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A Model of the Propagation Endemic of Emerging Infectious Diseases SEPI

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Abstract: ***In the field of epidemiology, there are two classes of dynamic models. There is the class of temporary models specialized for short-lived epidemics and the class of endemic models reserved for epidemics that persist for a long time. In the light of the new temporary dynamic model Susceptible, Exposed, Precontaged, Infected (SEPI) (1erclass) that we have proposed and in order to make our model more efficient, we will develop the endemic model SEPI (2thclass) and to propose the different studies relating to this model. This endemic SEPI model is specialized in epidemics that persist for a long time and 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

1. Introduction

In 1760, Daniel Bernoulli proposed the first epidemic model. In 1906, W. H. Hamer presented the first dynamic compartmental model. Since then, researchers have continued to improve the various existing models in order to arrive at a good model. A good model is one that is close enough to reality to provide valid and useful conclusions (J.M.M.ONDO (2012)).

In addition, it often happens that the epidemic persists and lasts for several periods in a population. This is called an endemic. This calls into question the effectiveness of the various models because they are developed in the case of temporary epidemic phenomena. To remedy the weaknesses of the epidemiological models, M. Fan (2001) incorporated the parameters immigration, birth and mortality by the disease in the Susceptible, Exposed, Infected (SEI) model proposed by (L.Q.Gao (1995)) in order to arrive at an endemic model. Then, other researchers such as (G. Li and al. (2006)) followed their method to apply to other models. But nowadays, this model presents weaknesses, because today, there are infectious diseases that spread with a very rapid speed and are transmitted even between individuals who do not yet present symptoms INSPQ (2021), Y.WELKER (2020), Alizon and al. (2020). This is what (OMS (2019)) and (Z.Hu and al. (2020)) called a new strain of corona virus or COVID-19. This is inconsistent with the SEI model hypothesis.

In the light of the new dynamic process of infection of infectious diseases caused by global warming and in order to make our SEPI model into a good model, this paper proposes an endemic SEPI model adapted to this new behaviour of infectious diseases caused by global warming. In what follows,

Our work is divided into ten sections. Section 2 remember the definition of assumptions. Section 3 defines the assumptions of the SEPI endemic model. Section 4 presents a schematic of the SEPI endemic model. Section 5develops the differential equations of the SEPI endemic model. Section 6 presents the simulation study of the SEPI model. Section 7 studies the equilibrium point of the SEPI endemic model. Section 8 determines the basic reproduction number. Section 9 proposes the study of the stability of the equilibrium point. Section 10 studies the local stability of the equilibrium point. Section 11 provides a conclusion.

2. Reminder of the definition of assumptions

According to the work of Masonova et al. (2021):

Definition 1: *An individual who has been infected with the disease pathogen and can also transmit the disease, but has no symptoms, is referred to as a* ***precontagious or precontaminated*** *individual.*

Definition 2: ***Precontaged*** *represents a compartment where the disease requires a period of pre-contagion. Pre-contagious individuals are capable of transmitting the disease into the population, but they do not yet show symptoms of the disease. They are therefore assigned to this compartment with the rate k called* ***precontagiousness rate****. In the following, the letter P will be used to refer to individuals who are infected and contagious, but do not yet represent symptoms of the disease.*

Definition 3: *The* period of ***precontagiousness*** *is a period of time when an infected individual does not yet show symptomatic signs of the disease, but can transmit the disease to another individual.*

Definition 4: *The **infected** compartment represents those who are not only already infected and have shown symptoms of the disease, but are 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 with symptoms of the disease and whose health is impaired) transmits a disease to the other individual.*

In the figure (1), we illustrate the context in which this event takes place: This schematic presentation shows the different phases (disease states).

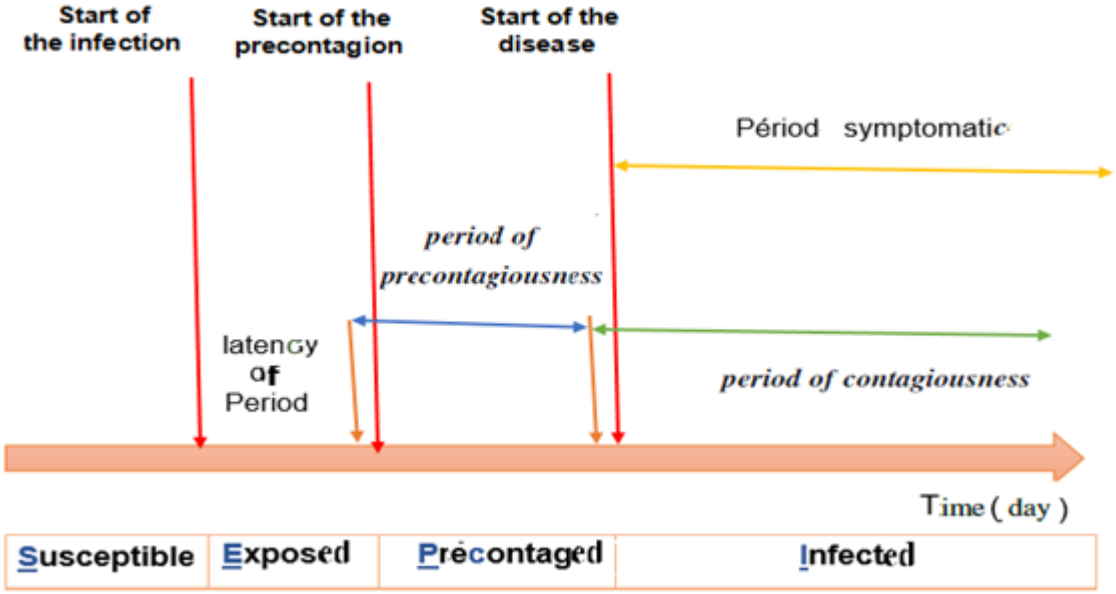

Figure 1: Representation of the contagion process

3. Assumptions of the endemic SEPI model

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

In order to develop the endemic model of SEPI, 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 with $S(0) = S_0>0$, $P(0) = P_0>0$and/or $I(0) = I_0>0$.
- **A5**: We admit that each susceptible individual in a period Δt is exposed, precontaged then infected.
- **A6**: The transmission of the infection is done through a direct contact between: firstly, susceptible Sand 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**: An infected individual remains and remains contagious until the end of his life in the rate λ.
- **A8**: Compartment D is used to store individuals who have died as a result of the disease at a rate λ in the time interval $[t, \quad t + \Delta t]$. Here, with respect 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 unnatural or not by the disease. Therefore, we consider here the deaths in these two compartments are already counted and included in the rate of λ.
- **A9**: During the time interval $[t; t+\Delta t]$, we admit that the population studied increases (birth of children)with the birth rate δ; it also undergoes natural death with the mortality rate $\mu$and it suffers in addition the loss of infectiousness of the disease only for the sick individuals (infected) with the mortality rate λ.
- **A10**: We consider that the disease studied does not transmit vertically (vertical transmission is from mother to child or genetically).
- **A11**: We consider that a constant average number of contacts cannot apply to all diseases: we can generalise a little by putting the proportionality coefficients β_p and β_i which depend on N.

4. Scheme of the endemic model of SEPI

We admit that an infected individual and a precontaminated individual meet on average β(S/N) individuals susceptible to be exposed per unit of time, with $\beta = \beta_p + \beta_i$. We note: β>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), ν>0: the

rate of contagiousness (or of transmission from the precontaged to the infected), λ>0: the rate of infected to die, μ >0: the rate of natural mortality, δ>0: the rate of birth.

The endemic model can be schematised as in the figure (2) below:

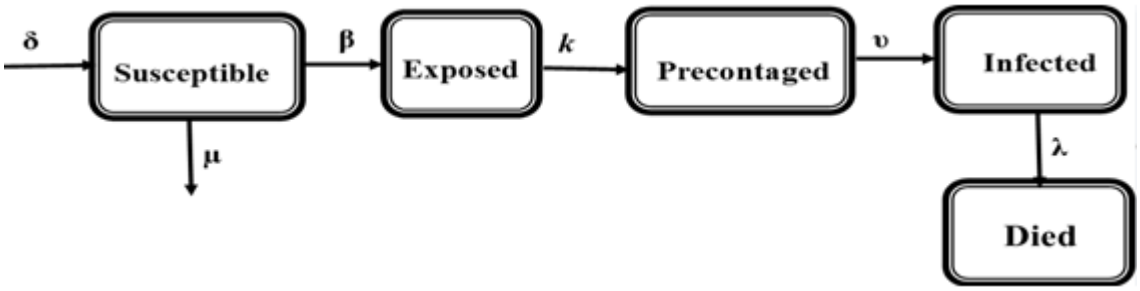

Figure 2: Scheme of the endemic model of SEPI

5. Differential equation representation of the SEPI endemic model

According to the hypothesis (A7) in section (3) above, we consider that during the time interval *dt*, the Susceptible comparison has increased in number δN of new born. 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 $\frac{\beta_i}{N}\boldsymbol{SI}$. And the new cases reached by the precontagion during the interval of time *dt* which are equal to $\frac{\beta_p}{N}\boldsymbol{SP}$. We obtain the new cases exposed to the disease during the time interval *dt* which will be equal to $\frac{\beta_p}{N}\boldsymbol{SP} + \frac{\beta_i}{N}\boldsymbol{SI} = \frac{S}{N}(\beta_p \boldsymbol{P} + \beta_i \boldsymbol{I})$. According to hypothesis (A5), we consider that during the time interval *dt* the compartment Precontaged by the disease has increased in number *kE* of individuals and at the same time, it loses the number *vP* of sick or infected individuals. According to the hypothesis (A5) and (A7), we consider that during the time interval *dt* the compartment Infected has increased in number *vP* of precontaged individuals, and, at the same time, it loses the number *λI* of the epidemic death. It represents in the form of the following system of differential equations (1):

$$\begin{cases} \frac{dS(t)}{dt} = \delta N - \mu S(t) - \frac{\boldsymbol{S}}{\boldsymbol{N}}\left(\beta_p \boldsymbol{P}(\boldsymbol{t}) + \beta_i \boldsymbol{I}(\boldsymbol{t})\right) \\ \frac{dE(t)}{dt} = \frac{\boldsymbol{S}}{\boldsymbol{N}}\left(\beta_p \boldsymbol{P}(\boldsymbol{t}) + \beta_i \boldsymbol{I}(\boldsymbol{t})\right) - kE(t) \\ \frac{dP(t)}{dt} = kE(t) - \nu P(t) \quad (1) \\ \frac{dI(t)}{dt} = \nu P(t) - \lambda I(t) \\ \frac{dD(t)}{dt} = \lambda I(t) \end{cases}$$

There is a unique solution for the model (***1***), under the initial conditions: $S(0)=S_0$, $E(0)=E_0$, $P(0)=P_0$, $I(0)=I_0$in particular, in the region $\Omega = \{(S,E,P,I), S > 0,\ P > 0, I > 0\}$which is positively invariantfor 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* **x** *is the solution of the system* $X' = F(X)$*(with* F *of class*C^∞*) verifying* x(0) = x_0, *then*$\forall$t ≥ 0, x(t)∈G**).**

6. Simulation of the SEPI endemic model

The different curves, 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, β_p= 0.2,β_i= 0.1, k=0.4, ν= 0.2, μ = 0.2 et λ=0.3. We consider a period t depends on the unit of transmission rates, and it is equivalent to day or week or month. By doing the simulation, we obtain the following curves:

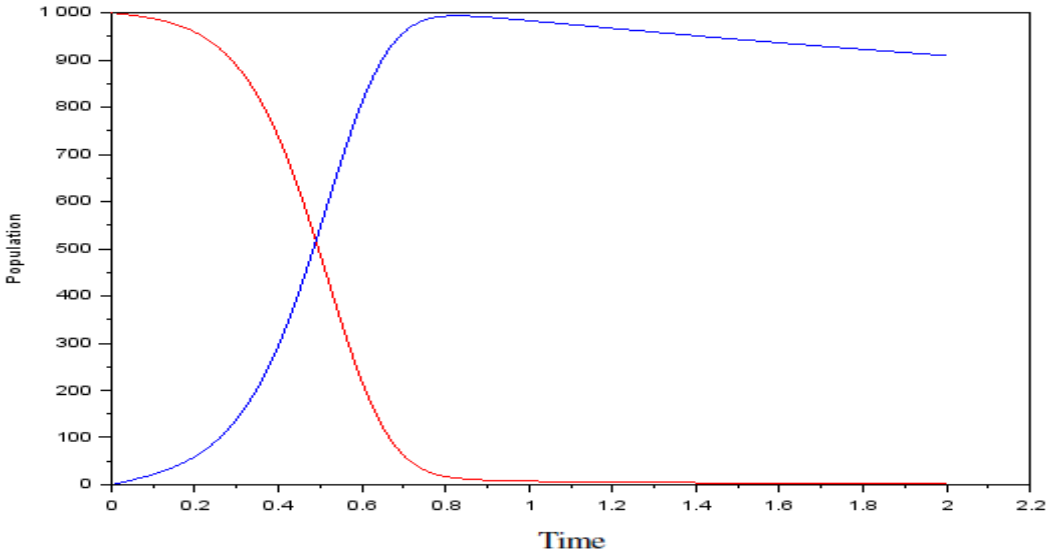

Figure 3: Curves of S(t) coloured in red and E(t) in blue

Interpretation:

From figure (3), we have noted that even with low precontagion and infection rates, the epidemic is spreading with a phenomenal and very rapid speed. Even with a birth rate ($\delta = 0.3$) that we consider to be the average population rate, any susceptible population is already exposed after only 2^etime period *t*

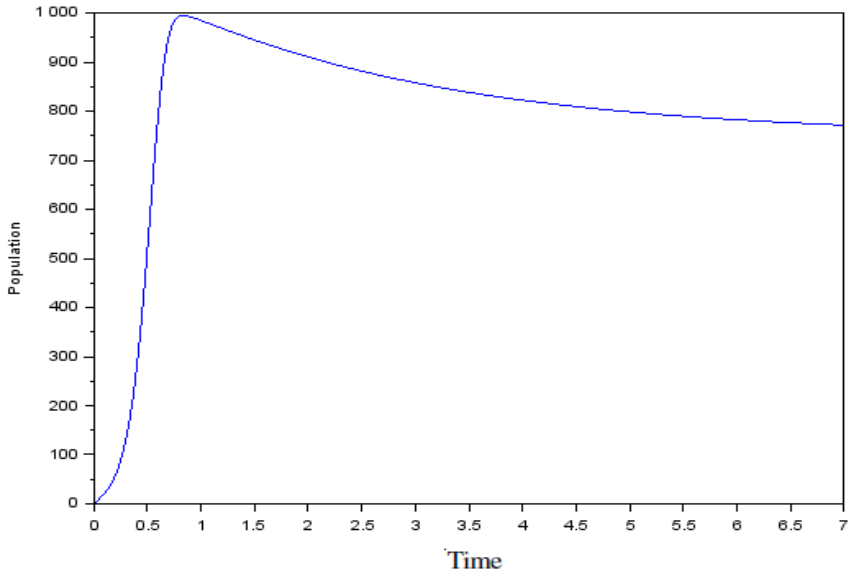

Figure 4: Curve of E(t) during a phase of the epidemic

Interpretation:

According to figure (4), after the phenomenal evolution of the epidemic, the curve of the Exposure decreases, and stablilises and becomes endemic after the 7^eperiod of time *t* of the epidemic.

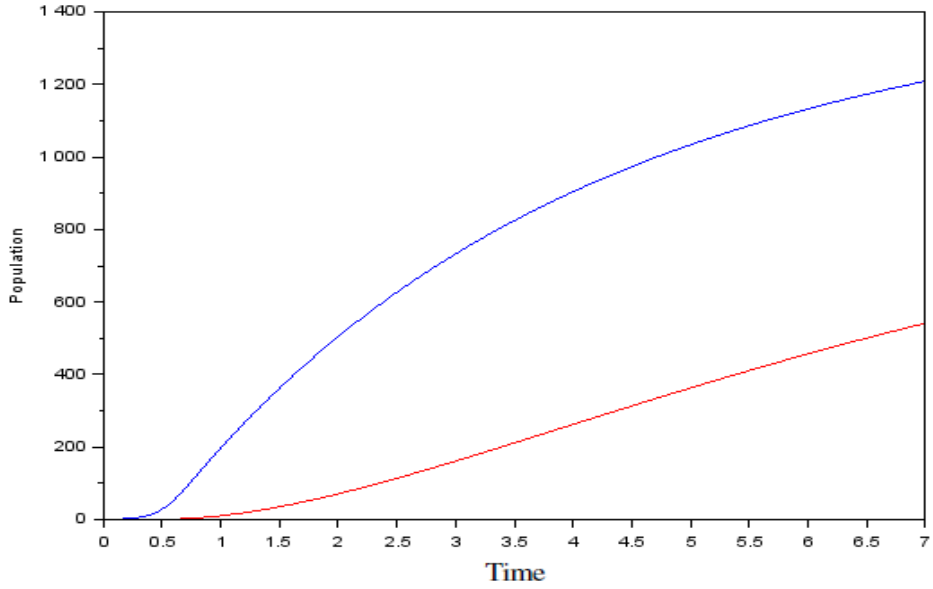

Figure 5: Curves of I(t) coloured in red and P(t) in blue

Interpretation:

From Figure (5), it appears that even after the 7^{e}period of time *t*, the curves for the Precontaged and the Infected are still increasing and are signs of a pandemic.

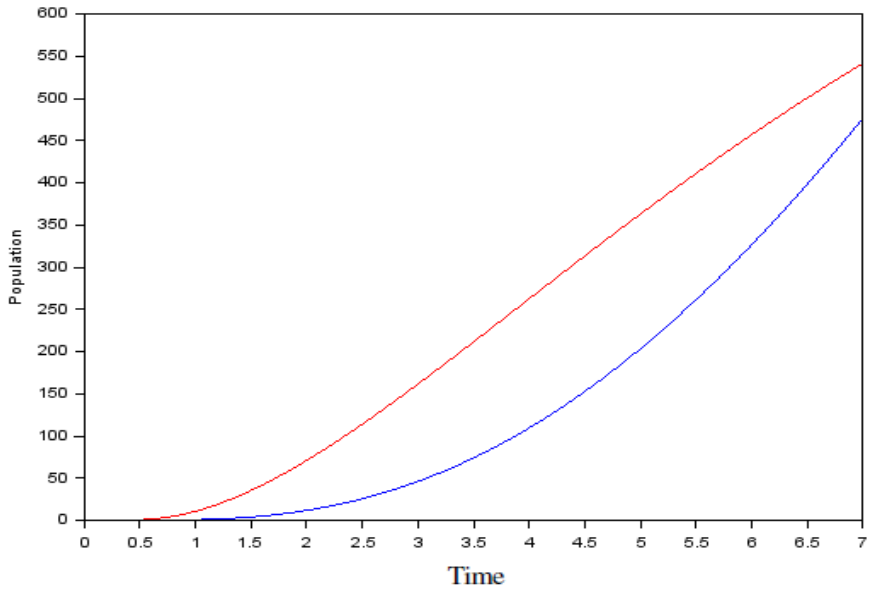

Figure 6: Curves of I(t) coloured in red and D(t) in blue

Interpretation

According to figure (6), parallel to the situation of the infected, the curve of D(t) also increases and we record the death of 450 individuals related to this disease in 7^{e}time period *t* only.

7. Study of the equilibrium point of the SEPI endemic model

According to the work of Lyapunov in (J.M.M.ONDO (2012)), we define 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* $\mathbb{R}$*, not bounded on the right. Let the two equations below (2) and (3):*

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

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

where the functions f: U→R^n *for the system (2) and* f: I × U→R^n*for the system (3) sare assumed to becontinuous. 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 ∈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*and* $\Omega = \{(S,E,P,I),\ S > 0, P > 0, I > 0\}$*, admits a unique solution (S,E,P,I) defined on*[0,+∞[.

Proof:

We calculate the equilibrium points in the absence of infection and/or precontagion. The equilibrium point ofthe model (1) satisfies:

$$\begin{cases} \delta N - \mu S - \frac{S}{N}(\beta_p P + \beta_i I) = 0 \\ \frac{S}{N}(\beta_p P + \beta_i I) - kE = 0 \\ kE - \nu P = 0 \\ \nu P - \lambda I = 0 \\ \lambda I = 0 \end{cases} \quad (4)$$

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

Proposition 2: *Let N>0, in the absence of the infection I=0 and the pre-contagion P=0, then the system (1)admits the equilibrium point:* $E_0 = \left(\frac{\delta N}{\mu}, 0, 0, 0\right)^T$.

Proof:
By replacing I=0 and P=0 in the first, second and third equations in the system (4), we obtain the first equilibrium point: $E_0 = (\hat{S}, \hat{E}, \hat{P}, \hat{I})$:

$$E_0 = \left(\frac{\delta N}{\mu}, 0, 0, 0\right)^T \quad (5)$$

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

Proposition 3. *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* $E_p^* = \left(\frac{\nu N}{\beta_p}, \frac{\delta N}{k} - \frac{\mu \nu N}{\beta_p k}, \frac{\delta N}{\nu} - \frac{\mu N}{\beta_p}, 0\right)^T$;
- *moreover, for all t>0 we have* $\delta\beta_p > \nu\mu$.

Proof:
By replacing I=0 in the third equation of the system (4), implies: $E^* = \frac{\nu P}{k}$**.**
By replacing E^*in the first equation of (4) with $P \neq 0$, we obtain: $S^* = \frac{\nu N}{\beta_p}$.
Replacing S^*in the first equation of (4), we obtain $\boldsymbol{P}^* = \frac{\delta N}{\nu} - \frac{\mu N}{\beta_p}$.
By replacing $\boldsymbol{P}^*$in $\boldsymbol{E}^*$, we then have the equilibrium point $E_p^* = (S^*, \boldsymbol{E}^*, \boldsymbol{P}^*, \boldsymbol{0})$as follows:

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

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

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

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

According to (7),(8),(9), we then have $\delta\beta_p > \nu\mu$**.**

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

Proposition 4. *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*
$E_p^\bullet = \left(\frac{\nu\lambda N}{\lambda\beta_p + \beta_i\nu}, \frac{\delta N}{k} - \frac{\mu\nu\lambda N}{k(\lambda\beta_p + \beta_i\nu)}, \frac{\delta N}{\nu} - \frac{\mu\lambda N}{\lambda\beta_p + \beta_i\nu}, \frac{\delta N}{\lambda} - \frac{\mu\nu N}{\lambda\beta_p + \beta_i\nu}\right)^T$;
- *moreover, for all t>0 we have* $\delta(\lambda\beta_p + \beta_i\nu) > \mu\nu\lambda$.

Proof:

If $P \neq 0$and $I \neq 0$, the system (4) becomes:

$$\begin{cases} \delta N - \mu S - \frac{S}{N}(\beta_p P + \beta_i I) = 0 \\ \frac{S}{N}(\beta_p P + \beta_i I) - kE = 0 \\ kE - \nu P = 0 \\ \nu P - \lambda I = 0 \\ \lambda I \neq 0 \end{cases} \quad (10)$$

The third and fourth equations of the system (10), imply: $I^\bullet = \frac{\nu P}{\lambda}$and $E^\bullet = \frac{\nu P}{k}$.

By replacing $I^\bullet$and $E^\bullet$in the second equation of the system (10), we obtain:

$$S^\bullet = \frac{\nu\lambda N}{\lambda\beta_p + \beta_i\nu}. \quad (11)$$

By replacing $S^\bullet$and $I^\bullet$in the first equation of the system (10), we obtain:

$$P^\bullet = \frac{\delta N(\lambda\beta_p + \beta_i\nu) - \mu\nu\lambda N}{\nu(\lambda\beta_p + \beta_i\nu)} = \frac{\delta N}{\nu} - \frac{\mu\lambda N}{\lambda\beta_p + \beta_i\nu}. \quad (12)$$

Replacing $P^\bullet$in $E^\bullet$and $I^\bullet$, we then have the endemic equilibrium point $E_p^\bullet = (S^\bullet, E^\bullet, P^\bullet, I^\bullet)$as follows:

$$E_p^\bullet = \left(\frac{\nu\lambda N}{\lambda\beta_p + \beta_i\nu}, \frac{\delta N}{k} - \frac{\mu\nu\lambda N}{k(\lambda\beta_p + \beta_i\nu)}, \frac{\delta N}{\nu} - \frac{\mu\lambda N}{\lambda\beta_p + \beta_i\nu}, \frac{\delta N}{\lambda} - \frac{\mu\nu N}{\lambda\beta_p + \beta_i\nu}\right)^T \quad (13)$$

Note that $\hat{S} > S^\bullet$: $\frac{\delta N}{\mu} > \frac{\nu\lambda N}{\lambda\beta_p + \beta_i\nu}$. (14)

So, we have: $\delta(\lambda\beta_p + \beta_i\nu) > \mu\nu\lambda$. (15)

From (15), we deduce that: $E^\bullet > 0, P^\bullet > 0$and $I^\bullet$.

8. The basic reproduction number R0of the SEPI endemic model

To find the $R_0$of the SEPI endemic model, we apply the study condition in the work (L.Chahrazed (2002))on R_0:

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

$$J\left(\frac{\delta N}{\mu},0,0,0\right)=\begin{bmatrix}-\mu & 0 & -\frac{\delta\beta_p}{\mu} & -\frac{\delta\beta_i}{\mu}\\ 0 & -k & \frac{\delta\beta_p}{\mu} & \frac{\delta\beta_i}{\mu}\\ 0 & k & -\nu & 0\\ 0 & 0 & \nu & -\lambda\end{bmatrix} \quad (16)$$

The characteristic equation of the system (1) in the neighbourhood of the equilibrium point $E_0$is given by:

$$det(J-\theta I)=\begin{bmatrix}-\mu-\theta & 0 & -\frac{\delta\beta_p}{\mu} & -\frac{\delta\beta_i c}{\mu}\\ 0 & -k-\theta & \frac{\delta\beta_p}{\mu} & \frac{\delta\beta_i}{\mu}\\ 0 & k & -\nu-\theta & 0\\ 0 & 0 & \nu & -\lambda-\theta\end{bmatrix}=0$$

It comes:

$$det(J-\theta I)=-(\mu+\theta)\left(-\theta^3-\theta^2(k+\nu+\lambda)-\theta(\nu k+k\lambda+\nu\lambda-\delta\beta pk\mu)+\delta\beta pk\lambda\mu+\delta\beta_i k\nu-\nu k\lambda\right)=0 \quad (17)$$

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

$$-\theta^3-\theta^2(k+\nu+\lambda)-\theta\left(\nu k+k\lambda+\nu\lambda-\frac{\delta\beta_p k}{\mu}\right)+\frac{\delta\beta_p k\lambda}{\mu}+\delta\beta_i k\nu-\nu k\lambda=0 \quad (18)$$

We admit that the equation (18) is the characteristic equation of the sub-matrix J_1:

$$J_1=\begin{bmatrix}-k & \frac{\delta\beta_p}{\mu} & \frac{\delta\beta_i}{\mu}\\ k & -\nu & 0\\ 0 & \nu & -\lambda\end{bmatrix} \quad (19)$$

We have the *trace* (J_1) = -[k + ν+ λ] <0, so:

$$det(J_1)=-k\nu\lambda+\frac{\delta\beta_p k\lambda}{\mu}+\frac{\delta\beta_i k\nu}{\mu} \quad (20)$$

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

According to the Varga and Poincaré-Lyapunov theorem of linearization in (G.Sallet (2010)), $R_0$is defined bythe expression below:

$$R_0=\frac{\nu\lambda\mu}{\delta(\beta_p\lambda+\beta_i\nu)}=\frac{\nu\lambda}{\frac{\delta}{\mu}(\beta_p\lambda+\beta_i\nu)}. \quad (21)$$

9. Study of the stability of the equilibrium point E_0

9.1 Local stability

L.Chahrazed (2002) defines the local stability of E_0as follows:

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.*

Indeed, according to the equations (19), (20) and (15) above:

$$\begin{cases} trace\,(J_1)=-[k+\nu+\lambda]<0;\\ det(J_1)=-k\nu\lambda+\frac{\delta\beta_p k\lambda}{\mu}+\frac{\delta\beta_i k\nu}{\mu}>0. \end{cases} \quad (22)$$

So we see that the conditions in (22) are met. Then the equilibrium point $E_0$of the system (1) is unique. And it remains locally asymptotically stable.

9.2 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 3. *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 4. *We say that $x_e$is an asymptotically stable equilibrium point, if it is a stable equilibrium pointand if the domain of attraction of $x_0$is a neighbourhood of x_0.*

Définition 5. *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 $\lim_{t\to+\infty} d\left(x(t,t_0,x_0,x_e)\right)=0$.*

Lemme 1: *The number of Suspects S in the model (1) admits that:*

$$\limsup_{t\to+\infty} S(t)\leq\frac{\delta N}{\mu}. \quad (23)$$

Proof:

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

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

Suppose that:

$$\dot{Z}=\delta N-\mu S(t). \quad (24)$$

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

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

Solving the equation (24) which is a first order linear ordinary differential equation in time $t>0$with $Z(0)=Z_0$, we obtain:

$$Z(t)=Z_0e^{-\mu t}+\frac{\delta N}{\mu}(1-e^{-\mu t}).$$

By determining the limit of *Z(t)*when *t* goes to infinity, we obtain:

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

Therefore

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

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

Proof:

Using (23) for $\eta>0$then there exists $T_1>0$such that $S(t) \leq \frac{\delta N}{\mu}+\eta$, for $t > T_1$. According to the system(1), we obtain:

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

Then for $t > T_1$, we pose:

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

We obtain the matrix D defined in the following way:

$$D = \begin{bmatrix} -k & \beta_p\left(\frac{\delta}{\mu}+\frac{\eta}{N}\right) & \beta_i\left(\frac{\delta}{\mu}+\frac{\eta}{N}\right) \\ k & -\nu & 0 \\ 0 & \nu & -\lambda \end{bmatrix} \quad (25)$$

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

And for the determinant, we have: $det(D) = -k\nu\lambda + \beta_p k\lambda\left(\frac{\delta}{\mu}+\frac{\eta}{N}\right) + \beta_i k\nu\left(\frac{\delta}{\mu}+\frac{\eta}{N}\right)$.

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

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

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

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)$\lim_{t\to+\infty} I(t) = 0$.

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

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

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

$$P(t) < \theta, I(t) < \sigma \text{ and } S(t) \leq \frac{\delta N}{\mu}+\eta \quad (26)$$

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

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

We consider:

$$\dot{V}(t) + \mu V(t) = \delta N - \beta_p\left(\frac{\delta}{\mu}+\frac{\eta}{N}\right)\theta - \beta_i\left(\frac{\delta}{\mu}+\frac{\eta}{N}\right)\sigma;$$
$$V(T_3) = (V)_0.$$

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

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

We pose $\eta_1 = -\beta_p\left(\frac{\delta}{\mu}+\frac{\eta}{N}\right)\theta - \beta_i\left(\frac{\delta}{\mu}+\frac{\eta}{N}\right)\sigma$.

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

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

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

This means that$\lim_{t\to+\infty} inf\ S(t) \geq \frac{\delta N+\eta_1}{\mu}$. (27)

From (26)and (27)if we chooseη_1, $\theta$and σvery small and $t>T_4>T_3$, then we get:

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

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

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

10. Local stability of the equilibrium E^*

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

Theorem 2: *If 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 5. *The epidemic is locally asymptotically stable if and only if all the eigen values of the Jacobian matrix J have a negative real part.*

Indeed, J is defined by:

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

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

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

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

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

The coefficients are:

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

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

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

$D = k\nu\lambda\left(\mu + \frac{1}{N}(\beta_p \boldsymbol{P}^* + \beta_i \boldsymbol{I}^*)\right) + \frac{\boldsymbol{k}\boldsymbol{S}^*}{\boldsymbol{N}^2}(\beta_p \boldsymbol{P}^* + \beta_i \boldsymbol{I}^*)(\beta_p \boldsymbol{\lambda} + \boldsymbol{\beta i \nu})$.

We have $A > 0$ et $B, C, D > 0$.

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

We calculate $AB - C$, we then obtain:

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

Therefore $\boldsymbol{E}^*$is locally asymptotically stable.

11. Conclusion

We were first interested in the formulation of an endemic model describing the dynamic behaviour of the transmission of mutant microorganisms on the population. The study proposed in this model, as well as the different numerical simulations, allow us to know the moment when control efforts must be applied, as well as their intensity in the face of emerging infectious diseases. The SEPI endemic model described above is a deterministic model.

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