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A New Vision On the Modelling of Emerging Infectious Diseases

Bruce Masonova SOLOZAFY BEMENA[1]
André TOTOHASINA[2]
Daniel Rajaonasy FENO[3]

[1]School Mixed Sambava B.P.90 – Sambava, Madagascar
[2]University of Antsiranana B.P.0 – Antsiranana, Madagascar
[3]University of Toamasina B.P.591 – Toamasina, Madagascar
Email: brucebemena@gmail.com

Abstract

Faced with the evolution of tools and method of mathematical modeling in epidemiology that we have for the treatment and prevention of the phenomenon of propagation of infectious diseases today, global warming plays a very important role in the genetic modification of microorganisms. This leads to the unsuitability of existing models such as the Susceptible, Exposed, Infected, Retired (SEIR) model proposed in the literature. The present paper proposes a Susceptible, Exposed, Precontaged, Infected, Retired (SEPIR) model adapted to the mechanism of diffusion of the new forms of the infectious diseases studied. This SEPIR model is specialized in cases where the infection spreads directly: first between precontagious individuals (asymptomatic) and susceptible individuals, second between infectious individuals (symptomatic) and susceptible individuals.

Keywords: *Susceptible, Exposed, Precontaged, Infected, Retired (SEPIR) model*

Introduction

Since epidemiology originated from the idea expressed by Hippocrates more than 2000 years ago (R.Bonita and al. (2010)), the losses caused by epidemic series are considerable and disastrous in all populations of the world. Since the creation of the WHO in 1948, the consequences of epidemics in the population seem to be controlled and mitigated because of different methods and strategies such as vaccination programmes to immunise the population against infectious diseases, early detection, training and development of sophisticated materials, creation of different antibiotic doses to fight against epidemic invasions, etc. Despite all these efforts, epidemics still continue to emerge (emerging infectious disease) and re-emerge (re-emerging infectious disease). To enrich the literature on epidemiological studies, the SEIR model was proposed by M.Y.Li and L.C.Wang (1995) to study infectious diseases that behave in such a way that, after the incubation period, the infected person becomes infectious and then, once cured, acquires

total immunity to the disease. This model is then improved by A.Korobeinikov and P.K.Maini (2004) to study stability.

Global warming plays a very important role in the reappearance of epidemic series and the appearance of new emerging infectious diseases (GIEC (2014)), the modification and genetic change of micro-organisms becoming very aggressive, stronger (adapted), very difficult to treat and tending to spread in a very rapid manner (B.Marçais et al. (2000)). This leads to the inadequacy of existing models for predicting and controlling the spread of infectious diseases (S.Morand and Lajaunie (2015)) comsuch as the SEIR model mentioned above. To regain the advantage, everyone is mobilising, as the climatologists (C.Pacteau and Joussaume (2015)) (column 1, paragraph 1, page 14) did research and left the perspective "build models integrating biodiversity modifications with climate variables" and also (OMS (2004))(column 2, paragraph 2, page 13) did a conference and left the perspective in its summary "Establish predictive scenario models". For today, there is already a new infectious disease behaviour that is spreading with very rapid speed and it is being transmitted even between individuals who are not yet showing symptoms (INSPQ (2021)), (Y.WELKER (2020)). This is what (OMS (2019)) and (Z.Hu and al. (2020)) have called a new strain of coronavirus or COVID-19.

In this paper, we propose a model adapted to the mechanism of diffusion of new behaviours of the studied infectious diseases. The rest of the paper is organised as follows. Section 2 presents the dynamic process of disease infection. Section 3 deals with the mechanism of disease spread. We present the different hypotheses in Section 4. Our model of spread is presented in section 5. We end with a short conclusion in section 6.

New dynamic process of infection of emerging and re-emerging infectious diseases

We accept that an individual is affected by an infectious disease when he or she comes into contact with a pathogen, which may be of various kinds (an infected individual, a mosquito, a well, etc.). We note that the modification and genetic change of pathogen micro-organisms caused by global warming will lead to the advancement or acceleration of the contagiousness period which we call "**early or premature contagion or precontagion**" (see figure (1)). This means that the infectious disease is spread not only by the sick individual (who shows symptoms) but also by the healthy carrier individual. This will cause the epidemic to spread very rapidly. We consider here that the change in the transfer of infection brought about by **the new behaviour of the pathogens** does not change **the total duration of the contraction of the disease** on the individual. But it does increase the time of the contagious period and decrease the latent period.

The character **precontagious** of an individual is acquired only after a period of **latency** after infection. And the **infected** individual also remains **contagious** for some time : he is then either ready to **contract** the disease again, or **resistant** to a new infection, or dead**.**

Mechanism of disease progression

The mechanism of evolution of an epidemic is presented in the following stages :

– Global warming has increased the temperature of the earth's surface.

- The increase in temperature has impacted the environment of living beings, including microorganisms.
- In the micro-organisms, those that are not killed by the increase in temperature, have managed to adapt, to mutate and they have sought the new favorable environment to live in (in the human organism).
- When micro-organisms arrive in human organisms, the mutants are able to adapt and multiply very quickly.
- After the latent phase, without having yet to cause the prodrome in the host organism, they can already contaminate other organisms from saliva, sexual intercourse, sneezing, blood, a few ordinary coughs, etc. i.e. a healthy carrier can contaminate the population if he is already infected.
- The infected individual remains contagious until the onset of symptoms of the disease and has continued to be contagious until some time after recovery or death.

In order to provide our solution to the study of the modelling of this phenomenon, we make the following definitions of assumptions that complement the definition of the susceptible, exposed, infected compartments and the latency period.

Definitions of the study's assumptions

Definition .1. *An individual who has been infected with the disease pathogen and is capable of transmitting it, but has no symptoms, is called* ***a precontaminated or precontagious individual****.*

Definition .2. ***Précontaged*** *individuals are assigned to this compartment with the rate of precontagion called the precontagion rate. Precontagious individuals are assigned to this compartment with the rate k called* ***precontagiousness rate****.*

The letter P will be used to refer to individuals who are infected and contagious, but do not yet show symptoms of disease.

Definition .3. *The* ***period of precontagiousness*** *is the time during which an infected person has no symptoms but can transmit the disease to another.*

Definition .4. *The* ***infected*** *compartment represents those who are not only already infected and have shown symptoms of the disease, but also capable of transmitting the disease back into the population.*

Definition .5. *The* ***period of contagiousness*** *is a distinct phase of time when the sick individual (person who has the symptoms of the disease and whose health is impaired) transmits a disease to the other individual.*

The figure (1) represents schematically the different phases of the disease.

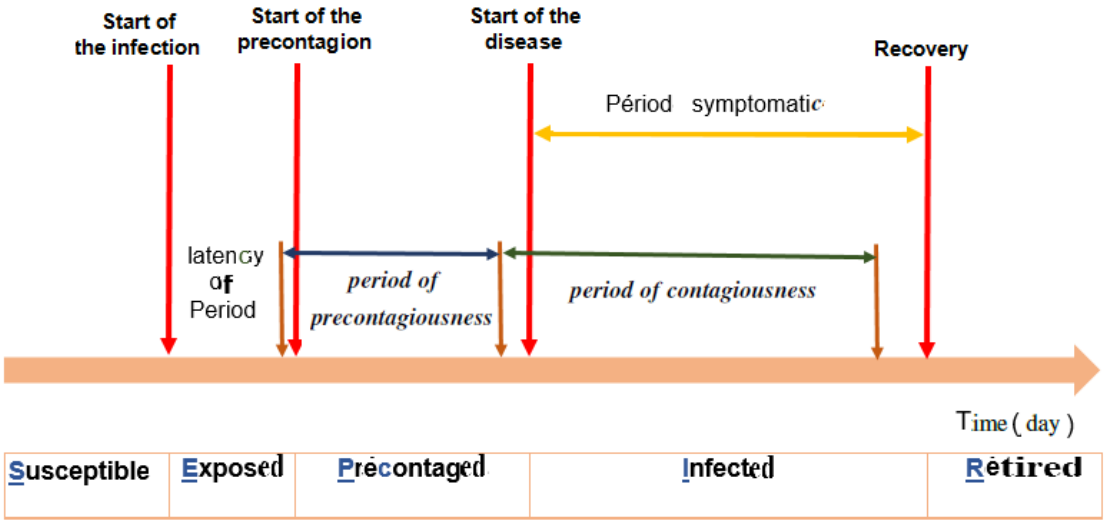

FIG. 1: Representation of the contagion process «SEPIR»

Proposed model of epidemic spread

In this section, we focus on modelling the new phenomenon of the spread of infectious diseases caused by global warming in a population. On the one hand, we highlight the new dynamic process of the infection of emerging infectious diseases, on the other hand, we present the different hypotheses of the infectious disease behaviours. The present study consists in proposing a propagation model able to understand the different behaviours of the infectious disease and the new mechanism of the rapidity of the infection diffusion. The model assumes that the population is constant and homogeneous (no age structure, no spatial or social structure).

Our objective is to develop a new compartmental model by integrating the above assumption of new dynamic infection processes into the SEIR compartmental model in the literature. Indeed, this integration effectively contributes to the modelling and simulation of any form of emerging diseases caused by global warming. In this work, epidemic modelling only considers cases where infection spreads directly : first, between precontagious (precontaminated) and susceptible individuals ; second, between infectious (infected) and susceptible individuals.

1. **Definition of the assumptions of the dynamic model of SEPIR**

In the SEPIR type (Susceptible, Exposed, Precontaged, Infected, Recovered or Retired), we consider that initially there is no cure, and the epidemic spreads very rapidly. After the infectious stage, the cured individual becomes immune to the disease. This type of modelling is suitable for such short interval periods. Natural mortality and emigration are balanced by birth and immigration.

In order to design our SEPIR model, we make the following assumptions.

- **A1** : The population size is equal to N (assumed fixed).
- **A2** : The time variable t is of discrete type, such that $t \in T$ or T is the total duration of the epidemic.
- **A3** : The time period $\Delta t = dt$ represents hours or days or weeks.
- **A4** :At each time t, the population N is subdivided into four compartments : S(t) : set of susceptible individuals, E(t) : set of exposed individuals, P(t) : set of precontaged individuals, I(t) : set of infected individuals and R(t) : set of Recovered or Retired individuals with $N = S(t)+E(t)+P(t)+I(t)+R(t)$ and $S(0) = S_0 > 0$, $P(0) = P_0 > 0$ and/or $I(0) = I_0 > 0$.
- **A5** : We assume that every susceptible individual over a period of time is exposed, precontaged and then infected, and those who have already had the disease and are now immune to it. We include in the immune group those who have died because they do not infect other individuals.
- **A6** :The transmission of the infection is done through a direct contact between : firstly of the susceptible S and one or more precontaged P with a factor of proportionality β_p (also called rate of precontagion or rate of transmission or rate of transmission of the susceptible to the exposed), secondly of the susceptible S and one or more infected I with a factor of proportionality β_i (also called rate of infection). We admit that a factor β is the total rate of transmission or of exposure, thus $\beta = \beta_p + \beta_i$.

2. Schematic of the SEPIR model

We assume that an infected individual and a precontaminated individual encounter on average $\beta(S/N)$ individuals likely to be exposed per unit of time, with $\beta = \beta_p + \beta_i$.

Let us note : $\beta > 0$: the rate of exposure (or of transmission from the susceptible to the exposed), $k > 0$: the rate of precontagiousness (or of transmission from the exposed to the precontaged), $\nu > 0$: the rate of contagiousness (or of transmission from the precontaged to the infected), $\gamma > 0$: the rate of recovery or of retired or of immunity (or of transmission from the infected to the retired). The SEPIR model is then illustrated in Figure (2) :

FIG. 2: Sepir model diagram

To these five different states, we can associate five eigenevolution equations (see formula (1)).

3. Representation in the form of the differential equations of the SEPIR model

According to hypothesis (A6) in section (5.1) above, we consider that during the time interval *dt*, the Susceptible compartment loses the number $S(\beta_p P + \beta_i I)$ of individuals exposed by disease. According to the hypothesis (A6), we consider the new cases reached by the infection during the time interval *dt* which are equal to $\beta_i SI$. And the new cases reached by the precontagion during the time interval *dt* which are equal to $\beta_p SP$. One obtains the new cases exposed to the disease during the time interval *dt* which will be equal to $\beta_p SP + \beta_i SI = S(\beta_p P + \beta_i I)$. According to hypothesis (A5), we consider that during the time interval *dt* the compartment precontaged by the disease has increased in number kE individuals and at the same time, it loses the number νP of sick or infected individuals. According to hypothesis (A5), we consider that during the time interval *dt* the compartment Infected has increased in the number νP of precontaged individuals, and it loses the number γI of cured individuals. According to hypothesis (A4), (A5), we consider that during the time interval *dt* the retired compartment has increased the number γI of infected individuals. And we obtain the SEPIR model as a system of differential equations below :

$$\begin{cases} \frac{dS(t)}{dt} = -S(t)\left(\beta_p P(t) + \beta_i I(t)\right) \ ; \\ \frac{dE(t)}{dt} = S(t)\left(\beta_p P(t) + \beta_i I(t)\right) - kE(t) \ ; \\ \frac{dP(t)}{dt} = kE(t) - \nu P(t) \ ; \\ \frac{dI(t)}{dt} = \nu P(t) - \gamma I(t) \ ; \\ \frac{dR(t)}{dt} = \gamma I(t). \end{cases} \quad (1)$$

There is a unique solution for this system (1), under the initial conditions :

$S(0) = S_0$, $E(0) = E_0$, $P(0) = P_0$, $I(0) = I_0$, $R(0) = R_0$ in particular, in the region, $\Omega = \{(S, E, P, I, R), S > 0, P > 0, I > 0, R > 0\}$ which is positively invariant for the system.

We admit that $\frac{dS(t)}{dt} + \frac{dE(t)}{dt} + \frac{dP(t)}{dt} + \frac{dI(t)}{dt} + \frac{dR(t)}{dt} = 0$, we deduce from this that for turn $t \geq 0$, $S(t) + E(t) + P(t) + I(t) + R(t) = S(0) + E(0) + P(0) + I(0) + R(0) = N$ avec $N > 0$.

Let $X = (S, E, P, I, R)$, we can rewrite this differential system (1) as $X' = F(X)$ with F of class C^∞.
By studying the system (1),we obtain the following theorem :

Theorem .1. *Let N, β_p, β_i, k, ν, γ, g $\in R$, in the absence of the infection or I=0 ; then the dynamic model is such that : SEPI=SEPIS=SIPIR.*

Proof :
It is obvious that if we put I=0 in these three models, they become identical.

4. Simulation of the SEPIR model

The different curves obtained, with Scilab, give us an idea of the evolution of the epidemic. For the simulation, we consider here to have a precontaged individual at time t = 0 with N=10000, β_p = 0.2, β_i = 0.1, k=0.4, ν = 0.2 et γ = 0.5. We consider a period *t* depending on the unit of transmission rates. It is equivalent to a day or week or month. By doing the simulation, we obtained the following curves :

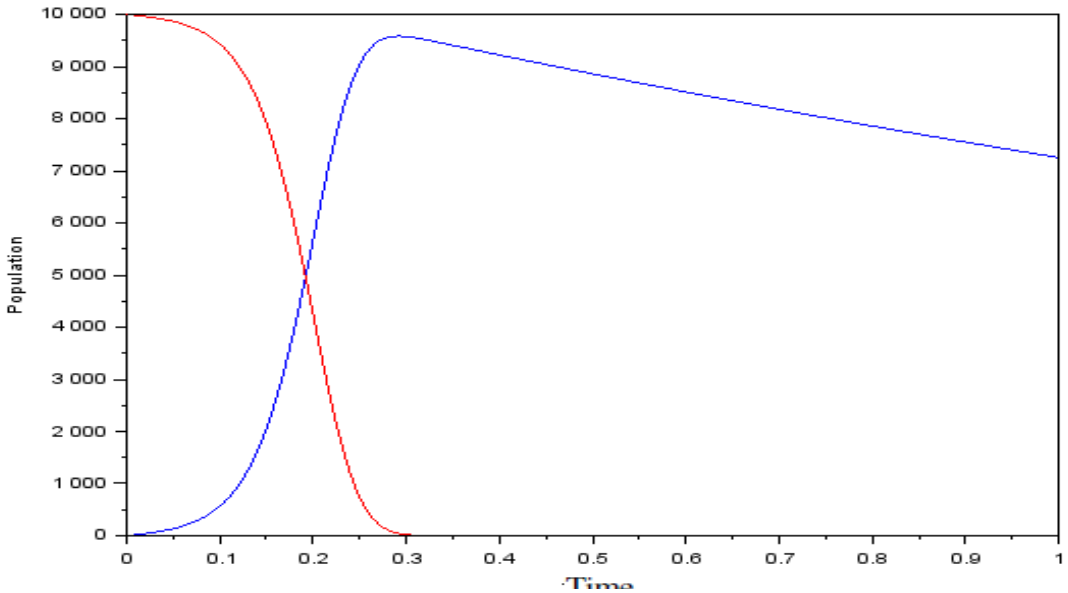

FIG. 3: Curves of S(t) coloured in red and E(t) in blue

Interpretation :

In figure (3), we have shown that even with low precontagion and infection rates, the epidemic is spreading with a phenomenal and very rapid speed. All susceptible individuals are already exposed after only 0,3 of a time period *t*.

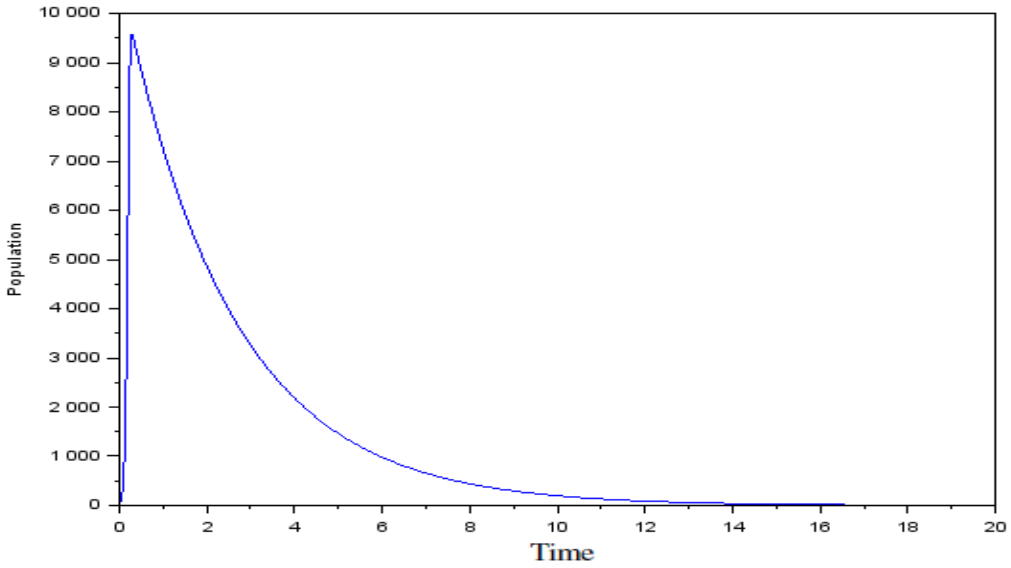

FIG. 4: Curve of E(t) during a phase of the epidemic

Interpretation :

From figure (4), after the phenomenal evolution of the epidemic, the curve of the Exposed stablilises and fades after the 16^{e} period *t* of the epidemic.

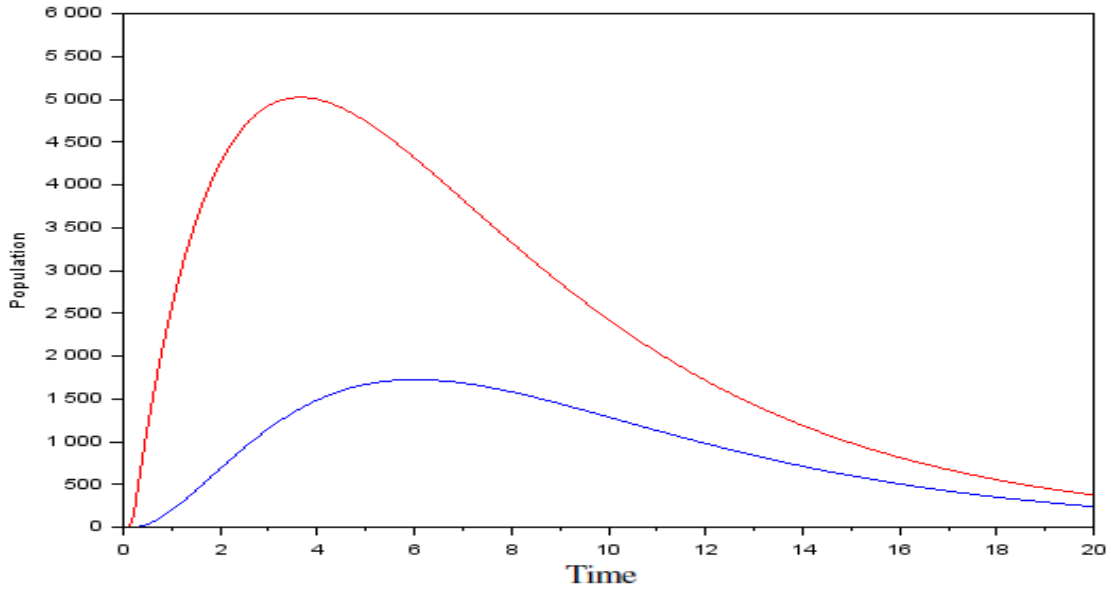

FIG. 5: Curves oP(t) coloured in red and I(t) in blue

Interpretation :

From Figure (5), it appears that after the sharp increase, the curves for the Precontaged and Infected stabilise and become a low endemic after the 20^{e} period of time

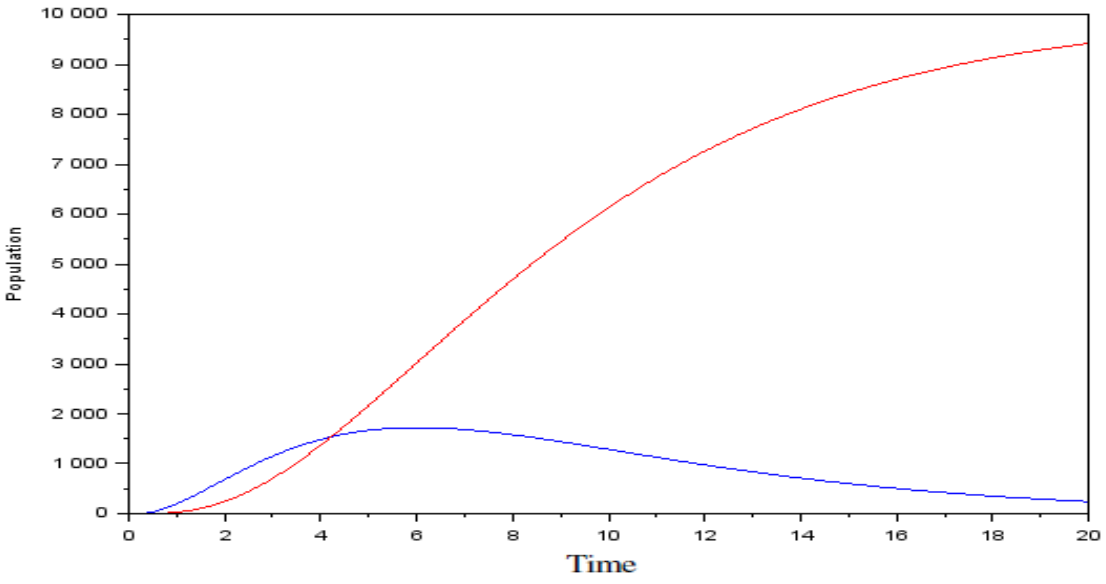

FIG. 6: Curves of I(t) coloured in blue and R(t) in red

Interpretation :

From figure (6), it appears that after the 20^{e} period of time *t*, the curve of R(t) stabilizes and almost 9500 individuals are recovered and immune from this disease.

5. Study of the equilibrium point of the SEPIR model

According to the Lyapunov stability theory in (J.M.M.ONDO (2012)), the equilibrium point is defined as follows.

Definition .6. *Consider U, a non-empty open of R^n containing 0, and I a non-empty interval of* R, *not bounded on the right. Let be systems of the form :*

$$\dot{x} = f(x) \tag{2}$$

$$\dot{x} = f(t, x) \tag{3}$$

where the functions f : U→ R^n *for the system (2) and* f : I × U→ R^n *for the system (3) are assumed to be continuous.*
A point a is a point of equilibrium or state of equilibrium or singular point of the system (2) (resp. (3)), if f(a) = 0 (resp. if, for any t∈I, f(t,a) = 0).

We then obtain the following proposition :

Proposition 1. *Let N>0. Then the system (1) with conditions S(0) = S_0, E(0) = E_0, P (0) = P_0, I(0) = I_0, R(0) = R_0 and S(t)+E(t)+P(t)+I(t)+R(t) = S(0)+E(0)+P(0)+I(0)+R(0) = N admits a unique solution (S,E,P,I,R) defined on [0, +∞[.*

Proof : Indeed,

Let's calculate the equilibrium points in the absence of infection and/or precontagion ignoring the death compartment.

The equilibrium point of the model (1) satisfies the system (4) below :

$$\begin{cases} -\beta_p SP - \beta_i SI = 0 \ ; \\ \beta_p SP + \beta_i SI - \ kE = 0 \ \ ; \\ kE - \ \nu P \ = 0 \ \ ; \\ \nu P \ - \ \gamma I = 0. \end{cases} \tag{4}$$

In the absence of the infection I=0 and the precontagion P=0, we obtain the following proposition (2) :

Proposition 2. *Let N>0, in the absence of the infection I=0 and the precontagion P=0, then, the system* (1) *admits the equilibrium point :* $E_0 = (N, 0, 0, 0, 0)^T$.

Proof :

By replacing I=0 and P=0 in the first, second and third equations in the system (1), with *S+E+P +I+R = N*. We obtain the first equilibrium point : $E_0 = (\hat{S},\ \hat{E},\ \hat{P},\ \hat{I},\ \hat{R})$:

$$E_0 = (N, 0, 0, 0, 0)^T. \tag{5}$$

In the absence of the infection I=0 and in the presence of the precontagion P≠0, we obtain the following proposition (3) :

Proposition 3. *Let N>0, in the presence of the precontagion P≠0 and in the absence of the infection I = 0 ; then :*

1. *The system (1) admits the equilibrium point :* $E_p^* = \left(\frac{\nu}{\beta_p},\ \frac{\nu(\beta_p N-\nu)}{\beta_p(k+\nu)},\ \frac{k(\beta_p N-\nu)}{\beta_p(k+\nu)},\ 0,\ 0\right)^T$;
2. Moreover, for *t*>0, we have $\beta_p N > \nu$.

Proof :

Replacing $P\neq 0$ and $I = 0$, if $\nu\neq 0$, the system (4) becomes :

$$\begin{cases} \beta_p SP - kE = 0 \ ; \\ kE - \nu P = 0 \ ; \\ \nu P \neq 0. \end{cases} \tag{6}$$

The second equation of (6) implies : $E^* = \frac{\nu P}{k}$ and by carrying this result in the first one, we obtain :$S^* = \frac{\nu}{\beta_p}$.

As I=0, we have : $N = S + E + P.$ (7)

Replacing S^* and E^* in (7), we get : $P^* = \frac{k(\beta_p N-\nu)}{\beta_p(k+\nu)}$.

Replacing P^*, we then have the equilibrium point : $E_p^* = (S^*, E^*, P^*, 0, 0)$ as follows :

$$E_p^* = \left(\frac{\nu}{\beta_p},\ \frac{\nu(\beta_p N-\nu)}{\beta_p(k+\nu)},\ \frac{k(\beta_p N-\nu)}{\beta_p(k+\nu)},\ 0,\ 0\right)^T \tag{8}$$

Note that if $\hat{S} > S^*$ implies : $N > \frac{\nu}{\beta_p}$. (9)

From (9), we deduce that : $P^* = \frac{k(\beta_p N-\nu)}{\beta_p(k+\nu)} > 0.$ (10)

And $E^* = \frac{\nu(\beta_p N-\nu)}{\beta_p(k+\nu)} > 0.$ (11)

From (9), (10) and (11), we have $\beta_p N > \nu$.

In the presence of the precontagion $P\neq 0$ and the infection $I\neq 0$, we obtain the following proposition (4) :

Proposition 4. *Let N>0, in the presence of the infection* $I\neq 0$ *and the precontagion* $P\neq 0$ *; then :*

1. *The system (1) admits the endemic equilibrium point :*

$E_p^{\bullet}$

$$= \left(\frac{\nu\gamma}{\beta_p\gamma + \beta_i\nu},\ \frac{\nu\gamma(N(\beta_p\gamma + \beta_i\nu) - \nu\gamma)}{(\nu\gamma + k\gamma + \nu k)(\beta_p\gamma + \beta_i\nu)},\ \frac{k\gamma(N(\beta_p\gamma + \beta_i\nu) - \nu\gamma)}{(\nu\gamma + k\gamma + \nu k)(\beta_p\gamma + \beta_i\nu)},\ \frac{k\nu(N(\beta_p\gamma + \beta_i\nu) - \nu\gamma)}{(\nu\gamma + k\gamma + \nu k)(\beta_p\gamma + \beta_i\nu)},\ 0\right)^T$$

2. Moreover, for *t*>0, we have $N(\beta_p\gamma + \beta_i\nu) > \nu\gamma.$

Proof :

For $I \neq 0$ and $P \neq 0$, the third and fourth equations of the system (4) involve : $I^{\bullet} = \frac{\nu P}{\gamma}$ and $E^{\bullet} = \frac{\nu P}{k}$.

By replacing $I^{\bullet}$ and $E^{\bullet}$ in the second equation of the system (4), we obtain $S^{\bullet} = \frac{\nu\gamma}{\beta_p\gamma + \beta_i\nu}$.

As $N = S + E + P + I$, replacing $S^{\bullet}$, $E^{\bullet}$ and $I^{\bullet}$, we obtain : $P^{\bullet} = \frac{k\gamma(N(\beta_p\gamma + \beta_i\nu) - \nu\gamma)}{(\nu\gamma + k\gamma + \nu k)(\beta_p\gamma + \beta_i\nu)}$.

By replacing $P^{\bullet}$ in $I^{\bullet}$ and $E^{\bullet}$, we obtain the endemic equilibrium point $E_p^{\bullet} = (S^{\bullet}, E^{\bullet}, P^{\bullet}, I^{\bullet}, 0)$ as follows :

$$E_p^{\bullet} = \left(\frac{\nu\gamma}{\beta_p\gamma + \beta_i\nu},\ \frac{\nu\gamma(N(\beta_p\gamma + \beta_i\nu) - \nu\gamma)}{(\nu\gamma + k\gamma + \nu k)(\beta_p\gamma + \beta_i\nu)},\ \frac{k\gamma(N(\beta_p\gamma + \beta_i\nu) - \nu\gamma)}{(\nu\gamma + k\gamma + \nu k)(\beta_p\gamma + \beta_i\nu)},\ \frac{k\nu(N(\beta_p\gamma + \beta_i\nu) - \nu\gamma)}{(\nu\gamma + k\gamma + \nu k)(\beta_p\gamma + \beta_i\nu)},\ 0\right)^T \quad (12)$$

Note that if $\hat{S} > S^{\bullet}$, implies : $N > \frac{\nu\gamma}{\beta_p\gamma + \beta_i\nu}$. (13)

From (13), we deduce : $E^{\bullet} > 0$, $P^{\bullet} > 0$ and $I^{\bullet} > 0$. (14)

According to (13) and (14), it comes $N(\beta_p\gamma + \beta_i\nu) > \nu\gamma$.

But according to the proposition (3) and (4), we obtain the following theorem.

Theorem .2. *Let $t > 0$, in the presence of the precontagion or $P \neq 0$, if $\nu = 0$ then the system (1) admits the endemic equilibrium point :*

$$E_p = (0,0,N,0,0)^T. \quad (15)$$

Proof :

It is obvious.

6. The basic reproduction number R_0 of SEPIR's model

From the concept of the basic reproduction number in (G.Sallet (2010)), we have the impression that $R_0 > 1$ then we will observe an increase in cases, thus an epidemic, and that if $R_0 < 1$ then the cases will disappear. Using the condition of the study of the base number R_0 in (L.Chahrazed (2002)) :

- If $R_0 < 1$, the equilibrium point E_0 is locally asymptotically stable ;
- If $R_0 > 1$, the equilibrium point E_0 is unstable.

First of all, we recall the definition of the spectral radius.

Definition .7. *The spectral radius of a matrix A is the maximum value of the modulus of the eigenvalues of A. We note : $\rho(A) = \max_{\lambda \in S_p(A)} |\lambda|$, with $S_p(A)$: the set of eigenvalues of the matrix A.*

Definition .8. *A matrix is said to be Metzler (resp. strict Metzler) if and only if its non-diagonal terms are positive (resp. strictly positive).*

According to the work of (G.Sallet (2010)), we define the R_0 as follows :

Definition .9. *(Basic reproduction rate) If the transmission matrix is stable, then we define* R_0 *by* $R_0 = \rho(-FV^{-1})$*. Since V is a Metzler matrix, it is stable and implies that* $-V^{-1} \geq 0$*. This proves that* $-FV^{-1}$ *is a positive matrix.*

To determine the R_0, one can be satisfied to consider the system (1) on the space (S, E, P, I), since if one knows (S,E, P, I), one knows R, it comes :

$$\begin{cases} \frac{dS(t)}{dt} = -S(t)\left(\beta_p P(t) + \beta_i I(t)\right) \quad ; \\ \frac{dE(t)}{dt} = S(t)\left(\beta_p P(t) + \beta_i I(t)\right) - kE(t) \ ; \\ \frac{dP(t)}{dt} = kE(t) - \nu P\ (t) \ ; \\ \frac{dI(t)}{dt} = \nu P\ (t) - \gamma I(t). \end{cases} \tag{16}$$

The biological domain is *(S, E, P, I) | 0 ≤ S ≤ N, 0 ≤ E ≤ N, 0 ≤ P ≤ N, 0 ≤ I ≤ N*.

The set $\Omega = \{(S,E,P,I) | \ 0 \leq S, 0 \leq E, 0 \leq P, 0 \leq I, S + E + P + I \leq 1\}$. We have a variety of balance points (S,0,0,0) | *0 ≤ S ≤ N* on the S axis. Let us take an equilibrium $(S_0, 0, 0, 0)^T$, that is to say $S_0 = N$, then we have at this point a Disease Free Equilibrium (D.F.E). According to the definition (9), it is enough to consider the carriers of pathogens and *(E, P, I)* for the calculation of the jacobians, and with the notations, it comes :

$$F\ (E,P,I) = \begin{bmatrix} \beta_p SP + \beta_i SI \\ 0 \\ 0 \end{bmatrix} \text{ and } \nu(E,P,I) = \begin{bmatrix} -kE \\ kE - \nu P \\ \nu P - \gamma I \end{bmatrix}.$$

$$\text{So } \mathrm{F(DFE)} = \begin{bmatrix} 0 & \beta_p S & \beta_i S \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \text{ and } \nu(\mathrm{DFE}) = \begin{bmatrix} -k & 0 & 0 \\ k & -\nu & 0 \\ 0 & \nu & -\gamma \end{bmatrix}.$$

$$\text{We obtain } -FV^{-1} = \begin{bmatrix} \frac{\beta_p S}{\nu} + \frac{\beta_i S}{\gamma} & \frac{\beta_p S}{\nu} + \frac{\beta_i S}{\gamma} & \frac{\beta_i S}{\gamma} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}.$$

$$\text{So} \qquad R_0 = \frac{\beta_p S}{\nu} + \frac{\beta_i S}{\gamma}. \tag{17}$$

In equilibrium or at *t*=0, we obtain

$$\mathrm{R_0} = \frac{\beta_p S_0}{\nu} + \frac{\beta_i S_0}{\gamma} = \frac{N\beta_p}{\nu} + \frac{N\beta_i}{\gamma}. \tag{18}$$

7. Study of the stability of the equilibrium point without disease or D.F.E.

According to Lyapunov's Theorem in (J.M.M.ONDO (2012)) :

Consider *U*, a non-empty open of R^n containing 0, and *I* a non-empty interval of R, not right bounded.

Definition .10. *Let* $f : I \times U \to R^n$, *we say that f is locally lipschitzian in the first variable, if for any* $(t_0, x_0) \in I \times U$, *there exists a neighbourhood V of* (t_0, x_0) *in* $I \times U$ *and a constant* $c > 0$ *such that :*

$$\forall\big((t,y),(t',y)\big) \in V^2,\ \ ||f(t,y) - f(t',y)|| \leq c\big||t - t'|\big|.$$

Definition .11. *Let* $t_0 \in$ I *and* $V: I \times U \to R$ *be continuously differentiable such that for all* t $\in$ I. *Suppose that* $x^* = 0$ *is an equilibrium point of the system (2). If there exists a neighbourhood* U_{t_0} *and a function* $V: U_{t_0} \to R,^+$ *continuous and with continuous partial derivatives, such that :*

1. *V being positive definite ;*
2. *The total derivative of V, i.e.* $\dot{V}$, *for the system (2), is negative, then 0 is stable for the system (2), and V is said to be a Lyapunov function* **;**
3. *If, in addition, the total derivative* $\dot{V}$ *for the system (2) is negative, then 0 is asymptotically stable. V is, in this case, a strict Lyapunov function.*

In effect :

The system (16) has a disease-free equilibrium or DFE, which is given by $(S_0, 0, 0, 0) = (N, 0, 0, 0)$**.** In studying the stability of the system, one must adopt the following lemma :

Lemme 1. *Let* $R_0, R_{0_1}, R_{0_2} \in$ R *and* $R_0 = R_{0_1} + R_{0_2}$ *be the basic reproduction number.*
– If $R_0 \leq 1$ *then* $R_{0_1} + R_{0_2} \leq 1$.

– And $\forall R_0 \leq 1$, $\exists R_{0_1} \leq 1$ *and* $\exists R_{0_2} \leq 1$ *such that* $R_0 = R_{0_1} + R_{0_2}$.

Proof :

It is obvious.

We obtain the following theorem :

Theorem .3. *If* $R_0 \leq 1$ *and* $R_{0_2} \geq 1$ *then the DFE is globally asymptotically stable on* Ω.
Proof :

Consider the Lyapunov function $V(S, E, P, I) = E + P + I$**.** By posing $R_{0_1} = \frac{\beta_p S}{\nu}$ and $R_{0_2} = \frac{\beta_i S}{\gamma}$ with $R_0 = R_{0_1} + R_{0_2}$. We obtain :

$$\begin{aligned}\dot{V} &= \dot{E} + \dot{P} + \dot{I} \\ &= \beta_p SP + \beta_i SI - \gamma I \\ &= \nu P\big(R_{0_1}\big) + I(R_{0_2} - 1)\,\gamma.\end{aligned}$$

According to the lemma (1), if $R_0 \leq 1$ then we get $R_{0_1} \leq 1 - R_{0_2}$. So

$$\begin{aligned}\dot{V} &\leq \nu P\big(1 - R_{0_2}\big) + I\big(R_{0_2} - 1\big)\gamma \\ &\leq -\nu P\big(R_{0_2} - 1\big) + I\big(R_{0_2} - 1\big)\gamma \\ &\leq \big(R_{0_2} - 1\big)(I\gamma - \nu P)\end{aligned}$$

$$\leq -\left(R_{0_2} - 1\right)(\nu P - I\gamma).$$

According to the system (16), $\frac{dI(t)}{dt} = \nu P\,(t) - \gamma I(t)$. Thus, we obtain

$$\dot{V} \leq -\left(R_{0_2} - 1\right)\dot{I}$$
$$\leq 0.$$

Moreover $\dot{V} = 0$, if $E + P + I = 0$ or $S = S_0$, R_0=1 and R_{0_2}=1. So the largest invariant set contained in this set is $\Psi = \left\{(S, E, P, I) \in \Omega \mid \dot{V}\,(S, E, P, I) = 0\right\}$ which is reduced to the DFE. Since we are in a positively invariant compact, according to the LaSalle invariance principle in (N.P.Bhatia and G.P.Szego (1970)), the DFE is globally asymptotically stable in Ω.

8. Global stability of the endemic balance

An equilibrium for the system (16), different from the DFE, is given by $(S^\bullet, E^\bullet, P^\bullet, I^\bullet)$ in the proposition (4), where $S^\bullet = \frac{\nu\gamma}{\beta_p\gamma + \beta_i\nu} = \frac{N}{R_0}$, $E^\bullet = \frac{\nu\gamma(N(\beta_p\gamma + \beta_i\nu) - \nu\gamma)}{(\nu\gamma + k\gamma + \nu k)(\beta_p\gamma + \beta_i\nu)} = \frac{\nu\gamma N(R_0 - 1)}{(\nu\gamma + k\gamma + \nu k)\,R_0}$,

$P^\bullet = \frac{k\gamma(N(\beta_p\gamma + \beta_i\nu) - \nu\gamma)}{(\nu\gamma + k\gamma + \nu k)(\beta_p\gamma + \beta_i\nu)} = \frac{k\gamma N(R_0 - 1)}{(\nu\gamma + k\gamma + \nu k)\,R_0}$ and $I^\bullet = \frac{k\nu(N(\beta_p\gamma + \beta_i\nu) - \nu\gamma)}{(\nu\gamma + k\gamma + \nu k)(\beta_p\gamma + \beta_i\nu)} = \frac{k\nu N(R_0 - 1)}{(\nu\gamma + k\gamma + \nu k)\,R_0}$.

This equilibrium is in the simplex, i.e. $0 \leq S^\bullet$, $0 \leq E^\bullet$, $0 \leq P^\bullet$, $0 \leq I^\bullet$ et $S^\bullet + E^\bullet + P^\bullet + I^\bullet \leq N$ if and only if $R_0 > 1$. Clearly $0 \leq E^\bullet$, $0 \leq P^\bullet$, $0 \leq I^\bullet$ is equivalent to $R_0 \geq 1$. Now we can write then :

$$S^\bullet + E^\bullet + P^\bullet + I^\bullet = N.$$

This equilibrium coincide with the DFE. Then, there is a unique equilibrium inside the simplex if and only $R_0 > 1$.

Theorem .4. *If $R_0 > 1$., the DFE is unstable and there is a unique endemic equilibrium $(S^\bullet, E^\bullet, P^\bullet, I^\bullet)$ which is globally asymptotically stable on the Ω domain.*

Proof :

According to the concept of R_0 in the section (5.6), if $R_0 > 1$ the DFE is unstable. Let $\Omega^\bullet$ be the set defined by $\Omega^\bullet = \left\{(S, E, P, I) \mid S \geq \frac{\nu\gamma}{\beta_p\gamma + \beta_i\nu}, E \geq 0, P \geq 0, I \geq 0, S + E + P + I \leq N\right\}$. The set $\Omega^\bullet$ is a positively invariant compact. We consider on $\Omega^\bullet$ the Lyapunov function defined by

$$V(S, E, P, I) = (S - S^\bullet) - \frac{\nu\gamma}{\beta_p\gamma + \beta_i\nu} log\frac{\beta_i S}{\beta_i S^\bullet} + (E - E^\bullet) - E^\bullet log\left(\frac{E}{E^\bullet}\right)$$
$$+ (P - P^\bullet) - P^\bullet log\left(\frac{P}{P^\bullet}\right) + (I - I^\bullet) - I^\bullet log\left(\frac{I}{I^\bullet}\right).$$

It is easy to check that V is positive definited, i.e. $V(S,E,P,I) \geq 0$ and $V(S^{\bullet}, E^{\bullet}, P^{\bullet}, I^{\bullet}) = 0$ if and only if $(S,E,P,I) = (S^{\bullet}, E^{\bullet}, P^{\bullet}, I^{\bullet})$. Its derivative along the trajectories of the system (16) is given by :

$$\dot{V}(S,E,P,I) = \dot{S} - \frac{\nu\gamma}{\beta_p\gamma + \beta_i\nu}\left(\frac{\beta_i\dot{S}}{\beta_i S}\right) + \dot{E} - E^{\bullet}\left(\frac{\dot{E}}{E}\right) + \dot{P} - P^{\bullet}\left(\frac{\dot{P}}{P}\right) + \dot{I} - I^{\bullet}\left(\frac{\dot{I}}{I}\right).$$

$$= -\nu I - \frac{\nu\gamma}{\beta_p\gamma + \beta_i\nu}\left(\frac{\beta_i(-\beta_p SP - \beta_i SI)}{\beta_i S}\right) - \frac{E^{\bullet}(\beta_p SP + \beta_i SI - kE)}{E} - \frac{P^{\bullet}(kE - \nu P)}{P} - \frac{I^{\bullet}(\nu P - \gamma I)}{I}$$

$$= -\nu I - \frac{\nu\gamma}{\beta_p\gamma + \beta_i\nu}\left(\frac{\beta_i(-\beta_p SP - \beta_i SI)}{\beta_i S}\right) - \frac{(N(\beta_p\gamma + \beta_i\nu) - \nu\gamma)}{(\nu\gamma + k\gamma + \nu k)(\beta_p\gamma + \beta_i\nu)}\left(\frac{\nu\gamma(\beta_p SP + \beta_i SI - kE)}{E} + \frac{k\gamma(kE - \nu P)}{P} + \frac{k\nu(\nu P - \gamma I)}{I}\right)$$

$$\leq 0.$$

We conclude that $\dot{V}$ is positive semidefinite. The endemic equilibrium is globally asymptotically stable.

Conclusion

Faced with the new behaviour of micro-organisms that are caused by today's climate change, Among other things, this study has identified a new dynamic process and mechanism of infection of emerging infectious diseases. This study is done to develop a dynamic model adapted to the new behaviour of infectious diseases caused by global warming. A model of the spread of an epidemic highlights a threshold parameter : R_0 and the study of an equilibrium point. Our model allows us to discern the situation in the case where the epidemic will spread and in the case where it will die out. In addition, it also allows us to perform our model stability study.

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