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Infectious Disease Behaviour Resulting In a Public Health Catastrophe

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Abstract

In epidemiology, the search for new behaviour of infectious agents, processes and mechanisms of diffusion of infection of emerging infectious diseases caused by global warming has already led to tangible results. This paper proposes a dynamic endemic model Susceptible, Exposed, Precontaged, Infected, Retired, Susceptible (SEPIRS). This model is specialized in epidemics that persist for a long time and in cases where the infection spreads directly: first between pre-contagious individuals (asymptomatic) and susceptible individuals, second between infectious individuals (symptomatic) and susceptible individuals. This model characterises the individual recovering from an infection to develop a temporary immunity and then become susceptible again after some time.

Keywords: Epidemic modeling, compartmental models, global warming, microorganisms

Introduction

In epidemiology, making a model sufficiently close to reality is a crucial challenge because each infectious disease has its own behaviour. There are infectious diseases that are resistant to vaccines and that generate temporary immunity. This is known as the bifurcation of disease (Greenhalgh (1997)). And there are infectious diseases that have a natural behaviour that the infectious individual, once cured. He does not acquire a permanent immunity, but he becomes susceptible again after some time (J.M.M.ONDO (2012)). In any case, this behaviour probably generates a second or even a third wave of the same epidemic in the same population with different periods. This requires taking into account the existence of individuals who were immune before the epidemic. For before a second wave of the epidemic begins, there are probably groups of temporarily immune individuals in the population. (Greenhalgh (1997)) attempted to solve this problem by working on Hopf bifurcations for the Susceptible, Exposed, Infected, Retired, Susceptible (SEIRS) model, under the hypothesis that some of the susceptible individuals are vaccinated and that there is the acquisition of a temporary immunity. The model is improved by (Melesse and Gumel (2010)) by studying the asymptotic behaviour of such a model with multiple infection steps. They obtained a famous Susceptible, Exposed, Infected, Retired, Susceptible (SEIRS) model of today. But nowadays, this model has a weakness, because today, there is an infectious disease that spreads with a very rapid speed and it is transmitted even between

individuals who do not yet show symptoms (INSPQ (2021)), (WELKER (2020)). This is what (OMS (2019)) and (Hu et al. (2020)) called a new strain of coronavirus or COVID-19. This is incompatible for the SEIRS model.

Moreover, the search for new behaviour of infectious agents, processes and mechanisms of diffusion of infection of emerging infectious diseases caused by global warming has already led to tangible results in our work (Masonova et al. (2021a)) and (Masonova et al. (2021b)). In the present work, we propose a model adapted to this new behaviour of infectious agents to remedy the weakness of existing models in the literature. In the following, our work is divided into ten sections. Section 2 presents the dynamic process of disease infection. We present the different Definitions of hypotheses in Section 3. The objective of proposed model of epidemic is presented in section 4. Section 5 propose the endemic model or SEPIRS. We end with a short conclusion in section 6.

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

According to our work of Masonova et al. (2021a) and Masonova et al. (2021b) that 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**.**

2.1 Mechanism of disease progression

According to our work of Masonova et al. (2021a) and Masonova et al. (2021b) that 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.

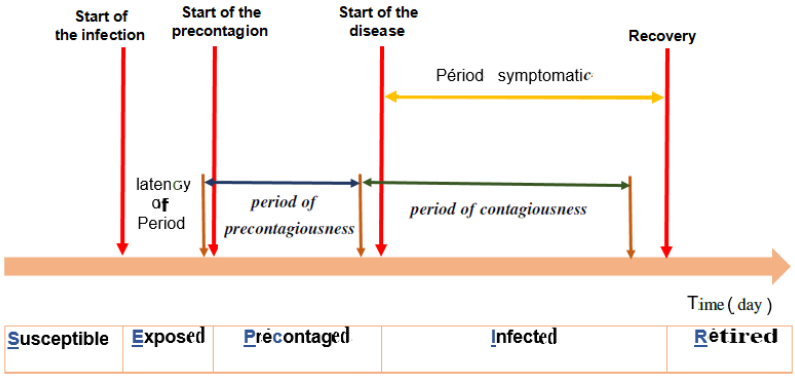


FIG. 1: Representation of the contagion process «SEPIR»

Objective of 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 SEIRS 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.

The endemic model of SEPIRS

In this type of model, we consider that the disease persists and continues to spread during a time interval $[t, t + \Delta t]$ (equivalent to a month or a quarter or a semester or a year). This leads us to consider, over time, the birth rate of the population, the natural mortality of the population and the loss of infectivity of the disease in the population. And we consider that emigration is balanced to immigration of inhabitants.

5.1 Definition of the assumptions of the endemic model of SEPIRS

In order to design our SEPIRS model, we make the following assumptions :

- **A1** : The size of the population 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.
- **A3** : The period of time $\Delta t = dt$ 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 Withdrawn individuals with $S(0) = S_0 > 0$, $P(0) = P_0 > 0$ and/or $I(0) = I_0 > 0$ and $R_0 \geq 0$ in the case of a second or even third wave of the same epidemic ;
- **A5** : We assume that each susceptible individual in a Δt period is exposed, precontaged and then infected and the cured individual is temporarily immune to the infection, and becomes susceptible again after some time ;
- **A6** : The transmission of the infection is done through a direct contact between : firstly, susceptible S and one or more precontaged P with a factor β_p proportionality (also called rate of precontagion or rate of transmission or rate of transmission from the susceptible to the infected), secondly, susceptible S and one or more infected I

with a factor β_i of proportionality (also called rate of infection) and it is admitted that a factor β is the rate of total transmission or of exposure such as

$\beta = \beta_p + \beta_i$.

- **A7** : During the time interval $[t,\ t + \Delta t]$, the population under study is assumed to increase (there are new births) with the birth rate δ. It also suffers the natural death on the susceptible population and temporarily recovery with the mortality rate μ and it suffers in addition the loss of infectivity of the disease only for the sick (infected) individuals with the mortality rate λ. Here, in relation to the aggressiveness of the micro-organism, it is difficult to determine the cause of death of each individual in the Exposed and Precontaged compartments if it is natural or related to this disease. Therefore, we consider here the deaths in these two compartments is already counted and ejected in the rate λ ;
- **A8** : Compartment D is used to store individuals who have died from the disease with a rate of λ in the time interval $[t,\ t + \Delta t]$;
- **A9** : We consider that a constant average number of contacts cannot be applied to all diseases : we can generalise by putting the proportionality coefficients βp and βi which depend on N.

5.2 Schematic of the endemic SEPIRS model

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

We 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), $r > 0$: the rate of retired or recovery or temporary immunity (or transmission from the infected to the Retired), $s > 0$: the rate of susceptibility (or transmission from the Retired to the susceptible), $\lambda > 0$: the rate of infected to die, $\mu > 0$: the rate of natural mortality, $\delta > 0$: the rate of birth.

The endemic model is schematized as in the figure (2) below :

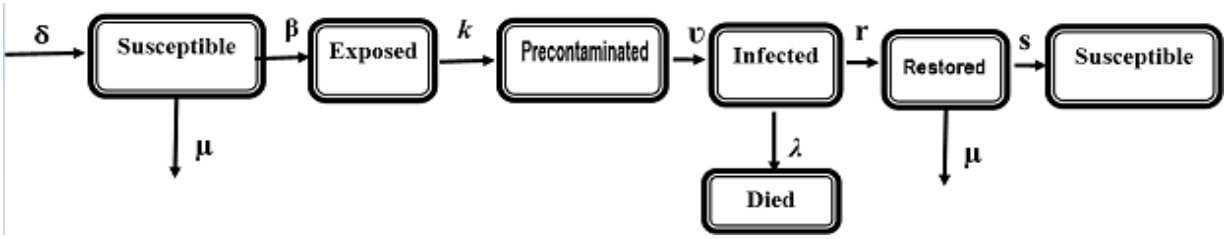


FIG. 2: Drawing of the SEPIRS endemic model

5.3 Differential equation representation of the endemic SEPIRS model

According to the hypothesis (A7) in section (5) above, we consider that during the time interval dt, the Susceptible compartment has increased in the number δN of newborns and in the number sR of individuals losing their immunity. But at the same time, it loses the number $\frac{S}{N}(\beta_p P + \beta_i I)$ of individuals exposed by the disease and the number μS of

individuals who died naturally. According to the hypothesis (A6), we consider the new cases reached by the infection during the time interval *dt* which are equal to $\frac{\beta_i}{N}SI$. And the new cases reached by the precontagion during the interval of time *dt* which are equal to $\frac{\beta_p}{N}SP$.. We obtain the new cases exposed to the disease during the time interval *dt* which will be equal to $\frac{\beta_p}{N}SP + \frac{\beta_i}{N}SI = \frac{S}{N}(\beta_p P + \beta_i I)$. 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) and (A7), we consider that during the time interval *dt* the compartment Infected has increased by νP of precontaged individuals, and, at the same time, it loses the number *rI* of the epidemic deaths and the number *rI* of the cured individuals. According to the hypothesis (A4), (A5) and (A7), we consider that during the time interval *dt* the compartment of Retired benefits the number *R*0 of the temporary immunized individuals and it increased the number *rI* of the infected individuals, and, at the same time, it loses the number *sR* of the individuals lost their immunity and the number *rI* of the naturally dead individuals. And it represents in the form of the following system of differential equations (1):

$$\begin{cases} \frac{dS(t)}{dt} = \delta N + sR(t) - \mu S(t) - \frac{S}{N}\left(\beta_p P(t) + \beta_i I(t)\right); \\ \frac{dE(t)}{dt} = \frac{S}{N}\left(\beta_p P(t) + \beta_i I(t)\right) - kE(t); \\ \frac{dP(t)}{dt} = kE(t) - \nu P(t); \\ \frac{dI(t)}{dt} = \nu P(t) - (r+\lambda)I(t); \\ \frac{dR(t)}{dt} = R_0 + rI(t) - (\mu + s)R(t); \\ \frac{dD(t)}{dt} = \lambda I(t). \end{cases} \quad (1)$$

We assume the initial conditions : $S(0) = S0$; $E(0) = E0$; $P(0) = P0$; $I(0) = I0$; $R(0) = R0$ and the biological domain : $\Omega = \{(S, E, P, I, R), S > 0, P > 0, I > 0, R > 0\}$ which is positively invariant for the system (a set G is said to be positively invariant if $\forall x_0 \in G$ *the trajectory passing through* x_0 *is contained in* G *after* x_0 : *if* $\mathbf{x}$ *is the solution of the system* $X' = F(X)$ *(with* F *of class* C^∞*) verifying* $x(0) = x_0$, *then* $\forall t \geq 0$, $x(t) \in G$). By studying the system (1), we obtain the following theorem :

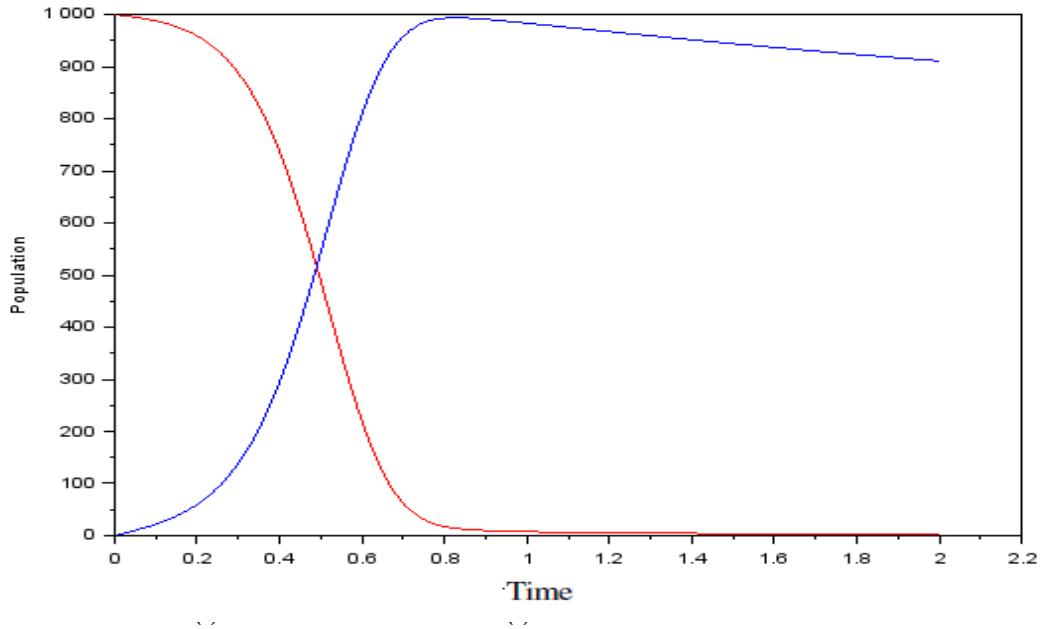
Theorem .1. *Let* $\forall N, \delta, \mu, \lambda, \beta_p, \beta_i, k, \nu, g, r, s \in \mathbb{R}$ *and* $R_0 = 0$, *the absence of infection or I=0, the endemic model SEPI=SEPIS=SEPIR=SEPIRS.*

Proof :

It is obvious that if we put I=0 and $R_0 = 0$ in the four model, they become identical.

5.4 Simulation of the SEPIRS model

The different curves below (obtained with Scilab) already give us an idea of the evolution of the epidemic. For the simulation, we consider here to have an individual precontaged at time t = 0 with N=1000, δ = 0,3 ; βp = 0,2 ; βi = 0,1 ; k=0,4 ; ν = 0,2 ; μ = 0,2 ; λ = 0,3 ; r = 0,5 ; R0 = 0 et s=0,3. We consider a period of time t which depends on the unit of the transmission rates, and it is equivalent to a day or a week or a month or a quarter or a semester (with unit time t day or week or month or quarter or semester). By running the simulation, we obtain the following curves :



Interpretation :

From the figure (3), we note that even with low pre-contagion and infection rates, the epidemic evolves with phenomenal and very rapid speed. Even with average birth rates (δ = 0,3) added to the Susceptible compartment, any Susceptible population is already exposed after only 2^e period of time t.

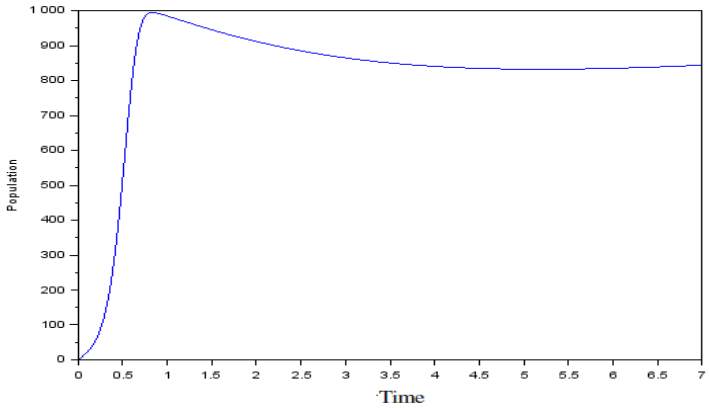


FIG. 4: Curve of E(t) during a phase of the epidemic

Interpretation :

From figure (4), after the phenomenal evolution of the epidemic up to the 1er time period *t*, the curve of the Exposé decreases, and stabilises and becomes endemic after the 7e time period *t* of the epidemic.

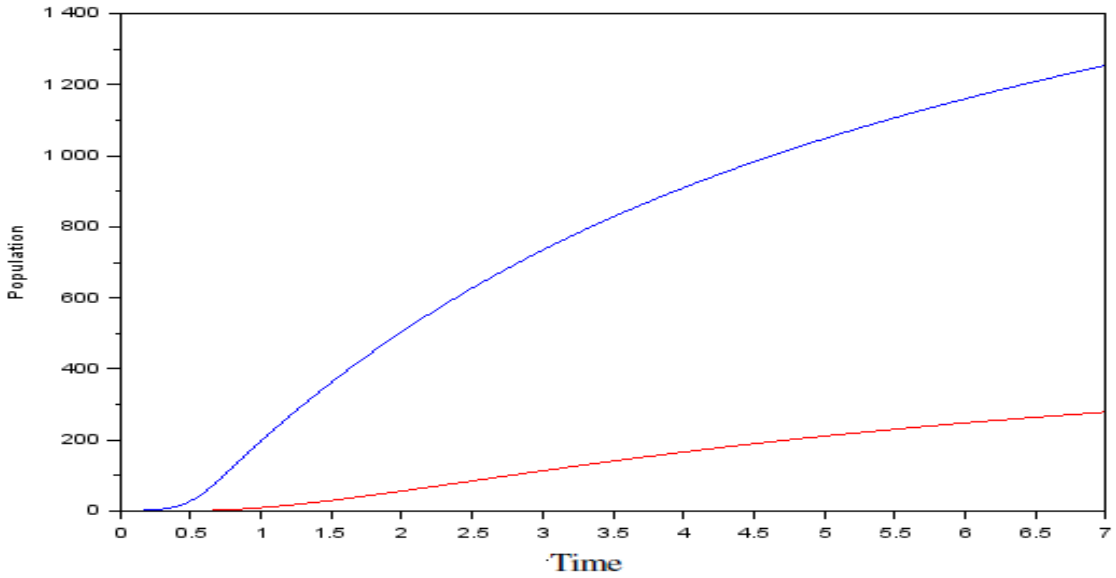


FIG. 5: Curves of I(t) coloured in red and P(t) in blue

Interpretation :

From the figure (5), it appears that even after the 7e period of time *t*, the curves of the Precontaged and the Infected still believe each other and this is a sign of a pandemic.

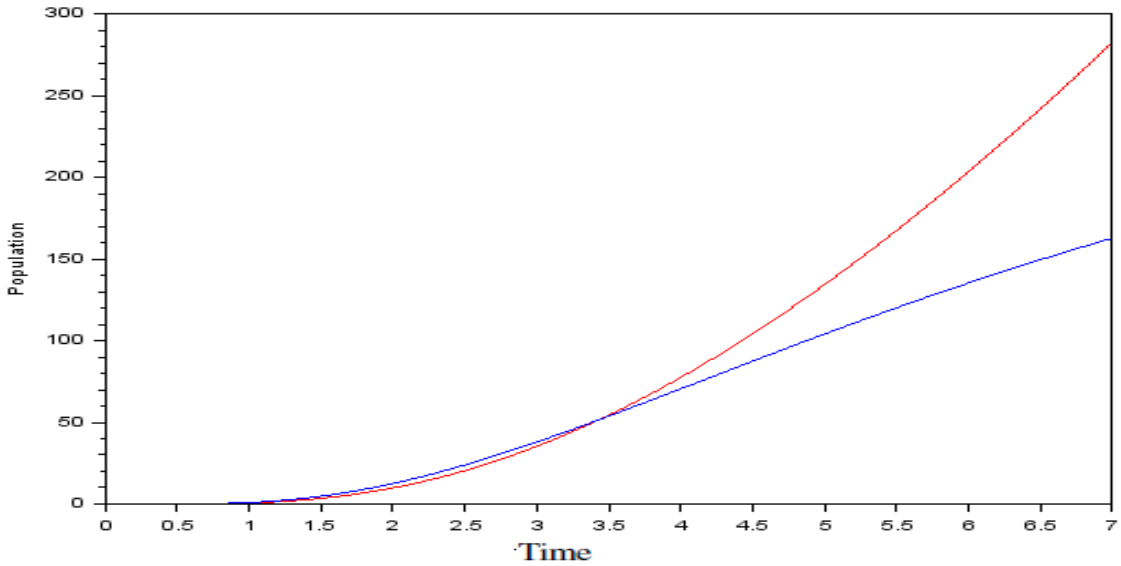


FIG. 6: Curves of D(t) coloured in red and R(t) in blue

Interpretation :

From the figure (6), it appears that after the 7e period of time *t*, the curves of R(t) and D(t) are still increasing and we have recorded almost 150 individuals are recovered and

temporarily immune from this disease, and almost 280 deaths related to this disease in 7e period of time *t* only.

5.5 Study of the equilibrium point of the SEPIRS endemic model

Lyapunov in (J.M.M.ONDO (2012)), defines the equilibrium point as follows :

Définition .1. *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 the two equations below (2) and (3) :*

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

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

where the functions $f : U \to R^n$ *for the system (2) and* $f : I \times U \to R^n$ *for the system (3) sare assumed to be continuous.*
A point "a" is an equilibrium point or equilibrium state or singular point of the system (2) (resp. (3)), if $f(a) = 0$ *(resp. if, for all* $t \in I$, $f(t, a) = 0$ *).*

From the definition (1) of the equilibrium point above, we obtain the following proposition :

Proposition 1. *Let N>0. Then, the system (1), with the condition* $S(0) = S_0$, $E(0) = E_0$, $P(0) = P_0$, $I(0) = I_0$, $R(0) = R0$ *and* $\Omega = \{(S, E, P, R),\ S > 0, P > 0, I > 0, R > 0\}$, *admits a unique solution (S,E,P,I,R) defined on* $[0, +\infty[$.
Proof :

Equilibrium points are calculated in the absence of infection and/or precontagion. The equilibrium point of the model (1) satisfies :

$$\begin{cases} \delta N + sR - \mu S - \dfrac{S}{N}(\beta_p P + \beta_i I) = 0 \\ \dfrac{S}{N}(\beta_p P + \beta_i I) - kE = 0 \\ kE - \nu P = 0 \\ \nu P - (r + \lambda) I = 0 \\ R_0 + rI - (\mu + s) R = 0 \end{cases} \tag{4}$$

In the absence of the infection I=0 and the pre-contagion P=0, we obtain the following proposition :

Corollary .1. *Let N>0, in the absence of infection I=0 and precontagion P=0, then :*

- *At t=0 and R(0) = R0, system (1) admits the equilibrium point :* $E_0 = \left(\frac{\delta N + sR_0}{\mu}, 0, 0, 0, R_0\right)^T$;
- *But if at t=0 and R0 = 0, then the equilibrium point becomes* $E_0 = \left(\frac{\delta N}{\mu}, 0, 0, 0, 0\right)^T$.

Proof :

By replacing P=0 and I=0 in the equations of the system (4), we obtain the equilibrium point : E_0 = ($\hat{S}, \hat{E}, \hat{P}, \hat{I}, \hat{R}$) as follows : $E_0 = \left(\frac{\delta N + sR_0}{\mu}, 0, 0, 0, R_0\right)^T$ considering that in t=0, $R(0) = R_0$.

But if at t=0 and $R0 = 0$, then the equilibrium point becomes $E_0 = \left(\frac{\delta N}{\mu}, 0, 0, 0,0\right)^T$.

In the presence of the precontagion P≠0 and in the absence of the infection I = 0, we obtain the following corollary :

Corollary .2. *Let N>0, in the absence of the infection I=0 and in the presence of the precontagion P≠0, then :*

- *the system (1) admits the following equilibrium point*

$$E_p^* = \left(\frac{\nu N}{\beta_p}, \frac{\delta N\beta_p + s\beta_p R_0 - \mu\nu N}{k\beta_p}, \frac{\delta N\beta_p + s\beta_p R_0 - \mu\nu N}{\nu\beta_p}, 0, R_0\right)^T ;$$

- *Moreover, for all t>0 we have* $\delta N\beta_p + s\beta_p R_0 > \mu\nu N$.

Proof :

By replacing I=0 the system (4) becomes :

$$\begin{cases} \delta N + sR - \mu S - \dfrac{\beta_p SP}{N} = 0 \\ \dfrac{\beta_p SP}{N} - kE = 0 \\ kE - \nu P = 0 \end{cases} \qquad (5)$$

The third equation of the system (5), implies : $E^* = \frac{\nu P}{k}$.

Replacing E^* in the second equation of (5) with $P\neq 0$, we obtain : $S^* = \frac{\nu N}{\beta_p}$.

Replacing S^* in the first equation of (5), we obtain : $P^* = \frac{\delta N\beta_p + s\beta_p R_0 - \mu\nu N}{\nu\beta_p}$ with I=0 implies that $R(t)$ still equals R_0.

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

$$E_p^* = \left(\frac{\nu N}{\beta_p}, \frac{\delta N\beta_p + s\beta_p R_0 - \mu\nu N}{k\beta_p}, \frac{\delta N\beta_p + s\beta_p R_0 - \mu\nu N}{\nu\beta_p}, 0, R_0\right)^T \qquad (6)$$

We note that $\hat{S} > S^*$: $\frac{\delta N + sR_0}{\mu} > \frac{\nu N}{\beta_p}$. (7)

From (7), we deduce that : $E^* = \frac{\delta N\beta_p + s\beta_p R_0 - \mu\nu N}{k\beta_p} > 0.$ (8)

And $P^* = \frac{\delta N\beta_p + s\beta_p R_0 - \mu\nu N}{\nu\beta_p} > 0.$ (9)

According to (7),(8),(9), we then have $\delta N\beta_p + s\beta_p R_0 > \mu\nu N.$

In the presence of the precontagion $P\neq 0$ and the infection $I\neq 0$, we obtain the following corollary:

Corollary .3. *Let $N>0$, in the presence of the precontagion $P\neq 0$ and the infection $I\neq 0$, then :*

- *the system (1) admits the following endemic equilibrium point* $E_p^\bullet = (S^\bullet, E^\bullet, P^\bullet, I^\bullet, R^\bullet)$ *with*

$$S^\bullet = \frac{\nu N(r+\lambda)}{\beta_p(r+\lambda)+\beta_i v},$$

$$E^\bullet = \frac{\nu(r+\lambda)\left((\delta N(\mu+s)+sR_0)(\beta_p(r+\lambda)+\beta_i v)-\mu\nu N(\mu+s)(r+\lambda)\right)}{k(\mu+s)(r+\lambda)(\beta_p\nu(r+\lambda)+\beta_i v^2)-sr\nu(\beta_p(r+\lambda)+\beta_i v)},$$

$$P^\bullet = \frac{(r+\lambda)\left((\delta N(\mu+s)+sR_0)(\beta_p(r+\lambda)+\beta_i v)-\mu\nu N(\mu+s)(r+\lambda)\right)}{(\mu+s)(r+\lambda)(\beta_p\nu(r+\lambda)+\beta_i v^2)-sr\nu(\beta_p(r+\lambda)+\beta_i v)},$$

$$I^\bullet = \frac{\nu\left((\delta N(\mu+s)+sR_0)(\beta_p(r+\lambda)+\beta_i v)-\mu\nu N(\mu+s)(r+\lambda)\right)}{(\mu+s)(r+\lambda)(\beta_p\nu(r+\lambda)+\beta_i v^2)-sr\nu(\beta_p(r+\lambda)+\beta_i v)},$$

$$R^\bullet = \frac{R_0 a + r\nu\left((\delta N(\mu+s)+sR_0)(\beta_p(r+\lambda)+\beta_i v)-\mu\nu N(\mu+s)(r+\lambda)\right)}{(\mu+s)^2(r+\lambda)(\beta_p\nu(r+\lambda)+\beta_i v^2)-sr\nu(\beta_p(r+\lambda)+\beta_i v)},$$

With $a = (\mu+s)(r+\lambda)(\beta_p\nu(r+\lambda)+\beta_i v^2)-sr\nu(\beta_p(r+\lambda)+\beta_i v)$;

- *Moreover, for all $t>0$ we have* $\delta N\beta_p(r+\lambda) + \delta N\beta_i v > \mu\nu N(r+\lambda).$

Proof :

If $P\neq 0$ and $I\neq 0$, the third, fourth and fifth equations of the system (4) involve : $E^\bullet = \frac{\nu P}{k}$, $I^\bullet = \frac{\nu P}{r+\lambda}$ and $R^\bullet = \frac{R_0+rI}{\mu+s} = \frac{(r+\lambda)R_0+r\nu P}{(\mu+s)(r+\lambda)}$.

Carry $E^\bullet$ and $I^\bullet$ into the second equation of (4) and we get : $S^\bullet = \frac{\nu N(r+\lambda)}{\beta_p(r+\lambda)+\beta_i v}$.

Replacing $S^\bullet$, $I^\bullet$ and $R^\bullet$ in the first equation of (4) we get :

$$P^\bullet = \frac{(r+\lambda)\left((\delta N(\mu+s)+sR_0)(\beta_p(r+\lambda)+\beta_i v)-\mu\nu N(\mu+s)(r+\lambda)\right)}{(\mu+s)(r+\lambda)(\beta_p\nu(r+\lambda)+\beta_i v^2)-sr\nu(\beta_p(r+\lambda)+\beta_i v)}.$$

By replacing $P^\bullet$ in $I^\bullet$ and $E^\bullet$, and $I^\bullet$ in $R^\bullet$, we obtain the endemic equilibrium point $E_p^\bullet$ as follows :

$$E_p^\bullet = (S^\bullet, E^\bullet, P^\bullet, I^\bullet, R^\bullet) \quad (10)$$

With $S^\bullet = \frac{\nu N(r+\lambda)}{\beta_p(r+\lambda)+\beta_i v}$,

$$E^\bullet = \frac{\nu(r+\lambda)\left((\delta N(\mu+s)+sR_0)(\beta_p(r+\lambda)+\beta_i v)-\mu\nu N(\mu+s)(r+\lambda)\right)}{k(\mu+s)(r+\lambda)(\beta_p\nu(r+\lambda)+\beta_i v^2)-sr\nu(\beta_p(r+\lambda)+\beta_i v)},$$

$$P^\bullet = \frac{(r+\lambda)\left((\delta N(\mu+s)+sR_0)(\beta_p(r+\lambda)+\beta_i v)-\mu\nu N(\mu+s)(r+\lambda)\right)}{(\mu+s)(r+\lambda)(\beta_p\nu(r+\lambda)+\beta_i v^2)-sr\nu(\beta_p(r+\lambda)+\beta_i v)},$$

$$I^\bullet = \frac{\nu\left((\delta N(\mu+s)+sR_0)(\beta_p(r+\lambda)+\beta_i v)-\mu\nu N(\mu+s)(r+\lambda)\right)}{(\mu+s)(r+\lambda)(\beta_p\nu(r+\lambda)+\beta_i v^2)-sr\nu(\beta_p(r+\lambda)+\beta_i v)},$$

$$R^\bullet = \frac{R_0 a + r\nu\left((\delta N(\mu+s)+sR_0)(\beta_p(r+\lambda)+\beta_i v)-\mu\nu N(\mu+s)(r+\lambda)\right)}{(\mu+s)^2(r+\lambda)(\beta_p\nu(r+\lambda)+\beta_i v^2)-sr\nu(\beta_p(r+\lambda)+\beta_i v)},$$

With $a = (\mu+s)(r+\lambda)(\beta_p\nu(r+\lambda)+\beta_i v^2)-sr\nu(\beta_p(r+\lambda)+\beta_i v)$

Let us note $\widehat{S} > S^\bullet$: $\frac{\delta N}{\mu} > \frac{\nu N(r+\lambda)}{\beta_p(r+\lambda)+\beta_i v}$. (11)

From (11) we deduce: $E^\bullet > 0, P^\bullet > 0, I^\bullet > 0$ and $R^\bullet > 0$. (12)

According to (11),(12), we then have : $\delta N\beta_p(r+\lambda) + \delta N\beta_i v > \mu\nu N(r+\lambda)$. (13)

5.6 The basic reproduction number $\mathcal{R}_0$ of the endemic model SEPIRS

To find the $\mathcal{R}_0$ of the SEPIRS endemic model, we apply the study condition in the work (L.Chahrazed (2002)) on $\mathcal{R}_0$:

- If $\mathcal{R}_0 < 1$, the equilibrium point E_0 is locally asymptotically stable ;
- If $\mathcal{R}_0 > 1$, the equilibrium point E_0 is unstable.

First, we calculate the Jacobian matrix of the linearized system (1) at the equilibrium point E_0 ignoring the death compartment and we obtain :

$$J\left(\frac{\delta N+sR_0}{\mu},0,0,0,R_0\right)=$$

$$\begin{bmatrix} -\mu & 0 & -\frac{\beta_p(\delta N+sR_0)}{N\mu} & -\frac{\beta_i(\delta N+sR_0)}{N\mu} & s \\ 0 & -k & \frac{\beta_p(\delta N+sR_0)}{N\mu} & \frac{\beta_i(\delta N+sR_0)}{N\mu} & 0 \\ 0 & k & -\nu & 0 & 0 \\ 0 & 0 & \nu & -(r+\lambda) & 0 \\ 0 & 0 & 0 & r & -(\mu+s) \end{bmatrix} \quad (14)$$

The characteristic equation of the system (1) in the vicinity of the equilibrium point E_0 is as follows :

$$det(J-\theta I)$$

$$=\begin{bmatrix} -\mu-\theta & 0 & -\frac{\beta_p(\delta N+sR_0)}{N\mu} & -\frac{\beta_i(\delta N+sR_0)}{N\mu} & s \\ 0 & -k-\theta & \frac{\beta_p(\delta N+sR_0)}{N\mu} & \frac{\beta_i(\delta N+sR_0)}{N\mu} & 0 \\ 0 & k & -\nu-\theta & 0 & 0 \\ 0 & 0 & \nu & -(r+\lambda)-\theta & 0 \\ 0 & 0 & 0 & r & -(\mu+s)-\theta \end{bmatrix}=0$$

He's coming :

$$det(J-\theta I)=-(\mu+\theta)\left(\theta^4+\theta^3(k+\nu+r+\lambda+\mu+s)-\theta^2\left(k\nu+kr+k\lambda+k\mu+ks+\nu r+\nu\lambda+\mu\nu+s\nu+\mu r+sr+\lambda\mu+s\lambda+\frac{\beta_p k(\delta N+sR_0)}{N\mu}\right)+\theta\left(k\nu r+k\nu\lambda+k\nu\mu+k\nu s+k\mu s+k\mu\lambda+k\lambda s+\mu r\nu+sr\nu+\lambda\mu\nu+s\lambda\nu+\frac{\beta_p kr(\delta N+sR_0)}{N\mu}+\frac{\beta_p k\lambda(\delta N+sR_0)}{N\mu}+\frac{\beta_p k(\delta N+sR_0)}{N}+\frac{\beta_p ks(\delta N+sR_0)}{N}\right)+k\nu\mu r+srk\nu+k\lambda\mu\nu+k\nu s\lambda+\frac{\beta_p kr(\delta N+sR_0)}{N}+\frac{\beta_p krs(\delta N+sR_0)}{N\mu}+\frac{\beta_p k\lambda(\delta N+sR_0)}{N}+\frac{\beta_p ks\lambda(\delta N+sR_0)}{N\mu}+\frac{\beta_p kr\nu(\delta N+sR_0)}{N\mu}\right)=0$$

Then, the first eigenvalue is $\theta = -\mu$ and it remains to study the equation :

$$\theta^4+\theta^3(k+\nu+r+\lambda+\mu+s)$$

$$-\theta^2\left(k\nu+kr+k\lambda+k\mu+ks+\nu r+\nu\lambda+\mu\nu+s\nu+\mu r+sr+\lambda\mu+s\lambda+\frac{\beta_p k(\delta N+sR_0)}{N\mu}\right)$$

$$+\theta\left(k\nu r+k\nu\lambda+k\nu\mu+k\nu s+k\mu s+k\mu\lambda+k\lambda s+\mu r\nu+sr\nu+\lambda\mu\nu+s\lambda\nu+\frac{\beta_p kr(\delta N+sR_0)}{N\mu}+\frac{\beta_p k\lambda(\delta N+sR_0)}{N\mu}+\frac{\beta_p k(\delta N+sR_0)}{N}+\frac{\beta_p ks(\delta N+sR_0)}{N}\right)$$

$$+ \ \mathrm{k}\nu\mu\mathrm{r} + \ srk\nu + \mathrm{k}\lambda\mu\nu + \mathrm{k}\nu s\lambda + \frac{\beta_p kr(\delta \mathrm{N} + s\mathrm{R}_0)}{N} + \frac{\beta_p krs(\delta \mathrm{N} + s\mathrm{R}_0)}{N\mu} + \frac{\beta_p k\lambda(\delta \mathrm{N} + s\mathrm{R}_0)}{N} + \frac{\beta_p ks\lambda(\delta \mathrm{N} + s\mathrm{R}_0)}{N\mu} + \frac{\beta_p kr\nu(\delta \mathrm{N} + s\mathrm{R}_0)}{N\mu} = 0$$

We assume that the above equation is the characteristic equation of the submatrix J_1 :

$$\mathrm{J}_1 = \begin{bmatrix} -\mathrm{k} & \frac{\beta_p(\delta \mathrm{N} + s\mathrm{R}_0)}{N\mu} & \frac{\beta_i(\delta \mathrm{N} + s\mathrm{R}_0)}{\mathrm{N}\mu} & 0 \\ \mathrm{k} & -\nu & 0 & 0 \\ 0 & \nu & -(\mathrm{r} + \lambda) & 0 \\ 0 & 0 & \mathrm{r} & -(\mu + \mathrm{s}) \end{bmatrix} \quad (15)$$

We have the *trace* $(\mathrm{J}_1) = -[\mathrm{k} + \nu + \mathrm{r} + \lambda + \mu + \mathrm{s}] < 0$, so :

$$\det(\mathrm{J}_1) = \mathrm{k}\nu(\mathrm{r} + \lambda)(\mu + \mathrm{s}) - \frac{\beta_p k(\delta \mathrm{N} + s\mathrm{R}_0)(\mathrm{r} + \lambda)(\mu + \mathrm{s})}{N\mu} - \frac{\beta_i \mathrm{k}\nu(\delta \mathrm{N} + s\mathrm{R}_0)(\mu + \mathrm{s})}{\mathrm{N}\mu} \quad (20)$$

If $det(J_1) > 0$, we get : $\frac{\frac{\delta \mathrm{N} + s\mathrm{R}_0}{N\mu}(\beta_p(\mathrm{r}+\lambda) + \beta_i\nu)}{\nu(\mathrm{r}+\lambda)} < 1.$

According to the Varga and Poincaré-Lyapunov theorem of linearization in (G.Sallet (2010)), $\mathcal{R}_0$ is defined by the expression below :

$$\mathcal{R}_0 = \frac{(\delta \mathrm{N} + s\mathrm{R}_0)\left(\beta_p(\mathrm{r} + \lambda) + \beta_i\nu\right)}{\mathrm{N}\mu\nu(\mathrm{r} + \lambda)}. \quad (17)$$

5.7 Study of the stability of the equilibrium point E_0

1. **Local stability**

According to the work of L.Chahrazed (2002), we define the local stability of E_0 :

Definition .2. *We say that E_0 is locally asymptotically stable if and only if the trace of the Jacobian matrix in the neighbourhood of E_0 is strictly negative and the determinant is strictly positive.*

In effect

From equations (15) and (16) above, we deduce that :

$$\begin{cases} trace\,(\mathrm{J}_1) = -[\mathrm{k} + \nu + \mathrm{r} + \lambda + \mu + \mathrm{s}] < 0\,; \\ \det(\mathrm{J}_1) = \mathrm{k}\nu(\mathrm{r} + \lambda)(\mu + \mathrm{s}) - \frac{\beta_p k(\delta \mathrm{N} + s\mathrm{R}_0)(\mathrm{r} + \lambda)(\mu + \mathrm{s})}{N\mu} - \frac{\beta_i \mathrm{k}\nu(\delta \mathrm{N} + s\mathrm{R}_0)(\mu + \mathrm{s})}{\mathrm{N}\mu} > 0. \end{cases}$$

Thus, we see that if the conditions in (18) are met then the equilibrium point *E*0 of the system (1) is unique and remains locally asymptotically stable.

2. Global stability

According to Lyapunov's method in the works (Richard (1969)), (Moulay (1969)) and (Richard (2012)), we obtain the definitions below . We consider that *U* always designates a non-empty open of R^n ($n \in N^*$) containing 0 and *I* a non-empty interval of R, not bounded on the right.

Définition .8. *Let* $f : I \times U \to R^n$ *be a continuous application and a Cauchy-Lipschitz function, we associate the system :* $\dot{x} = f(x, t)$ *(*),* $\forall t_0 > 0$, $\forall t \geq t_0$, *we have* $x \in R^n$ *et* $x(t, t_0, x_0)$ *denotes a solution of the system such that* $x(t_0) = x_0$.
An equilibrium point x_e *such that for all t,* $f(x_e, t) = 0$ *is (globally) attractive if the function* $\varphi(t, t_0, x_0)$ *tends to* x_e *when t tends to* $+\infty$.

Définition .9. *We say that* x_e *is an asymptotically stable equilibrium point, if it is a stable equilibrium point and if the domain of attraction of* x_0 *is a neighbourhood of* x_0.

Définition .10. *Let* x_e *be a non-empty compact of U, we consider the system (*). We say that* x_e *is a globally asymptotically stable equilibrium point for the system (*) if :*

1. x_e *is stable on the system (*)*
2. *for all* $t_0 \in I$, *and* $x_0 \in U$, $x(t, t_0, x_0)$ *is defined for all* $t \geq t_0$ *and* $\lim_{t \to +\infty} d\big(x(t, t_0, x_0, x_e)\big) = 0$.

According to the definitions (8), (9) and (10) concerning the global stability of the point *E*0, we proceed to the study of global stability :

Lemme 1. *The number of susceptibles S in the model (1) verifies the relation (19) below:*

$$\lim_{t \to +\infty} \sup S(t) \leq \frac{\delta N + sR_0}{\mu}. \qquad (19)$$

Proof :

According to the model (1), we have the equation $\dot{S} = \delta N + sR - \mu S - \frac{\beta_p SP}{N} - \frac{\beta_i SI}{N}$, avec $S(0) = S_0$ and $R(0) = R0$. And it is obvious that :

$$\dot{S} < \delta N + sR - \mu S(t).$$

For $t > 0$, in the absence of the infection $I = 0$ et $R0 > 0$, we obtain :

$$\dot{S} < \delta N + sR_0 - \mu S(t).$$

Assume that :

$$\dot{Z} = \delta N + sR_0 - \mu S(t). \qquad (20)$$

With the initial condition $Z(0) = Z_0 = S_0$.

And we have $Z(t), S(t) \in C^1[0, +\infty[$, $Z(0) = Z_0 = S_0 > 0$ et $t \in [0, +\infty[$.

Solving the equation (20) which is an ordinary linear differential equation of the first order in time $t > 0$ with $Z(0) = Z_0$, we obtain :

$$Z(t) = Z_0 e^{-\mu t} + \frac{\delta N + sR_0}{\mu}(1 - e^{-\mu t}).$$

Determining the limit of $Z(t)$ when t goes to infinity, we obtain :

$$\lim_{t\to+\infty} Z(t) = \frac{\delta N + sR_0}{\mu}.$$

So

$$\lim_{t\to+\infty} \sup S(t) \leq \lim_{t\to+\infty} Z(t) = \frac{\delta N + sR_0}{\mu}.$$

We now have the following theorem :

Theorem .2. *If* $\frac{(\delta N+sR_0)(\beta_p(r+\lambda)+\beta_i\nu)}{N\mu\nu(r+\lambda)} < 1$ *then the equilibrium point* E_0 *of the system* (1) *is globally asymptotically stable.*

Proof :

Using (19) for $\eta > 0$ then there exists $T_1 > 0$ such that $S(t) \leq \frac{\delta N+sR_0}{\mu} + \eta$, for $t > T_1$. According to the system (1), we obtain :

$$\dot{E}(t) \leq \frac{\beta_p}{N}\left(\frac{\delta N + sR_0}{\mu} + \eta\right)P(t) + \frac{\beta_i}{N}\left(\frac{\delta N + sR_0}{\mu} + \eta\right)I(t) - kE(t);$$

$$\dot{P}(t) \leq kE(t) - \nu P(t);$$

$$\dot{I}(t) \leq \nu P(t) - (r+\lambda)I(t);$$

$$\dot{R}(t) \leq R_0 + rI(t) - (\mu+s)R(t).$$

Then for $t > T_1$, we pose :

$$\dot{J}(t) = \beta_p\left(\frac{\delta}{\mu} + \frac{sR_0+\mu\eta}{\mu N}\right)K(t) + \beta_i\left(\frac{\delta}{\mu} + \frac{sR_0+\mu\eta}{\mu N}\right)L(t) - kJ(t);$$

$$\dot{K}(t) = kJ(t) - \nu K(t);$$

$$\dot{L}(t) = \nu K(t) - (r+\lambda)L(t).$$

$$\dot{M}(t) = M_0 + rL(t) - (\mu+s)M(t).$$

We obtain the matrix D defined in the following way :

$$D = \begin{bmatrix} -k & \beta_p\left(\frac{\delta}{\mu} + \frac{sR_0+\mu\eta}{\mu N}\right) & \beta_i\left(\frac{\delta}{\mu} + \frac{sR_0+\mu\eta}{\mu N}\right) & 0 \\ k & -\nu & 0 & 0 \\ 0 & \nu & -(r+\lambda) & 0 \\ 0 & 0 & r & -(\mu+s) \end{bmatrix} \quad (21)$$

We have the *trace(D)* $= -(k + \nu + r + \lambda + \mu + s)$.

And for the determinant, we have :

$$\det(D) = k\nu(r+\lambda)(\mu+s) - \beta_p k(r+\lambda)(\mu+s)\left(\frac{\delta}{\mu} + \frac{sR_0+\mu\eta}{\mu N}\right) - \beta_i k\nu(\mu+s)\left(\frac{\delta}{\mu} + \frac{sR_0+\mu\eta}{\mu N}\right).$$

If $\frac{(\delta N+sR_0)(\beta_p(r+\lambda)+\beta_i\nu)}{N\mu\nu(r+\lambda)} < 1$ and $\eta <<< 0$, then *det(D)* > 0 implies :

$$k\nu(r+\lambda)(\mu+s) - \beta_p k(r+\lambda)(\mu+s)\left(\frac{\delta}{\mu} + \frac{sR_0+\mu\eta}{\mu N}\right) - \beta_i k\nu(\mu+s)\left(\frac{\delta}{\mu} + \frac{sR_0+\mu\eta}{\mu N}\right) > 0.$$

It comes :$\frac{(\delta N+sR_0)(\beta_p(r+\lambda)+\beta_i\nu)}{N\mu\nu(r+\lambda)} < 1.$

So

$\lim_{t\to+\infty} J(t) = 0$, in comparison with the system (1) $\lim_{t\to+\infty} E(t) = 0$;
$\lim_{t\to+\infty} K(t) = 0$, in comparison with the system (1) $\lim_{t\to+\infty} P(t) = 0$;
$\lim_{t\to+\infty} L(t) = 0$, in comparison with the system (1) $\lim_{t\to+\infty} I(t) = 0$;
$\lim_{t\to+\infty} M(t) = M_0$, in comparison with the system (1) $\lim_{t\to+\infty} R(t) = R_0$.

As $S(t) \le \frac{\delta N+sR_0}{\mu} + \eta$, for $t > T_1$.

And if $\lim_{t\to+\infty} S(t) = \frac{\delta N+sR_0}{\mu}$, then $\lim_{t\to+\infty} P(t) = 0$, $\lim_{t\to+\infty} I(t) = 0$ and $\lim_{t\to+\infty} R(t) = R_0$, for all $\theta > 0$, $\rho > 0$ and $R_0 > 0$, there exists $T_2 > 0$ such that : $P(t) < \theta$, $I(t) < \rho$ and $R(t) < R_0$, for $t > T_2$.

Let T_3, = *max*(T_1 , T_2), for $t > T_3$, we get:

$$P(t) < \theta, I(t) < \rho, \; R(t) < R_0 \; and \; S(t) \le \frac{\delta N + sR_0}{\mu} + \eta \qquad (22)$$

It comes : $\dot{S} > \delta N + sR_0 - \mu S - \frac{\beta_p}{N}\left(\frac{\delta N+sR_0}{\mu} + \eta\right)\theta - \frac{\beta_i}{N}\left(\frac{\delta N+sR_0}{\mu} + \eta\right)\rho.$

So $\dot{S} + \mu S > \delta N + sR_0 - \frac{\beta_p}{N}\left(\frac{\delta N+sR_0}{\mu} + \eta\right)\theta - \frac{\beta_i}{N}\left(\frac{\delta N+sR_0}{\mu} + \eta\right)\rho.$

We consider that:

$$\dot{V}(t) + \mu V(t) = \delta N + sR_0 - \frac{\beta_p}{N}\left(\frac{\delta N+sR_0}{\mu} + \eta\right)\theta - \frac{\beta_i}{N}\left(\frac{\delta N+sR_0}{\mu} + \eta\right)\rho ;$$

$$V(T_3) = (V)_0.$$

Solving this first-order linear differential equation, we get :

$$V(t) = (V)_0 e^{-\mu(t-T_3)} + \frac{1}{\mu}\left(\delta N + sR_0 - \frac{\beta_p}{N}\left(\frac{\delta N + sR_0}{\mu} + \eta\right)\theta - \frac{\beta_i}{N}\left(\frac{\delta N + sR_0}{\mu} + \eta\right)\rho\right)\left(1 - e^{-\mu(t-T_3)}\right),$$

$for\ t > T_3.$

Let's put $\eta_1 = \frac{\beta_p}{N}\left(\frac{\delta N + sR_0}{\mu} + \eta\right)\theta + \frac{\beta_i}{N}\left(\frac{\delta N + sR_0}{\mu} + \eta\right)\rho$.

It comes : $V(t) = (V)_0 e^{-\mu(t-T_3)} + \frac{\delta N + sR_0 - \eta_1}{\mu}\left(1 - e^{-\mu(t-T_3)}\right)$.

So $\lim_{t\to+\infty} V(t) = \frac{\delta N + sR_0 - \eta_1}{\mu}$.

Since $S(t), V(t) \in C^1([0, +\infty])$ and $S(T_3) = V(T_3)$, we have: $S(t) \geq V(t)$, for $t > T_3$.

Means that

$$\lim_{t\to+\infty} inf\ S(t) \geq \frac{\delta N + sR_0 - \eta_1}{\mu}. \qquad (23)$$

From (22) and (23) if we choose η_1, θ and ρ very small and $t > T_4 > T_3$, then we get :

$$\frac{\delta N + sR_0}{\mu} - \frac{\eta_1}{\mu} < S(t) < \frac{\delta N + sR_0}{\mu} + \frac{\eta_1}{\mu}.$$

Moving to the limit : $\lim_{t\to+\infty} S(t) = \frac{\delta N + sR_0}{\mu}$.

Hence the equilibrium point E_0 of the system (1) is globally asymptotically stable.

5.8 Local stability of the equilibrium E^*

By studying the local stability of E^*, we obtain the following theorem :

Theorem .3. *If $\mathcal{R}_0 > 1$, then the endemic equilibrium E^* of the system (1) is locally asymptotically stable.*

Proof :

According to the proposition in the works of L.Chahrazed (2002) and CHABOUR (2000), we characterize the local stability of E^*, as follows :

Proposition 2. *The epidemic is locally asymptotically stable if and only if all the eigenvalues of the Jacobian matrix J have a negative real part.*

According to the proposition (2), we define J as follows :

$$J(S^*, E^*, P^*, I^*, R^*) = \begin{bmatrix} -\mu - \frac{\beta_p P^*}{N} & -\frac{\beta_i I^*}{N} & 0 & -\frac{\beta_p S^*}{N} & -\frac{\beta_i S^*}{N} & s \\ \frac{\beta_p P^*}{N} + \frac{\beta_i I^*}{N} & -k & \frac{\beta_p S^*}{N} & \frac{\beta_i S^*}{N} & 0 \\ 0 & k & -\nu & 0 & 0 \\ 0 & 0 & \nu & -(r+\lambda) & 0 \\ 0 & 0 & 0 & r & -(\mu+s) \end{bmatrix}$$

The eigenvalues can be determined by solving the equation $det(J - \theta I)$:

$$\det(\mathrm{J} - \theta \mathrm{I}) = \begin{bmatrix} -\mu - \frac{\beta_p P^*}{N} & -\frac{\beta_i I^*}{N} - \theta & 0 & -\frac{\beta_p S^*}{N} & -\frac{\beta_i S^*}{N} & s \\ \frac{\beta_p P^*}{N} & +\frac{\beta_i I^*}{N} & -k - \theta & \frac{\beta_p S^*}{N} & \frac{\beta_i S^*}{N} & 0 \\ 0 & & k & -\nu - \theta & 0 & 0 \\ 0 & & 0 & \nu & -(r + \lambda) - \theta & 0 \\ 0 & & 0 & 0 & r & -(\mu + s) - \theta \end{bmatrix}$$

So the characteristic function is written with the coefficients defined below ::

$\theta^5 + A\theta^4 + B\theta^3 + C\theta^2 + D\theta + E = 0.$

The coefficients are :

$A = 2\mu + k + \nu + r + \lambda + s + \frac{1}{N}\left(\beta_i I^* + \beta_p P^*\right)$;

$B = (k + \nu + r + \lambda + \mu + s)\left(\mu + \frac{1}{N}\left(\beta_p P^* + \beta_i I^*\right)\right) + (r + \lambda + \mu + s)(k + \nu) + (\mu + \mathbf{s})(r + \lambda) + k\left(\nu - \frac{\beta_p S^*}{N}\right)$;

$C = \left((r + \lambda + \mu + s)(k + \nu) + (\mu + \mathbf{s})(r + \lambda) + k\left(\nu - \frac{\beta_p S^*}{N}\right)\right)\left(\mu + \frac{1}{N}\left(\beta_p P^* + \beta_i I^*\right)\right) + (\mu + \mathbf{s})(k + \nu)(r + \lambda) + k\left(\frac{\beta_p S^*}{N}\right)\left(\frac{1}{N}\left(\beta_p P^* + \beta_i I^*\right)\right) + (r + \lambda + \mu + s)\left(\nu - \frac{\beta_p S^*}{N}\right) - \frac{\beta_i S^*}{N} k\nu$;

$D = \left(k\nu(r + \lambda + \mu + s) + (\mu + \mathbf{s})(k + \nu)(r + \lambda) - \frac{\beta_p S^*}{N}(r + \lambda + \mu + s) - \frac{\beta_i S^*}{N} k\nu\right)\left(\mu + \frac{1}{N}\left(\beta_p P^* + \beta_i I^*\right)\right) + k\nu(\mu + \mathbf{s})\left(r + \lambda - \frac{\beta_i S^*}{N}\right) + k\left(\frac{1}{N}\left(\beta_p P^* + \beta_i I^*\right)\right)\left((r + \lambda + \mu + s)\left(\frac{\beta_p S^*}{N}\right) + \frac{\beta_i S^*}{N}\nu\right)$;

$E = \left(k\nu(\mu + \mathbf{s})\left(r + \lambda - \frac{\beta_i S^*}{N}\right)\right)\left(\mu + \frac{1}{N}\left(\beta_p P^* + \beta_i I^*\right)\right) + k\nu(\mu + \mathbf{s})\left(\frac{1}{N}\left(\beta_p P^* + \beta_i I^*\right)\right)\left((r + \lambda)\left(\frac{\beta_p S^*}{N}\right) + \frac{\beta_i S^*}{N}\nu\right) - k\nu rs\left(\frac{1}{N}\left(\beta_p P^* + \beta_i I^*\right)\right).$

We have $A > 0$ and $B, C, D, E > 0$ *if* $\nu - \frac{\beta_p S^*}{N} > \mathbf{0}$.

Using the Routh-Hurwitz criterion in the work of (J.M.M.ONDO (2012)), we have : *AB - C > 0.*

We calculate *AB - C*, we get :

$$
\begin{aligned}
&\mathrm{AB} - \mathrm{C} \\
&\quad = (\mathrm{k} + \nu + \mathrm{r} + \lambda + \mu + \mathrm{s})\left(\left(\mu + \frac{1}{\mathrm{N}}(\beta_p P^* + \beta_i I^*)\right)^2\right. \\
&\quad \left. + (\mathrm{k} + \nu)(\mathrm{r} + \lambda + \mu + \mathrm{s})\right) + \frac{\beta_i S^*}{\mathrm{N}} k\nu \\
&\quad + (\mu + \mathbf{s})(\mathrm{r} + \lambda)(\mathrm{r} + \lambda + \mu + \mathrm{s}) + k(\mathrm{k} + \nu)\left(\nu - \frac{\beta_p S^*}{\mathrm{N}}\right) \\
&\quad - \left(\frac{\beta_p S^*}{\mathrm{N}}\right)\left(\frac{1}{\mathrm{N}}(\beta_p P^* + \beta_i I^*)\right).
\end{aligned}
$$

Therefore E^* is locally asymptotically stable.

Conclusion

In spite of the limits induced by this model, it has a lot of originality to take into account if we want to predict and study the new behaviours of emerging infectious diseases within a population. Indeed, this model does not give a direct solution to an epidemic, but thanks to the multitude of parameters (study of $\mathcal{R}_0$, stability, equilibrium point) and the simulation, it offers us the means to better understand the dynamics of the propagation of infectious diseases caused by global warming. The proposed model helps us to make more radical decisions to better prepare for the development of measures to maintain public health.

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