and middle east respiratory syndrome: it helps to partly understand the complex spread of the Covid-19. Magal et al. (2020) predicts the number of reported and unreported cases for the COVID-19 epidemic in South Korea, Italy, France and Germany through a mathematical model. Their study practically evaluates the impact of asymptomatic infectious. The same team in Liu et al. (2020a) uses again a compartmental modeling to predict the cumulative number of cases for the COVID-19 epidemic in China from early data, and focuses again on the importance of reported cases and unreported cases. Liu et al. (2020b) continues the work in understanding unreported cases in the COVID-19 epidemic outbreak in Wuhan (China), and the importance of major public health interventions. Shahid et al. (2020) presents a short-term predictions and prevention strategies for COVID-2019 through a model based study able to support government strategies of containment. Ngonghala et al. (2020) uses also mathematical modeling to measure the impact of non-pharmaceutical interventions on curtailing the Covid-19 since the treatment of Covid-19 is not fixed (HydroxyChloriquine or not?).

An interesting paper (Pedro et al. (2020)) studies conditions for a second wave of COVID-19 due to interactions between disease dynamics and social processes interpreted as the outcomes of non-linear interactions between disease dynamics and population behaviour. Countries like Cameroon have their own reality (economical and government strength and weakness). That is why some studies started to use other methods like parameter estimation (as Bayesian estimation for Zio et al. (2020)) in some African countries. All these works, even if they are globally essential, are not focused on the African realities through the force of infection. Hence, there is still a need to elaborate in the Cameroonian context a very realistic model and a force of infection that consider different impacts of several subpopulations/compartments of COVID-19 infectious individuals. The distinction of several compartments of infectious individuals allow us to make previsions after the first peak and useful recommendations to public health planners.

Cameroon (Central Africa) officially recorded its first case on the fifth of March 2020 (Ministry of Public Health of Cameroon (2020); Nkeck and Ebangue (2020); Covid-19 (Cameroon) (2020); CSSEGIS (2020)). As other countries affected by Covid-19, Cameroon adopts a containment strategy with barrier measures during the first weeks of the outbreak (Cameroon's Prime Ministry (2020)).

In this paper, we focus, with the Cameroonian context, on the prevention of the possible multi-peaks during the epidemic of Covid-19 with the caution of the neglect of barrier measures after the first Covid-19 outbreak. In fact, many Cameroonians think that the relaxation of the government constraints for bars, markets and night-clubs, implies the end of the disease (JdC (2020)). This idea could lead to a dramatic restart of the epidemic if we use Cameroonians data as initial values (see Figures 1 in Section 5). The South Korean case (AP News (2020); News UN (2020); VOA News (2020)) suggests the multi-peaks assumption studied here. Compared to previous publications on COVID-19, this model uses a more realistic force of infection with the key states of infectious individuals (isolated/hospitalized humans and quarantined individuals, reported or unreported) and susceptible individuals (free, isolated and contained), in order to predict the period of the first peak and the possible other peaks in the sequel in the case where the sanitary conditions are neglected by the population. Our main conclusion is to continue to follow strictly and individually the healthy anti-Covid-19 actions (hands washing, wear of the face mask, etc.) since it is compulsory to re-launch social and economical activities as going to work if we want to avoid a catastrophic crisis.

The paper is organised as follows. Section (2) presents the model. Section (3) analyzes this model (well-posedness and dynamical properties). Section 4 makes a study on the sensitivity analysis on the basic reproduction number for some parameters. Section (5) comments several simulations with the possibility of multi-peaks and strategies. Section (6) discusses about the results, and Section (7) concludes the paper.

2. Model Description

Many mathematical models of COVID are proposed, respecting specifications according to the countries. We propose a variant of the COVID model for the case of Cameroon. The total population is subdivided into ten subgroups, namely the free susceptible $S_f(t)$ representing the individuals susceptible to contracting the virus, the confined susceptible $S_c(t)$ even for reasons not directly linked to Covid-19, the isolated susceptible $S_i(t)$ representing those who are brought under control following a case detected in their home, the infected $E(t)$, the infectious reported $I_r(t)$ representing those identified following a test, the infectious not reported $I_u(t)$, the infectious hospitalized $I_h(t)$ representing those who make the serious forms of the disease, the infectious isolated $I_i(t)$ representing those placed under surveillance and not presenting a worrying clinical aspect, the treated $T(t)$ representing those who have recovered from COVID and finally the immune $R(t)$ representing those who have acquired a relative immunity following infection with COVID. The total population is $N(t) = S_f(t) + S_i(t) + S_c(t) + E(t) + I_r(t) + I_u(t) + I_h(t) + I_i(t) + T(t) + R(t)$. All recruitments are made only through the free susceptible class. A susceptible person following contact with an infectious (reported, unreported, hospitalized or isolated) or a treated patient can

become infected at a rate β representing the force of infection and defined by

$$\beta = \frac{(1 - \omega_r)\beta_r I_r + (1 - \omega_u)\beta_u I_u + (1 - \omega_i)\beta_i I_i + (1 - \omega_h)\beta_h I_h + (1 - \omega_t)\beta_t T}{N - S_c - S_i - \omega_r I_r - \omega_u I_u - \omega_i I_i - \omega_h I_h - \omega_t T},$$

where $\beta_r, \beta_u, \beta_i, \beta_h$ and β_t represent respectively transmission rates of infectious reported, not reported, isolated, hospitalized and treated. The parameters $\omega_r, \omega_u, \omega_i, \omega_h$ and ω_t are a form of control taking values between 0 and 1, that public authorities can set up to influence the infection capacities of the different infectious groups. For example, by properly equipping health personnel, which translates into a value of ω_h close to 1, we would reduce the probability of becoming infected in a hospitalized patient, by applying strict isolation, which corresponds to a value of ω_i close to 1, one reduces the probability of becoming infected in a sick patient, and by emphasizing advertisement on Covid-19 and rigor on the application of barrier measures such as wearing face masks, washing hands and cleaning surfaces, which corresponds to a value of ω_u close to 1, it would reduce the probability of getting infected in a non-carryover. It should also be noted that for a detected case, the free susceptible persons are placed in isolation, that is to say, goes to compartment S_i at a rate

$$w = \frac{\nu p E}{S_f + E + I_u}.$$

Following an infection, a susceptible person becomes infected, that is to say, that he has the virus but does not participate in transmission. The infected become infectious at a rate σ , among the infectious a proportion p will be detected thanks to the test and will therefore be I_r and another $1 - p$ will not be and will pass to I_u . Unreported infectious can become reported at a rate ϵ . The infectious reported carry out the compartment at a rate δ , a proportion q becomes hospitalized and another proportion $1 - q$ is isolated. All infectious can be treated at various rate and after the treatment compartment, one can become immune at a rate λ . This immunity can be lost with a γ rate. All individuals in the population can die naturally at a rate μ and those in the infectious compartments can also die due to illness at a rate d (equal to $\tilde{\mu} - \mu$).

We suppose that:

- * The population is homogeneously spread out,
- * Those susceptible to confinement or isolation do not become infected,
- * Infectious people in isolation do not have a severe form of the disease.

The parameters and variables of the model are summarized in the tables (1) and (2).

The compartment diagram 1 showing the propagation dynamics is as follows in Figure 1.

3. Mathematical Analysis of the Model

We assume that population is strictly subdivided in these compartments: $S_f, S_c, S_i, E, I_r, I_u, I_h, I_i, T$ or R . We also assume that, at each time, the population inside a territory is homogeneously distributed and that new births are free susceptible people. The evolution of the compartments

Table 1. Variable of model

| Variable | Description |
|----------|--|
| humans | |
| S_c | Number of confined susceptible humans in the population |
| S_f | Number of free susceptible humans in the population |
| S_i | Number of isolated susceptible humans in the population |
| E | Number of infected humans in the population |
| I_r | Number of reported infectious humans in the population |
| I_u | Number of unreported infectious humans in the population |
| I_h | Number of hospitalized infectious humans in the population |
| I_i | Number of isolated infectious humans in the population |
| T | Number of treated humans in the population |
| R | Number of (relative) immune humans in the population |

Table 2. Parameters of model

| Parameters | Interpretation | Value | Reference |
|--|--|---------------------------|----------------------|
| Γ | Recruitment rate | 100 | assumed |
| u | Transmission from confined susceptible to free susceptible | $[0, 1]$ | |
| v | Transmission from free susceptible to confined susceptible | $[0, 1]$ | |
| z | Transmission from isolated susceptible to free susceptible | $[0, 1]$ | |
| ν | Average number of people isolated after a reported case | variable | assumed |
| μ | Natural death rate | $\frac{1}{59 \times 365}$ | (WHO (2020)) |
| d | Diseases induced mortality rate | 6.8331×10^{-6} | (Tang et al. (2020)) |
| γ | Rate of lost of immunity | $\frac{1}{90}$ | assumed |
| θ | Rate at which isolated infected become treated | 0.11624 | (Tang et al. (2020)) |
| σ | Rate at with infected becomes infectious | $\frac{1}{7}$ | (Tang et al. (2020)) |
| π | Rate at which hospitalized infected become treated | 0.33029 | (Tang et al. (2020)) |
| ζ | Rate at which unreported infected become treated | 0.1914 | assumed |
| p | Proportion of infected that becomes reported infectious | $[0, 1]$ | |
| q | Proportion of reported infectious that becomes hospitalized | $[0, 1]$ | |
| λ | Recovery rate from treated individuals | 0.5 | assumed |
| ϵ | Rate at which unreported infected become reported | 0.001 | assumed |
| $\beta_r, \beta_u, \beta_i, \beta_h, \beta_t$ | Disease contact rate of a person in the corresponding compartments | 0.3531 | (Zio et al. (2020)) |
| $\omega_r, \omega_u, \omega_i, \omega_h, \omega_t$ | Control measure of a person in the corresponding compartments | $[0, 1]$ | |

mentioned above is modeled by the following system (3) of ordinary differential equations where ' denotes the derivation:

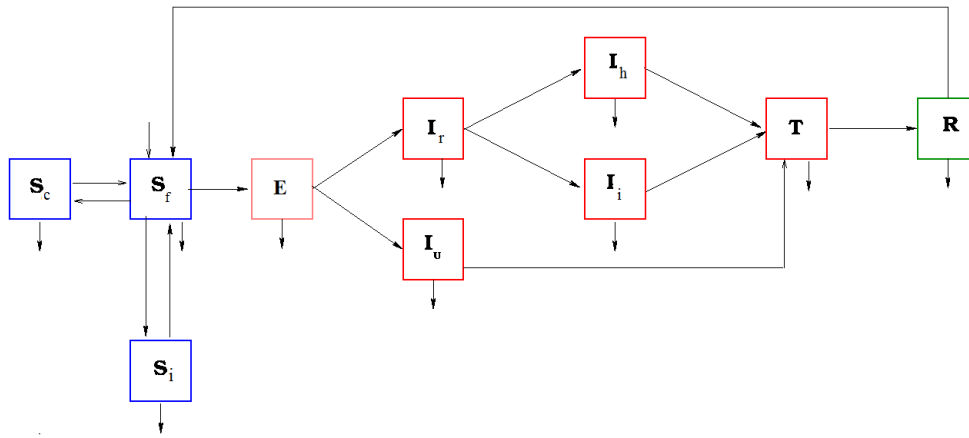


Figure 1. Compartment flow diagram

$$\begin{cases} S'_f = \Gamma + uS_c + zS_i + \gamma R - (v + w + \beta + \mu)S_f, \\ S'_c = vS_f - (u + \mu)S_c, \\ S'_i = wS_f - (z + \mu)S_i, \\ E' = \beta S_f - (\sigma + \mu)E, \\ I'_r = p\sigma E + \epsilon I_u - (\delta + \tilde{\mu})I_r, \\ I'_u = (1 - p)\sigma E - (\epsilon + \zeta + \tilde{\mu})I_u, \\ I'_h = q\delta I_r - (\pi + \tilde{\mu})I_h, \\ I'_i = (1 - q)\delta I_r - (\theta + \tilde{\mu})I_i, \\ T' = \pi I_h + \zeta I_u + \theta I_i - (\lambda + \tilde{\mu})T, \\ R' = \lambda T - (\gamma + \mu)R. \end{cases} \quad (1)$$

(supplemented with initial conditions at $t = 0$ in $(\mathbb{R}_+)^{10}$).

3.1. Positivity and well posedness of the model

The system (3) can be rewritten in matrix form as

$$\mathbf{x}' = \mathbf{A}(\mathbf{x})\mathbf{x} + \mathbf{b}, \quad (2)$$

where

$$\mathbf{A} = \begin{pmatrix} -(v + w + \beta + \mu) & u & z & \gamma & 0 & 0 & 0 & 0 & 0 & 0 \\ v & -(u + \mu) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ w & 0 & -(z + \mu) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \beta & 0 & 0 & -(\gamma + \mu) & 0 & 0 & 0 & 0 & 0 & \lambda \\ 0 & 0 & 0 & 0 & -(\sigma + \mu) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & p\sigma & -(\delta + \tilde{\mu}) & \epsilon & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & (1 - p)\sigma & 0 & -(\epsilon + \zeta + \tilde{\mu}) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & (1 - q)\delta & 0 & -(\theta + \tilde{\mu}) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & q\delta & 0 & 0 & -(\pi + \tilde{\mu}) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \zeta & \theta & 0 & -(\lambda + \tilde{\mu}) \end{pmatrix},$$

and

$$\mathbf{b} = (\Gamma, 0, 0, 0, 0, 0, 0, 0, 0, 0)^t.$$

Because \mathbf{A} is Metzler matrix, we have the following proposition.

Proposition 3.1.

The nonnegative cone \mathbb{R}_+^{10} is positively invariant for system (3).

Proof:

The proof of the positive invariance of \mathbb{R}_+^{10} under the system (3) relies on the application of the Proposition B.7 [p. 203, Smith and Waltman (1995)]. ■

Proposition 3.2.

The simplex

$$\Omega = \left\{ (S_f, S_c, S_i, R, E, I_r, I_u, I_i, I_h, T) \in \mathbb{R}_+^{10} / 0 \leq N \leq \frac{\Gamma}{\mu} + 1 \right\},$$

is a compact forward-invariant and absorbing set for system (3).

Proof:

We observed from the system that

$$\Gamma - \tilde{\mu}N \leq N' \leq \Gamma - \mu N.$$

The proof is easy and comes from this inequality,

$$\frac{\Gamma}{\mu + d} + \left(N(t_0) - \frac{\Gamma}{\mu + d} \right) e^{-(\mu+d)t} \leq N(t) \leq \frac{\Gamma}{\mu} + \left(N(t_0) - \frac{\Gamma}{\mu} \right) e^{-\mu t}. \quad \blacksquare$$

3.2. Disease free equilibrium (DFE)

Proposition 3.3.

The system (3) admits a trivial equilibrium named disease free equilibrium (DFE) given by:

$\mathbf{x}^* = (\mathbf{x}_S^*, \mathbf{x}_I^*)$, with $\mathbf{x}_I^* = 0 \in \mathbb{R}^6$ and $\mathbf{x}_S^* = (S_f^*, S_c^*, S_i^*, R^*)$ where,

$$S_f^* = \frac{\Gamma(u + \mu)}{\mu(u + v + \mu)}, \quad S_c^* = \frac{v\Gamma}{\mu(u + v + \mu)} \quad \text{and} \quad S_i^* = R^* = 0.$$

Proof:

It is obtained by straightforward computations using the fact that at DFE, one has:

$$\begin{cases} \Gamma + uS_c - (v + \mu)S_f = 0, \\ vS_f - (u + \mu)S_c = 0, \\ S_i = 0, \\ E = 0, \\ I_r = 0, \\ I_u = 0, \\ I_h = 0, \\ I_i = 0, \\ T = 0, \\ R = 0. \end{cases} \quad (3) \quad \blacksquare$$

3.3. Computation of basic reproduction number

Let $X = \sigma + \mu$, $Y = \delta + \tilde{\mu}$, $Z = \theta + \tilde{\mu}$, $W = \zeta + \epsilon + \tilde{\mu}$, $J = \pi + \tilde{\mu}$, $Q = \lambda + \tilde{\mu}$ and

$$\beta_r^* = (1 - \omega_r)\beta_r, \quad \beta_u^* = (1 - \omega_u)\beta_u, \quad \beta_i^* = (1 - \omega_i)\beta_i, \quad \beta_h^* = (1 - \omega_h)\beta_h, \quad \beta_t^* = (1 - \omega_t)\beta_t,$$

Proposition 3.4.

Following the Van de Driessche method (Driessche and Watmough (2002)), the basic reproduction number is

$$\mathcal{R}_0 = \mathcal{R}_0^h + \mathcal{R}_0^r + \mathcal{R}_0^u + \mathcal{R}_0^i + \mathcal{R}_0^t,$$

where

$$\mathcal{R}_0^u = \frac{\sigma\beta_u^*(1-p)}{WX},$$

$$\mathcal{R}_0^r = \frac{\sigma\beta_r^*(\epsilon(1-q) + Wp)}{WXY},$$

$$\mathcal{R}_0^h = \frac{\sigma\beta_h^*\delta q(\epsilon(1-q) + Wp)}{JWXY},$$

$$\mathcal{R}_0^i = \frac{\sigma\beta_i^*\delta(1-q)(\epsilon(1-q) + Wp)}{WXYZ},$$

$$\mathcal{R}_0^t = \frac{\sigma\beta_t^*[JYZ\zeta(1-p) + \delta((1-q)J\theta + \pi Zq)(\epsilon(1-q) + Wp)]}{JQWXYZ}.$$

Proof:

To compute the basic reproduction number R_0 , we use VDD method (Driessche and Watmough (2002)), which consists in determining the matrix F and V and determining the spectral radius of the matrix FV^{-1} . For this, we assemble the compartments traducing the infected individuals from the system (3) and decompose the right hand-side as $\mathcal{F} - \mathcal{V}$, where \mathcal{F} is the transmission part, expressing the production of new infected/infectious, and \mathcal{V} the transition part, which describes the change in state.

$$\mathcal{F} = \begin{bmatrix} \frac{(1 - \omega_r)\beta_r I_r + (1 - \omega_u)\beta_u I_u + (1 - \omega_i)\beta_i I_i + (1 - \omega_h)\beta_h I_h + (1 - \omega_t)\beta_t T}{N - S_c - S_i - \omega_r I_r - \omega_u I_u - \omega_i I_i - \omega_h I_h - \omega_t T} S_f \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix},$$

$$\mathcal{V} = \begin{bmatrix} (\sigma + \mu)E \\ -p\sigma E + \epsilon I_u + (\delta + \tilde{\mu})I_r \\ -(1 - p)\sigma E + (\epsilon + \zeta + \tilde{\mu})I_u \\ -(1 - q)\delta I_r + (\theta + \tilde{\mu})I_i \\ -q\delta I_r + (\pi + \tilde{\mu})I_h \\ -\pi H - \zeta I_u - \theta I_i + (\lambda + \tilde{\mu})T \end{bmatrix}.$$

Now we calculate the Jacobian of \mathcal{F} and \mathcal{V} at DFE \mathbf{x}^* ,

$$F = \frac{\partial \mathcal{F}}{\partial X} = \begin{bmatrix} 0 & (1 - \omega_r)\beta_r & (1 - \omega_u)\beta_u & (1 - \omega_i)\beta_i & (1 - \omega_h)\beta_h & (1 - \omega_t)\beta_t \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

$$V = \frac{\partial \mathcal{V}}{\partial X} = \begin{bmatrix} (\sigma + \mu) & 0 & 0 & 0 & 0 & 0 \\ -p\sigma & \delta + \tilde{\mu} & -\epsilon & 0 & 0 & 0 \\ -(1 - p)\sigma & 0 & (\epsilon + \zeta + \tilde{\mu}) & 0 & 0 & 0 \\ 0 & -(1 - q)\delta & 0 & (\theta + \tilde{\mu}) & 0 & 0 \\ 0 & -q\delta & 0 & 0 & \pi + \tilde{\mu} & 0 \\ 0 & 0 & -\zeta & -\theta & -\pi & \lambda + \tilde{\mu} \end{bmatrix}.$$

Then, $\mathcal{R}_0 = \rho(FV^{-1})$, where ρ is the spectral radius of the next-generation matrix (FV^{-1}) . ■

3.4. Local stability of the disease free equilibrium

ASSUMPTION A: Let us assume that:

$$1) \omega := \omega_r = \omega_u = \omega_i = \omega_h = \omega_t \text{ and } \beta := \beta_r = \beta_u = \beta_i = \beta_h = \beta_t,$$

$$2) \lim_{t \rightarrow +\infty} T(t) = 0 \text{ exponentially for } T(0) \text{ close to } 0,$$

$$3) \mathcal{R}_0^u < \frac{\tilde{\mu}(1-p)}{W},$$

$$4) \frac{\tilde{\mu}(1-p)}{W} < 1.$$

Proposition 3.5.

Under ASSUMPTION A, the disease free equilibrium (DFE) is locally asymptotically stable.

Proof:

We set $d = (\tilde{\mu} - \mu)$,

$$N(t) = S_f(t) + S_c(t) + S_i(t) + E(t) + I_r(t) + I_u(t) + I_h(t) + I_i(t) + T(t) + R(t),$$

$$P(t) := I_r(t) + I_u(t) + I_h(t) + I_i(t) + T(t),$$

at equilibrium

$$X^e \equiv (S_f^e, S_c^e, S_i^e, E^e, I_r^e, I_u^e, I_h^e, I_i^e, T^e, R^e),$$

with

$$P^e = I_r^e + I_u^e + I_h^e + I_i^e + T^e.$$

We obtain from (3):

$$N' = \Gamma - \mu N - dP. \tag{4}$$

Locally in a domain (e.g., a disk of an arbitrary radius $\varepsilon > 0$ centered at the origin DFE):

$$\begin{cases} P' = -\tilde{\mu}P - \lambda T + \sigma E, \\ E' = -(\sigma + \mu)E + \frac{\beta(1-\omega)P}{S_f - \omega P} S_f. \end{cases} \tag{5}$$

We (locally) replace (5) by $\lim_{t \rightarrow +\infty} T(t) = 0$ exponentially following ASSUMPTION A and obtain:

$$\begin{cases} P' = -\tilde{\mu}P + \sigma E, \\ E' = -(\sigma + \mu)E + \frac{\beta(1-\omega)P}{S_f^* - \omega P} S_f^*. \end{cases} \tag{6}$$

The Jacobian of

$$f^*(P, E) = \begin{bmatrix} -\tilde{\mu}P + \sigma E \\ -(\sigma + \mu)E + \frac{\beta(1-\omega)P}{S_f^* - \omega P} S_f^* \end{bmatrix},$$

at disease free equilibrium is

$$Jac_{(P^e=0, E=0)_{DFE}} = \begin{pmatrix} -\mu & -d & 0 \\ 0 & -\tilde{\mu} & \sigma \\ 0 & \beta(1-\omega) & -(\sigma + \mu) \end{pmatrix},$$

whose characteristic polynomial is

$$P(\lambda) = \lambda^2 + [\tilde{\mu} + \sigma + \mu]\lambda + \tilde{\mu}(\sigma + \mu) \left[1 - \frac{W}{\tilde{\mu}(1-p)} \mathcal{R}_0^u \right],$$

with a positive discriminant by straightforward computations. P admits two negative roots if

$$\frac{W}{\tilde{\mu}(1-p)} \mathcal{R}_0^u < 1,$$

(obviously, then, $\mathcal{R}_0^u < 1$). (4) implies that

$$\lim_{t \rightarrow +\infty} N(t) = \frac{\Gamma}{\mu},$$

or

$$\lim_{t \rightarrow +\infty} (S_f(t) + S_c(t)) = \frac{\Gamma}{\mu},$$

when

$$\mathcal{R}_0^u < \frac{\tilde{\mu}(1-p)}{W}.$$

If Assumption A is not true, then it is possible to have the result under relatively stronger conditions. ■

Remark 3.1.

The condition

$$\frac{W}{\tilde{\mu}(1-p)} \mathcal{R}_0^u - 1 < 0,$$

rewrites as

$$\mathcal{R}_0^u < \frac{\tilde{\mu}(1-p)}{W},$$

with

$$\frac{\tilde{\mu}(1-p)}{W} < 1.$$

That is obvious since the basic reproduction number is

$$\mathcal{R}_0 = \mathcal{R}_0^h + \mathcal{R}_0^r + \mathcal{R}_0^u + \mathcal{R}_0^i + \mathcal{R}_0^t,$$

and the distance between $\frac{\tilde{\mu}(1-p)}{W}$ and 1 is represented by $\mathcal{R}_0^h + \mathcal{R}_0^r + \mathcal{R}_0^i + \mathcal{R}_0^t$. The assumption

$$\frac{\tilde{\mu}(1-p)}{W} < 1,$$

reminds us a kind of comparison between J, W, Z and Q .

4. The Sensitivity Analysis

We have an explicit expression of the basic reproduction number \mathcal{R}_0 . The main goal of the public health planners must be to use all the means so that the value of the basic reproduction number becomes lower than 1. For this, it is therefore necessary to take decisions thus influencing precise values of the parameters in order to make the \mathcal{R}_0 less than 1. We have identified the following parameters $u, v, z, \nu, \beta_r, \beta_u, \beta_h, \beta_i, \beta_t$ and p as those on which we can influence through administrative decisions. For each parameter ρ , we calculate the values

$$\Upsilon_{\rho}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \rho} \times \frac{\rho}{\mathcal{R}_0},$$

presented by the following table:

| Parameter(ρ) | $\Upsilon_{\rho}^{\mathcal{R}_0^r}$ | $\Upsilon_{\rho}^{\mathcal{R}_0^u}$ | $\Upsilon_{\rho}^{\mathcal{R}_0^i}$ | $\Upsilon_{\rho}^{\mathcal{R}_0^h}$ | $\Upsilon_{\rho}^{\mathcal{R}_0^t}$ |
|---------------------|-------------------------------------|-------------------------------------|-------------------------------------|-------------------------------------|---|
| ω_r | $-\frac{\omega_r}{1-\omega_r}$ | 0 | 0 | 0 | 0 |
| ω_u | 0 | $-\frac{\omega_u}{1-\omega_u}$ | 0 | 0 | 0 |
| ω_i | 0 | 0 | $-\frac{\omega_i}{1-\omega_i}$ | 0 | 0 |
| ω_h | 0 | 0 | 0 | $-\frac{\omega_h}{1-\omega_h}$ | 0 |
| ω_t | 0 | 0 | 0 | 0 | $-\frac{\omega_t}{1-\omega_t}$ |
| p | $\frac{Wp}{\epsilon(1-q) + Wp}$ | $-\frac{p}{1-p}$ | $\frac{Wp}{\epsilon(1-q) + Wp}$ | $\frac{Wp}{\epsilon(1-q) + Wp}$ | $\frac{p[Y\zeta - W\delta\pi q\theta(1-q)]}{-\delta\epsilon\pi\theta q(1-q)(\epsilon(1-q) + Wp) - Y\zeta(1-p)}$ |

For a parameter ρ , we have

$$\Upsilon_{\rho}^{\mathcal{R}_0} = \frac{1}{\mathcal{R}_0} \left(\Upsilon_{\rho}^{\mathcal{R}_0^r} \mathcal{R}_0^r + \Upsilon_{\rho}^{\mathcal{R}_0^u} \mathcal{R}_0^u + \Upsilon_{\rho}^{\mathcal{R}_0^i} \mathcal{R}_0^i + \Upsilon_{\rho}^{\mathcal{R}_0^h} \mathcal{R}_0^h + \Upsilon_{\rho}^{\mathcal{R}_0^t} \mathcal{R}_0^t \right), \tag{7}$$

with

$$\begin{aligned} \Upsilon_{\omega_r}^{\mathcal{R}_0} &= -\frac{(\epsilon(1-q) + Wp)}{WXY\mathcal{R}_0} \omega_r \beta_r \sigma, & \Upsilon_{\omega_u}^{\mathcal{R}_0} &= -\frac{1-p}{WX\mathcal{R}_0} \beta_u \omega_u \sigma, \\ \Upsilon_{\omega_i}^{\mathcal{R}_0} &= -\frac{(\epsilon(1-q) + Wp)(1-q)}{WXYZ\mathcal{R}_0} \beta_i \omega_i \delta \sigma, & \Upsilon_{\omega_h}^{\mathcal{R}_0} &= -\frac{\epsilon(1-q) + Wp}{JWXY\mathcal{R}_0} \beta_h \omega_h q \delta \sigma, \\ \Upsilon_{\omega_t}^{\mathcal{R}_0} &= -\frac{\delta\pi q\theta(1-q)(Wp + \epsilon(1-q)) + Y\zeta(1-p)}{QWXY\mathcal{R}_0} \beta_t \omega_t \sigma. \end{aligned}$$

$\Upsilon_{\rho}^{\mathcal{R}_0}$ is computed as in (7). Straightforward computations lead to this proposition.

Proposition 4.1.

We can compare the sensitivity parameters $|\Upsilon^{\mathcal{R}_0}|$ as follow in particular cases:

- (1) $\omega_r \beta_r$, $\frac{1-q}{\theta + \tilde{\mu}} \delta \omega_i \beta_i$ and $\frac{q}{\pi + \tilde{\mu}} \delta \beta_h \omega_h$ (**or** ω_r , $\frac{1-q}{\theta + \tilde{\mu}} \delta \omega_i$ and $\frac{q}{\pi + \tilde{\mu}} \delta \omega_h$ if $\beta_r = \beta_i = \beta_h$) have the same order than the sensitivity parameters $\Upsilon_{\omega_r}^{\mathcal{R}_0}$, $\Upsilon_{\omega_i}^{\mathcal{R}_0}$ and $\Upsilon_{\omega_h}^{\mathcal{R}_0}$.
- (2) By setting $\kappa = \frac{\zeta \beta_t \omega_t}{Q \beta_u \omega_u}$ and $\chi = \frac{\pi q \theta \beta_t \omega_t}{\beta_i \omega_i}$ such that

$$|\Upsilon_{\omega_t}^{\mathcal{R}_0}| = \kappa |\Upsilon_{\omega_u}^{\mathcal{R}_0}| + \chi |\Upsilon_{\omega_i}^{\mathcal{R}_0}|,$$

we can see that:

- (a) if $\kappa \geq 1$ that is $\frac{\beta_t \omega_t}{\beta_u \omega_u} \geq \frac{Q}{\zeta}$, then $|\Upsilon_{\omega_t}^{\mathcal{R}_0}| \geq |\Upsilon_{\omega_u}^{\mathcal{R}_0}|$;
- (b) if $\chi \geq 1$ that is $\frac{\beta_t \omega_t}{\beta_i \omega_i} \geq \frac{1}{\pi q \theta}$, then $|\Upsilon_{\omega_t}^{\mathcal{R}_0}| \geq |\Upsilon_{\omega_i}^{\mathcal{R}_0}|$;
- (c) Generally, $\min \{|\Upsilon_{\omega_i}^{\mathcal{R}_0}|; |\Upsilon_{\omega_u}^{\mathcal{R}_0}|\} \leq \frac{|\Upsilon_{\omega_t}^{\mathcal{R}_0}|}{\kappa + \chi} \leq \max \{|\Upsilon_{\omega_i}^{\mathcal{R}_0}|; |\Upsilon_{\omega_u}^{\mathcal{R}_0}|\}$.

Proof:

The proof of (1) and (2) relies on straightforward computations using these simple properties.

For (1), consider two positive number a and b . $\frac{a}{b} < 1$ is equivalent to $a < b$.

For (2), assume that $u \geq 1$. (2)(a) and (2)(b) come from $c = u \times a + v \times b$ with $a, b, c, u, v \geq 0$, which implies that $c \geq u \times a \geq a$. Moreover, (2)(c) is a consequence of the property

$$(u + v) \times \min \{a, b\} \leq c \leq (u + v) \times \max \{a, b\}. \quad \blacksquare$$

Remark 4.1.

We observe that roughly speaking, the comparison of $\Upsilon_{\omega_r}^{\mathcal{R}_0}$, $\Upsilon_{\omega_u}^{\mathcal{R}_0}$, $\Upsilon_{\omega_t}^{\mathcal{R}_0}$ conditionally depends on the comparison of $\beta_r \omega_r$, $\beta_u \omega_u$, $\beta_t \omega_t$ (or ω_r , ω_u , ω_t if $\beta_r = \beta_u = \beta_t$).

5. Numerical simulations

In the sequel, all the variables represented in the Figures (1) to (9) are described in Table (1) with $I = I_r + I_u + I_h + I_i$. The scatterplot ("o") represents the data of the reported infectious cases in Cameroon the 25 first days after the 5th of March 2020, in the first phase (when the cases were essentially imported).

5.1. Strategy 1: $\omega_r = \omega_u = \omega_i = \omega_h = \omega_t = 0$ (no control)

In the Figures 2 and 3, we adjust the numbers I_r of reported cases to the scatterplot -o-o- of the Cameroon's data of the new reported cases (Roser et al. (2020); Covid-19 (Cameroon) (2020)).

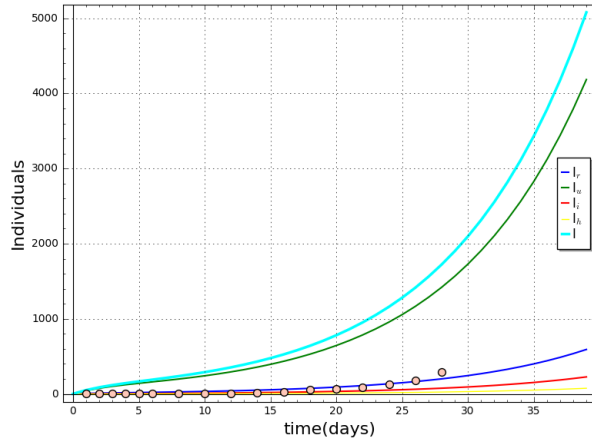


Figure 2. Evolution of the number of cases for $\mathcal{R}_0 = 2.8646$ with $u = v = z = \nu = 0$, $p = 0.1$, and $q = 0.4$: $0 \leq t < 40$ days

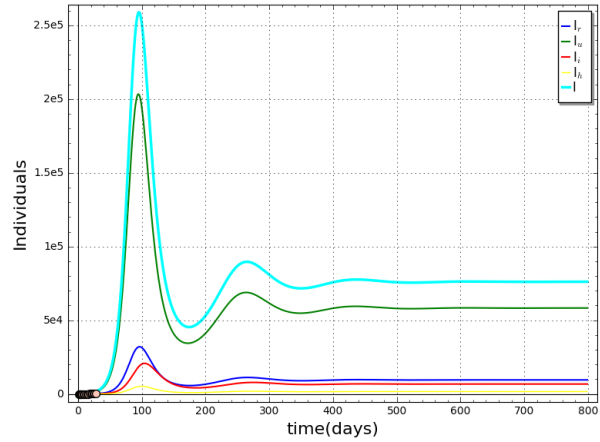


Figure 3. Evolution of the number of cases for $\mathcal{R}_0 = 2.8646$ with $u = v = z = \nu = 0$, $p = 0.1$, and $q = 0.4$: $0 \leq t < 300$ days

5.2. Strategy 2: $\omega_r, \omega_i, \omega_h, \omega_t, p$ close to 1 (control on intensive care, treated, reported and isolated infectious with massive test)

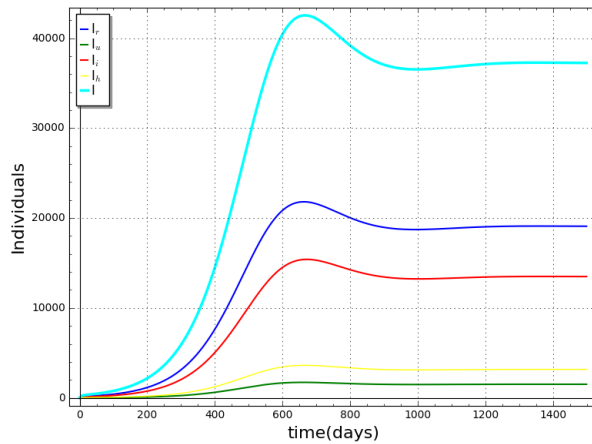


Figure 4. Evolution of the number of cases for $\mathcal{R}_0 = 1.1971$ with $u = v = z = \nu = 0$, $p = 0.9$, $q = 0.4$, $\omega_u = 0$ and $\omega_r = \omega_h = \omega_i = \omega_t = 0.8$

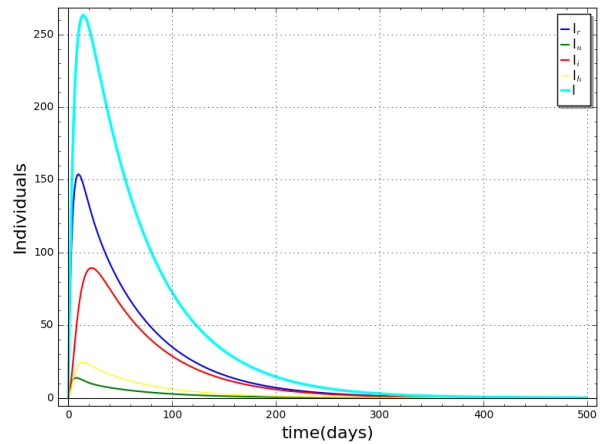


Figure 5. Evolution of the number of cases for $\mathcal{R}_0 = 0.75$ with $u = v = z = \nu = 0$, $p = 0.9$, $q = 0.4$, $\omega_u = 0$, $\omega_r = 0.89$, $\omega_h = 0.8$, $\omega_i = 0.9$ and $\omega_t = 0.9$

In multiple simulations similar to Figures 4 and 5, we see that more is the intensive control, less is the magnitude of the the maximum of the total number of infectious, with a postponed date of the first peak.

5.3. Strategy 3: $\omega_r, \omega_i, \omega_h, \omega_t$ close to 1 with p close to 0 (control on intensive care, treated, reported and isolated infectious with a low percentage of tests)

It concerns Figures 6 and 7 with an efficient containment for relatives of infected individuals.

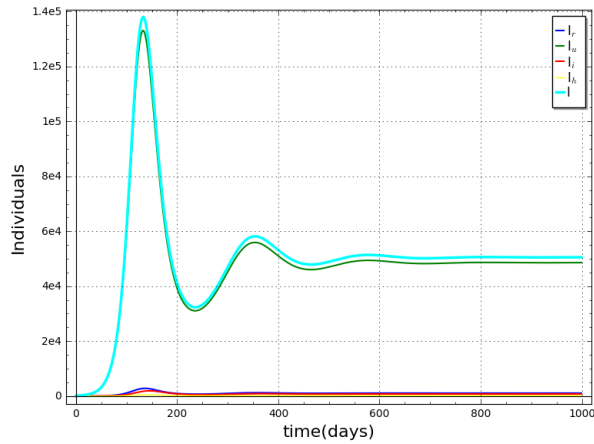


Figure 6. Evolution of the number of cases for $w = 0$ (no containment for relatives of infected individuals): $\mathcal{R}_0 = 1.9795$

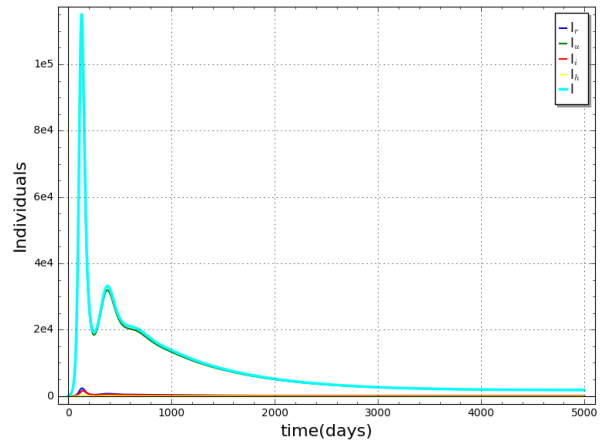


Figure 7. Evolution of the number of cases for $w > 0$ (efficient containment for relatives of infected individuals): $\mathcal{R}_0 = 1.9795$

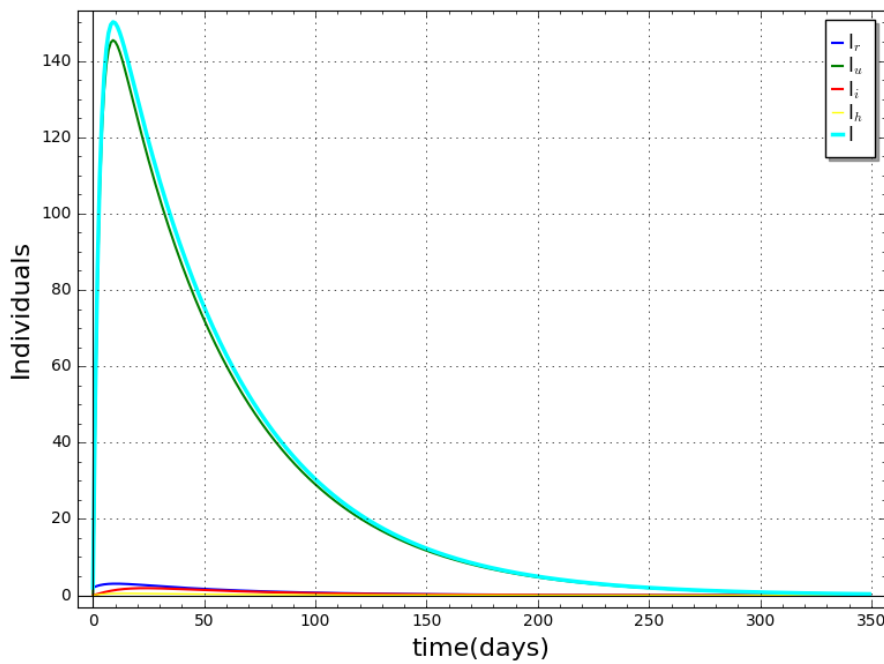


Figure 8. Evolution of the number of cases for $\mathcal{R}_0 = 0.7789$ (massive advertisement on healthy behaviour against Covid-19)

5.4. Strategy 4: $\omega_r, \omega_u, \omega_i, \omega_h, \omega_t$ close to 1 (efficient sensitization on barrier actions against Covid-19)

It concerns Figure 8 with massive advertisement on healthy behaviour against Covid-19.

5.5. Strategy 5: u, v variable in time (a two strategies approach with high multi-peaks)

All the countries adopted at least two different strategies: one at the beginning and another just after the first of June 2020 when they tried to get out of the containment. $u(t) = \begin{cases} 0.1 & \text{if } t < 150, \\ 0.4 & \text{if } t \geq 150, \end{cases}$ and $v(t) = \begin{cases} 0.5 & \text{if } t < 150, \\ 0.1 & \text{if } t \geq 150, \end{cases}$ with time t in days. We present in Figures 9, 10, 11 and 12 a scheme where the susceptible individuals are more contained with a few relaxation ($u(t) < v(t)$) for $t < 150$ days, thereafter, the susceptible individuals are less contained with a more relaxation (JdC (2020); Cameroon’s Prime Ministry (2020)) in the exposition to the disease ($u(t) > v(t)$) for $t \geq 150$ days. In this scheme, the second peak is much more severe than the first.

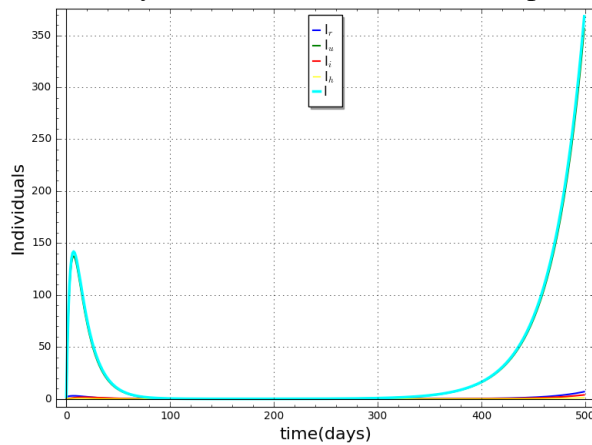


Figure 9. Evolution of the number of cases for $\omega_r = 0.45, \omega_u = \omega_i = \omega_h = \omega_t = 0.3, \mathcal{R}_0 = 1.8175; 0 \leq t \leq 500$ days

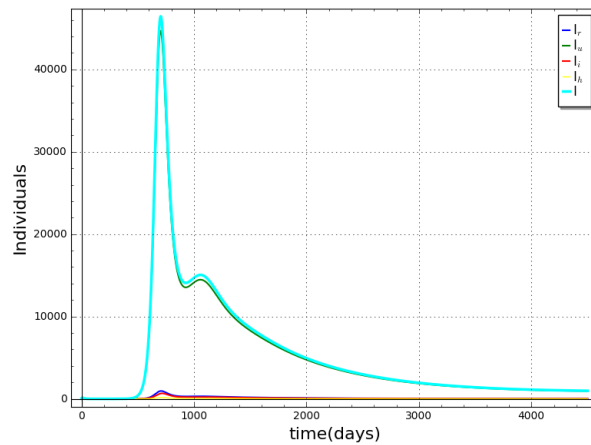


Figure 10. Evolution of the number of cases for $\omega_r = 0.45, \omega_u = \omega_i = \omega_h = \omega_t = 0.3, \mathcal{R}_0 = 1.8175; 0 \leq t \leq 4000$ days

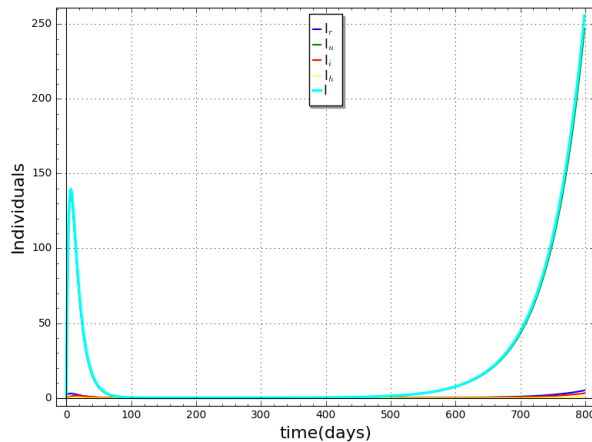


Figure 11. Evolution of the number of cases for $\omega_r = 0.45, \omega_u = \omega_i = \omega_h = \omega_t = 0.4, \mathcal{R}_0 = 1.5562; 0 \leq t \leq 800$ days

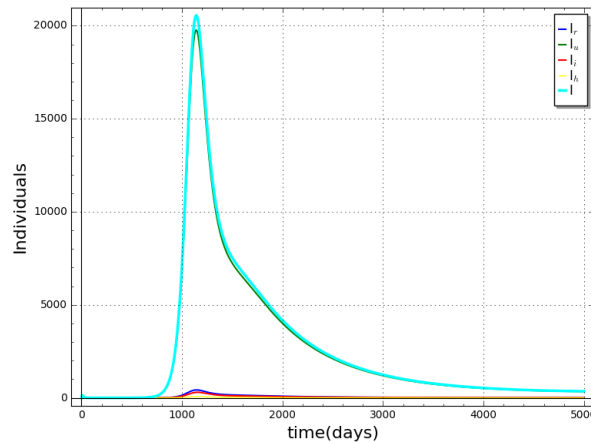


Figure 12. Evolution of the number of cases for $\omega_r = 0.45, \omega_u = \omega_i = \omega_h = \omega_t = 0.4, \mathcal{R}_0 = 1.5562; 0 \leq t \leq 5000$ days

The Figures 13 and 14 prove that strong policies (with high ω .) lead to good results even if the population adopt a wrong relaxation strategy.

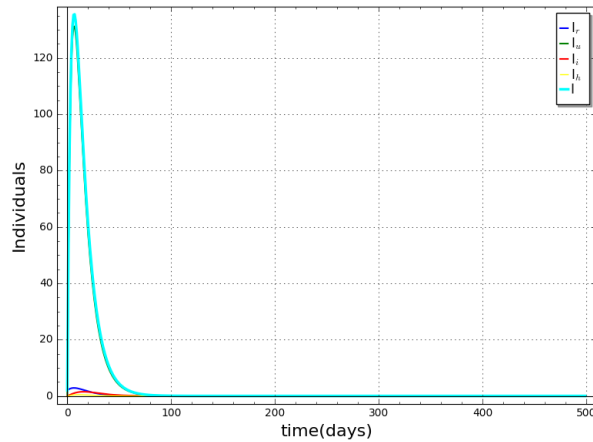


Figure 13. Evolution of the number of cases for $\omega_r = \omega_u = \omega_i = \omega_h = \omega_t = 0.6$, $\mathcal{R}_0 = 1.0386$; $0 \leq t \leq 500$

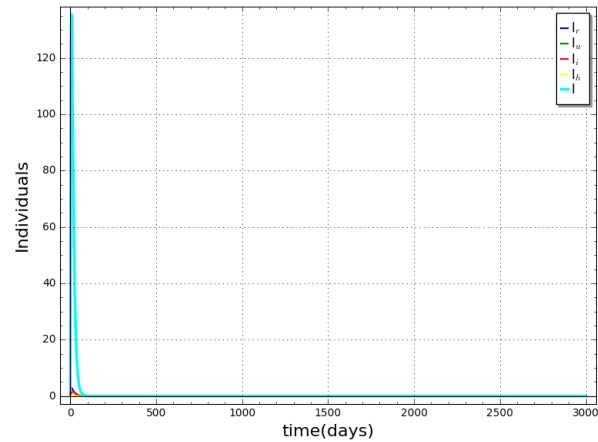


Figure 14. Evolution of the number of cases for $\omega_r = \omega_u = \omega_i = \omega_h = \omega_t = 0.6$, $\mathcal{R}_0 = 1.0386$; $0 \leq t \leq 3000$

In multiple simulations similar to 9 and 12, we observe that a bang-bang strategy in the containment process could make the epidemic restart. This Figure illustrates the fact that the end of the first peak doesn't signify the end of the epidemic if the relaxation in barrier actions against COVID are abandoned with no caution. Then, a good strategy followed by careless management of the disease, could lead to a severe peak of the disease.

6. Discussion

Like our model, Nkeck and Ebangue (2020) found also with SIR models almost the same period for the first peak: between the thirteen of June and the first of July 2020 for Cameroon. Our results show then several possible peaks of the Covid-19 epidemic starting at the end of June 2020 in Cameroon. These results are very important compared to other works cited therein. We numerically observe the impact, on Covid-19, of the asymptomatic or unreported infected/infectious of Covid-19 through initial conditions. In Figure 1 and others therein, several peaks appear (even three) that are similar to results reported by Adam (2020): “a second wave of the pandemic might be expected later in the year.” See also Pedro et al. (2020). Moreover, it could be possible to study the Hopf bifurcation whenever $R_0 > 1$ for a well chosen set of parameters – that would be the worse situation with an infinite number of regular peaks. We observe also that anti-Covid-19 actions numerically postponed the date of the first peak of the disease (from June 2020 - Figures 2 and 3 - to other dates in 2020 - Figures 8 and 14). But, this translation is followed with a reduction of the magnitude of the first peak of the disease. It is possible to study more deeply the several peaks case by using the almost periodic theory (Xu et al. (2019); Xu et al. (2020)).

7. Conclusion

In this work, we proposed a model of Covid-19 integrating almost all aspects in the Cameroonian context. We determined the basic reproduction number, studied the local stability of the disease-

free equilibrium, and performed a sensitivity study. Subsequently, using numerical simulations, we were interested in what could happen after the first peak of Covid-19 if the barrier measures stopped being respected. We obtain that, the real danger after the first peak is the relaxation of the barrier measures that could restart the epidemic. Finally, we can say that, the key words are “Self-discipline, effective measures, and testing,” even after the first peak of the disease. They can be several successive peaks of the disease with high magnitudes if barrier measures are neglected.

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