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Spiking systems in population-infection dynamics

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***Abstract*— Motivated by a class of models in population dynamics, we introduce the concept of spiking dynamical systems. A *spiking system* admits an asymptotically stable equilibrium but, under proper perturbations on the initial conditions in a compact region including the equilibrium, its output exhibits a spike of arbitrarily large magnitude before the state returns within the region. We consider a model that describes a well-documented phenomenon in caterpillar-virus dynamics: a sudden increase of the caterpillar population occurs, due to a temporary reduction of the viral population, and is then followed by a sudden decrease. We prove that the caterpillar-virus system is spiking according to our proposed mathematical definition: the model can yield arbitrarily large population densities for caterpillars, and then the original conditions are suddenly restored. When the model also takes into account environmental constraints that keep the caterpillar population bounded, the spike cannot be arbitrarily large, but the population density can get arbitrarily close to the maximal one that can be achieved in the absence of virus.**

***Index Terms*— Ecological systems, Infection dynamics, Population dynamics, Spiking systems.**

I. Introduction and Motivation

SPIKING phenomena are well documented in nature and widely discussed in the ecological literature [2], [18]. Such phenomena are often referred to as *outbreaks*, a term that typically denotes the sudden increase in the number of individuals in a population, possibly – but not necessarily – followed by a rapid decay due to some limiting factor. Outbreaks related to the sudden increase in the number of infected individuals, in a population exposed to an infectious agent, are captured by epidemic models [5], [8]; in epidemiology, a disease outbreak is defined as the sudden occurrence of infection cases in excess of what would normally be expected in a given community, location and season, during a specific period of time [21].

We denote in general by *outbreak* the sudden growth of a population, and specifically by *spike* an outbreak followed by a sudden restoration of the original system conditions.

In ecology and population dynamics, one of the well-known mechanisms that induce a population spike is the presence of a predator or a pathogen that affects the population and becomes particularly active after the outbreak, thus suppressing most of the population and restoring it to its previous size [11], [12]. An example is offered by the interaction between some insect populations, including the well-studied case of the western tent caterpillar, and viruses that cause high-mortality infections in those populations; this case study has been thoroughly investigated in ecology [3], [6], [7], [10], [18], [19], and has also inspired several mathematical modelling studies [9]–[12], [14], [17], [22]. The spiking behaviour occurs because, while viral circulation is extremely limited when the caterpillar population density is low, the virus spreads very quickly and effectively when the population density becomes high, and the high mortality associated with the infection causes an ensuing drastic reduction in the number of caterpillars.

The Lotka-Volterra predator-prey model [16], [20]

$$\dot{x}_1(t) = \alpha x_1(t) - \beta x_1(t) x_2(t) \quad (1)$$

$$\dot{x}_2(t) = -\gamma x_2(t) + \delta x_1(t) x_2(t) \quad (2)$$

where x_1 is the population density of prey and x_2 of predator, is known to produce periodic solutions, with recurrent peaks of both prey and predator densities. Regular fluctuations have been observed in real ecosystems, such as in the case of lynx and snowshoe hare population dynamics [13]. However, many ecological systems – including the insect-virus case, and in particular the caterpillar-virus case – exhibit instead only occasional peaks, depending on unpredictable circumstances: which is the mechanism that can generate these?

Here, we address this question by considering a variation of the predator-prey model (1)-(2), in which the interaction terms $\beta x_1 x_2$ and $\delta x_1 x_2$ among the two species are replaced by the new terms $\beta x_1^p x_2$ and $\delta x_1^p x_2$, with $p > 1$. In fact, while in the predator-prey model the interaction terms are proportional to the likelihood of a prey encountering a predator (approximated by the product of the two population densities), the situation is different in the caterpillar-virus case. Once infected by a virus, a caterpillar dies within a very short time-span, due to the high mortality of the infection. Still, the virus population can proliferate only when non-infected caterpillars encounter infected ones, which depends more than linearly on the caterpillar density [9], [10]. Therefore, we assume that the dependence of the interaction terms on the population density of caterpillars is not linear, but polynomial of degree $p > 1$, to capture the evidence that viral transmission is very low for small caterpillar densities (when an infected caterpillar is unlikely to encounter a non-infected one and spread the contagion), while it increases significantly, much more than linearly, when the caterpillar density becomes higher.

The contributions of this manuscript are summarised below.

- We introduce the new notion and mathematical definition of *spiking system*. We define as *spiking* a system that admits an asymptotically stable equilibrium but that, under

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proper perturbations on the initial conditions, exhibits a spike of arbitrarily large magnitude, before reverting back to its initial configuration (Section II).

- To showcase spiking dynamics, we consider a simple variation of the Lotka-Volterra predator-prey model that captures widely observed and meaningful caterpillar-virus dynamics by introducing a polynomial dependence of the interaction term on the population density of the caterpillars (Section III), and we prove that the considered model is spiking according to our definition (Section IV).
- We also consider a modification of the model that takes into account environmental constraints, such as finite resources, that keep the population bounded. In this case, the system is not *spiking*, since the spike cannot have an arbitrarily large magnitude, but the population density can get arbitrarily close to the maximal population size in a virus-free setting (Section V). This behaviour is similar to that of *excitable systems* [1], [15], for which a small perturbation can cause "large" transients before the trajectory returns to the equilibrium.

Section VI illustrates our results on spiking systems through numerical simulations of the caterpillar-virus model.

II. Spiking Systems: the Concept

Consider the ordinary-differential-equation system

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

without external inputs, where $x \in \mathbb{R}^n$ and $y \in \mathbb{R}$. The output $y(t)$ linearly depends on the state just for simplicity, but the theory could be generalised to the case of outputs that are suitable nonlinear functions of the state.

Assumption 1. *The function $f\colon \mathbb{R}^n \to \mathbb{R}^n$ in* (3) *is locally Lipschitz. Also, for all initial conditions, the solution to* (3) *exists and depends continuously on the initial conditions.*

A *spiking* system of the form (3) admits an equilibrium point $\bar{x}$ (such that $f(\bar{x}) = 0$) that is locally asymptotically stable and lies within the interior of a compact set such that, for some initial conditions within the set, the output becomes arbitrarily large, but then the state returns within the set.

Definition 1. *System* (3) *is* spiking *if*
a) it admits a locally asymptotically stable equilibrium $\bar{x}$;
b) there exists a compact set $\mathcal{W}$ that includes $\bar{x}$ as an interior point and has the property that, for all $\mu > 0$ (no matter how large), there exist an initial condition $x_0 \in$ int$(\mathcal{W})$, a time t_1 and a time $t_2 > t_1$ such that, for $x(0) = x_0$, we have $y(t_1) > \mu$ and $x(t_2) \in$ int$(\mathcal{W})$.

The notion of *spiking system* is similar to that of *excitable system* [1], [15], but also crucially different. According to [1], an excitable system has a globally attractive equilibrium and, if the initial condition is perturbed beyond a *threshold*, then a "large" transient can occur, even though the system eventually returns to the equilibrium; the concept is associated with singular perturbations. According to [15, Chapter 7], a system with a stable equilibrium is excitable if there is a "large-amplitude" trajectory that starts in a small neighbourhood of the equilibrium, leaves the neighbourhood, and then returns to the equilibrium, and the mathematical characterisation of the property requires the system to be "near a bifurcation from resting to oscillatory dynamics". Our definition does not consider any threshold, nor does it involve singular perturbations or bifurcation phenomena. Furthermore, global attractivity does not hold in our case: according to our definition, the "spike" can be arbitrary large, which essentially requires the existence of an *unbounded solution* $x_U(t)$ to the system that originates on the boundary of $\mathcal{W}$ and is unbounded (and yields an unbounded $y_U(t)$), while any other solution starting from int$(\mathcal{W})$, arbitrarily close to the unbounded solution $x_U(t)$, eventually detaches from it and returns within int$(\mathcal{W})$.

III. Caterpillar-Virus Population Dynamics

We consider the population dynamics involving western tent caterpillars (Lepidoptera-Lasiocampidae *Malacosoma californicum*), parasites of trees, and some insect baculoviruses (nucleopolyhedrovirus), widely studied in the ecological literature [3], [6], [7], [10], [18], [19]. Denoting by c the population density of caterpillars and by v the population density of virus, we propose the equations

$$\dot{c}(t) = \alpha c(t) - \beta v(t) c(t)^p \tag{4}$$

$$\dot{v}(t) = -\gamma v(t) + \delta v(t) c(t)^p \tag{5}$$

that capture the growth rate of caterpillars in isolation, associated with $\alpha > 0$, the decay of virus, associated with $\gamma > 0$, the death rate of caterpillars induced by viral infection, associated with $\beta > 0$, and the replication of virus within infected hosts, associated with $\delta > 0$. As the system output, we choose the population density of caterpillars: $y(t) = c(t)$.

For $p = 1$, the model (4)-(5) reduces to the predator-prey model (1)-(2), which is well known to admit periodic orbits for any positive initial condition. Here, we assume that $p > 1$.

In fact, for a fixed $\bar{v}$, the infection term

$$\beta \bar{v} c^p$$

would be proportional to the population density c of caterpillars for $p = 1$, while assuming $p > 1$ allows us to take into account the following specific features of the caterpillar-virus population dynamics (see Fig. 1):

- For a small population density c, the caterpillars are spread out in their habitat and, given the average distance between two individuals, virtually no contagion is possible. Therefore, an infected caterpillar typically dies before having had the opportunity to infect others.
- For a large population density c, the caterpillars are very close to one another, and hence the contagion grows much faster than linearly with their population density: since the average distance between individuals is very small, each infected caterpillar ends up infecting many others.

Various models could describe caterpillar-virus dynamics; here we aim to provide a significant example of spiking system in biology that is as simple as possible. Our model (4)-(5) is intrinsically different from SIR-like epidemiological models involving susceptible, infected, recovered individuals [5], [8], since it models the population dynamics of both caterpillars

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and virus, and it does not distinguish between different stages of the disease for caterpillars. The model also differs from that suggested by Dwyer in [9], [10], which considers three differential equations that model the time evolution of the population densities of susceptible caterpillars (S), infected caterpillars (I), and pathogens (P). It is also different from the discrete-time model introduced by Dwyer in [11], [12], which involves two difference equations where the key function representing infection is given by an implicit expression.

IV. The Caterpillar-Virus System is Spiking

The system (4)-(5) admits an *unbounded solution*. In fact, if the initial condition is $c(0) > 0$ and $v(0) = 0$, then $v(t) \equiv 0$ while $c(t) = c(0)e^{\alpha t}$.

To prove that system (4)-(5) is spiking, we need to show that it meets both requirements in Definition 1. First, we show that it admits a locally asymptotically stable equilibrium point (which is the only nontrivial equilibrium).

Proposition 1. *System* (4)-(5)*, for $p > 1$, admits a single positive equilibrium, which is locally asymptotically stable.*

Proof. From equation (5), the only positive equilibrium value for c is $\bar{c} = \left(\frac{\gamma}{\delta}\right)^{\frac{1}{p}}$. Substituting in (4) allows us to derive the corresponding unique equilibrium for v, $\bar{v} = \frac{\alpha}{\beta}\left(\frac{\gamma}{\delta}\right)^{\frac{1-p}{p}}$. To prove local asymptotic stability of the equilibrium, we show that the corresponding linearisation is Hurwitz. The system Jacobian at the equilibrium is

$$J_{(\bar{c},\bar{v})} = \begin{bmatrix} \alpha - p\beta v c^{p-1} & -\beta c^p \\ p\delta v c^{p-1} & -\gamma + \delta c^p \end{bmatrix}\Bigg|_{(\bar{c},\bar{v})} = \begin{bmatrix} \alpha(1-p) & -\beta\frac{\gamma}{\delta} \\ p\delta\frac{\alpha}{\beta} & 0 \end{bmatrix}.$$

For $p > 1$, the characteristic polynomial

$$\psi(\lambda) = \det[\lambda I - J_{(\bar{c},\bar{v})}] = \lambda^2 + \alpha(p-1)\lambda + p\alpha\gamma$$

has positive coefficients, and the equilibrium is therefore locally asymptotically stable. □

Then, we show that condition *b)* in Definition 1 is satisfied.

Proposition 2. *Consider any set of the form*

$$\mathcal{W} = \{(c, v) \colon 0 \le c \le a, \quad 0 \le v \le b\},$$

with $a > \bar{c}$ and $b > \bar{v}$ chosen so that the equilibrium $P = (\bar{c}, \bar{v})$ of system (4)-(5) *is in the interior of $\mathcal{W}$. For all $\mu > 0$, there exists an initial condition $(\bar{c}, \epsilon)$ (on the segment P-Q in Fig. 2, with $Q = (\bar{c}, 0)$) such that $c(t_1) > \mu$ for some t_1 and $c(t_2) = \bar{c}$ for some $t_2 > t_1$, with $0 < v(t_2) \le \bar{v}$ (namely, the state will return on the segment P-Q in Fig. 2).*

To prove the proposition, we need a preliminary lemma.

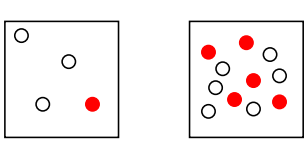

Fig. 1. Small and large population density of caterpillars: contagion is almost absent at small density (left), while it grows faster than linearly at large density (right).

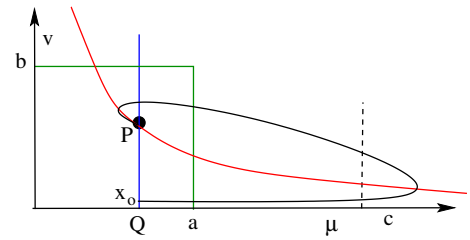

Fig. 2. Any trajectory of system (4)-(5) originating on the segment $\boldsymbol{P}$-$\boldsymbol{Q}$ will eventually return to the segment $\boldsymbol{P}$-$\boldsymbol{Q}$ (and possibly to $\boldsymbol{P}$), where the equilibrium point $\boldsymbol{P} = (\bar{c}, \bar{v})$ is the intersection of the nullclines $\dot{c} = \alpha c - \beta v c^p = 0$ (red) and $\dot{v} = -\gamma v + \delta c^p v = 0$ (blue), while $\boldsymbol{Q} = (\bar{c}, 0)$. The green segments delimit the set $\mathcal{W} = \{(c, v) \colon 0 \le c \le a, \ 0 \le v \le b\}$.

Lemma 1. *Consider a compact set $\mathcal{C}$ with non-empty interior. Assume that any solution $x(t)$ of* (3) *has the property that each of the derivatives $\dot{x}_i(t)$ has the same sign for all t when $x \in \mathcal{C}$. Then, for $x(0) \in \mathcal{C}$, the trajectory $x(t)$ either leaves the set or converges to an equilibrium.*

Proof. Assume that $x(t)$ never leaves $\mathcal{C}$. Since each derivative $\dot{x}_i(t)$ has the same sign for all t as long as $x \in \mathcal{C}$, each component $x_i(t)$ is monotonic, hence it converges to a finite limit. Then $x(t)$ has a limit $\hat{x} \in \mathcal{C}$. Therefore, it must be $f(\hat{x}) = 0$, otherwise $\hat{x}$ could not be the limit, and hence $\hat{x}$ is an equilibrium. □

Proof of Proposition 2. The proof is geometric and we illustrate it with the support of Fig. 3. We show that any solution originating on the segment P-Q rotates counter-clockwise around the equilibrium point P. To this aim, we consider four regions of the positive orthant, delimited by the nullclines.

Region 1 (cyan in Fig. 3): the derivatives have sign $[+, +]$. We show that the solution originating on the segment P-Q intersects the nullcline $\dot{c} = 0$ (red in Fig. 3) at a point U. In the (cyan) compact set delimited by the horizontal segment x_0-L (with L belonging to the nullcline), the nullcline between L and P and the vertical segment P-x_0, since both derivatives are increasing, the solution cannot reach the only equilibrium P and thus it must leave this compact set, in view of Lemma

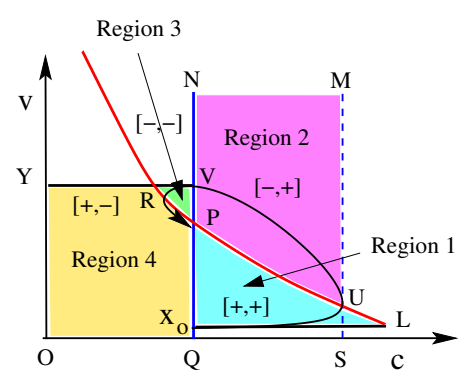

Fig. 3. Any trajectory of system (4)-(5) originating on segment $\boldsymbol{P}$-$\boldsymbol{Q}$ eventually comes back to the segment $\boldsymbol{P}$-$\boldsymbol{Q}$ (and possibly to the equilibrium $\boldsymbol{P}$ itself). The nullclines ($\dot{v} = 0$, in blue, and $\dot{c} = 0$, in red) intersect at the equilibrium $\boldsymbol{P}$ and partition the positive orthant into four regions. The signs of the derivatives of c and v in each region are reported in square brackets.

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1, crossing the nullcline at a point U between P and L.

Region 2 (magenta in Fig. 3): the derivatives have sign $[-,+]$. Consider the (magenta) non-compact set delimited by the vertical half-line P-N, the nullcline $\dot{c}=0$ (red) between P and U and the vertical half-line U-M. Since the set is *not compact*, we cannot directly apply Lemma 1. However, $x(t)$ has monotonic components and thus either it has a limit (possibly infinite) within the region, or it leaves the region.

If it leaves the region, then it must cross the half-line P-N, because within the region $\dot{c}(t)<0$ and $\dot{v}$ is positive along the corresponding portion of the nullcline $\dot{c}=0$, hence the trajectories point vertically upwards and the nullcline cannot be reached from above.

Now we need to rule out the case of solutions that remain in this non-compact set with $v(t)\to+\infty$. This cannot happen because, within this region, the value of c is lower and upper bounded as $\bar{c}\le c\le\tilde{c}$, where $\tilde{c}$ is the value of c at the crossing point U. Hence $\dot{c}=\ \alpha c-\beta vc^p\le\ \alpha\tilde{c}-\beta v\bar{c}^p$. So, for large values of v, the negative derivative of c becomes arbitrarily large in absolute value (it converges to $-\infty$), and hence the solution must cross the half line P-N at a finite point V.

Region 3 (green in Fig. 3): the derivatives have sign $[-,-]$. Consider the (green) compact set delimited by the vertical segment P-V, the horizontal segment V-R and the nullcline $\dot{c}=0$ (red) between R and P. By Lemma 1, the solution either leaves the set, or reaches P. Since both derivatives are negative, the solution must leave the set and cross the nullcline between R and P.

Region 4 (yellow in Fig. 3): the derivatives have sign $[+,-]$. Consider the (yellow) compact set delimited by the nullcline $\dot{c}=0$ (red) between P and R and the segments R-Y, Y-O, O-Q and Q-P, where O is the origin and Y lies on the v-axis. By Lemma 1, the solution either converges to the equilibrium P or leaves the set. Since c is increasing, the solution may only leave the set by crossing the nullcline between R and P or the segment P-Q. However, the solution cannot reach the nullcline between R and P, because on this curve we have $\dot{v}<0$ and $\dot{c}=0$, and hence the derivative of the solution points downwards. Then, the solution must reach again the segment P-Q (and possibly the equilibrium P itself).

To complete the proof, we show that $c(t)$ can become arbitrarily large. This is a consequence of the continuous dependence of the solution on the initial conditions. The solution starting from point Q, i.e. $c(0)=\bar{c}$ and $v(0)=0$, is $c(t)=\bar{c}e^{\alpha t}$ and $v(t)\equiv 0$. Given a large $\mu>0$, consider the time instant $\hat{t}>0$ at which $c(\hat{t})=2\mu$, namely $\hat{t}=\log(2\mu/\bar{c})/\alpha$. In view of the continuous dependence of the solution on the initial condition, we can take an initial condition x_0 on the segment P-Q such that $c(0)=\bar{c}$ and $v(0)=\epsilon$, where $\epsilon>0$ is small and such that the corresponding solution gets arbitrarily close to the point $(c(\hat{t}),0)$, hence for such a solution we must have $c(\hat{t})>\mu$. □

We have thus proven that system (4)-(5) is spiking according to Definition 1.

Since the system enjoys the property of being spiking for all possible values of its positive parameters, provided that $p>1$, it can be considered *structurally spiking* [4].

V. Finite Spiking with Population Saturation

To take into account environmental constraints that prevent a population from becoming *arbitrarily* large, we consider in (4) the logistic growth term $\alpha c(1-c/d)$, where d is the maximal population density achieved, in the absence of virus, due to environmental limits only:

$$\dot{c}(t) = \alpha c(t)[1-c(t)/d]-\beta v(t)c(t)^p \quad (6)$$

$$\dot{v}(t) = -\gamma v(t)+\delta v(t)c(t)^p \quad (7)$$

The system admits three equilibria: the trivial $(0,0)$, the virus-free equilibrium $D=(d,0)$ and the positive equilibrium $P=(\bar{c},\bar{v})$. From (7) we obtain $\bar{c}=\left(\frac{\gamma}{\delta}\right)^{\frac{1}{p}}$, while substituting in (6) yields $\bar{v}=\frac{\alpha}{\beta}\left(1-\frac{\bar{c}}{d}\right)(\bar{c})^{1-p}$.

We consider $p>1$. We assume that the equilibrium density of caterpillars in the presence of virus is less than the maximum population density d in the absence of virus.

Assumption 2. *For the equilibrium $\bar{c}=\left(\frac{\gamma}{\delta}\right)^{\frac{1}{p}}$, we have $\bar{c}<d$.*

The system Jacobian is

$$J=\begin{bmatrix}\alpha-2\alpha\frac{c}{d}-p\beta vc^{p-1} & -\beta c^p\\ p\delta vc^{p-1} & -\gamma+\delta c^p\end{bmatrix}.$$

Hence, the equilibrium $(0,0)$ is locally unstable (being associated with eigenvalues $\alpha>0$ and $-\gamma$), the equilibrium $D=(d,0)$ is locally unstable (being associated with eigenvalues $-\alpha$ and $-\gamma+\delta d^p>0$ in view of Assumption 2), the equilibrium $P=(\bar{c},\bar{v})$ is locally asymptotically stable because

$$J_P=\begin{bmatrix}-\alpha\frac{\bar{c}}{d}-(p-1)\beta\bar{v}\bar{c}^{p-1} & -\beta\frac{\gamma}{\delta}\\ p\delta\frac{\alpha}{\beta}(1-\frac{\bar{c}}{d}) & 0\end{bmatrix}$$

is sign definite, with $(J_P)_{11}$ negative, $(J_P)_{12}$ negative and $(J_P)_{21}$ positive, and is therefore a Hurwitz matrix.

System (6)-(7) is not spiking according to Definition 1. However, it has quite similar properties, with the exception that its "spikes" are, as expected, bounded by d.

Proposition 3. *System* (6)-(7)*, for $p>1$, admits a single positive equilibrium, which is locally asymptotically stable. All the trajectories with initial condition $0<c(0)\le d$ are bounded.*

Proof. The existence of the positive equilibrium and its local asymptotic stability was proved by direct computation of the equilibrium and of the corresponding Jacobian matrix.

The boundedness of the solutions can again be proved geometrically, with the support of Fig. 4. Consider the trajectory originating in a point $A=(d,\hat{v})$, with large enough $\hat{v}$. In this region delimited by the nullclines, the derivatives have sign $[-,+]$. This trajectory reaches the line $c=\bar{c}$, because $\dot{v}>0$ and hence v increases, becoming larger than $\hat{v}$. For sufficiently large v, $\dot{c}=\alpha c(1-c/d)-\beta vc^p<\alpha d-\beta v\bar{c}^p<0$, since $\bar{c}\le c\le d$. The negative derivative $\dot{c}$ becomes arbitrarily large in absolute value for large enough values of v, and therefore the trajectory must reach the line $c=\bar{c}$ at a finite point B.

Consider the region delimited by the system trajectory from A to B and the segments B-C, C-O, O-D and D-A, highlighted in red in Fig. 4. We can show that this region is positively invariant, since no trajectory originating inside the region can leave it. In fact, no trajectory can cross the curve

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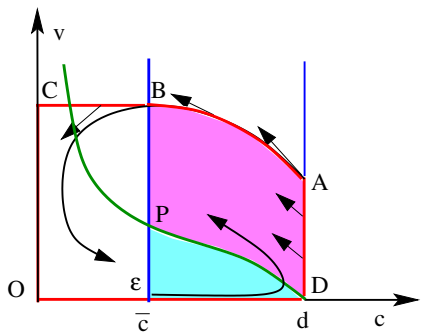

Fig. 4. The trapping region for the trajectories of system (6)-(7) is delimited by the red contour. The nullclines ($\dot{\boldsymbol{v}} = \mathbf{0}$ in blue and $\dot{\boldsymbol{c}} = \mathbf{0}$ in green) intersect at the positive equilibrium $\boldsymbol{P}$. Another equilibrium is $\boldsymbol{D} = (\boldsymbol{d}, \mathbf{0})$.

A-B, since such a curve is a trajectory itself; no trajectory can cross the segment B-C because, along that segment, $\dot{v} \leq 0$; no trajectory can cross the segments C-O or O-D because the system is positive; no trajectory can cross the segment D-A because, along that segment, the derivative $\dot{c}$ is negative, with the exception of point $D = (d, 0)$, which is an equilibrium.

To conclude the proof, we just notice that, since we can take A with an arbitrarily large ordinate $\hat{v}$, in order to accommodate for any initial condition with $0 < c(0) \leq d$, every trajectory is bounded and the corresponding invariant region can be constructed as discussed above. □

The behaviour of system (6)-(7) resembles that of excitable systems [1], [15]. For "small" variations of the initial conditions, the transient is "large": c approaches the upper limit $\bar{c}$, the virus-free equilibrium population.

Proposition 4. *The trajectory of system* (6)-(7) *with initial condition* $c(0) = \bar{c}$ *and* $v(0) = \epsilon$ *gets arbitrarily close to the unstable equilibrium* $D = (d, 0)$*, provided that* ϵ *is small enough, and then comes back to the segment* P-B *(and possibly to the asymptotically stable equilibrium* P*).*

Proof. We prove the result geometrically with the support of Fig. 4. Consider the trajectory originating at $(\bar{c}, \epsilon)$. Within the (cyan) region delimited by the c-axis, the nullcline $\dot{c} = 0$ (green) and the nullcline $\dot{v} = 0$ (blue), the derivatives are $\dot{c} > 0$ and $\dot{v} > 0$, hence the trajectory crosses from below the nullcline $\dot{c} = 0$, which has equation $v = \frac{\alpha(1-c/d)}{\beta c^{p-1}}$. Then, in the (magenta) compact region delimited by the nullcline $\dot{c} = 0$ (green), the segment A-D, the curve from A to B and the segment P-B (blue), we can apply Lemma 1 to conclude that the trajectory must reach the segment P-B (including, possibly, the equilibrium P) because $\dot{c} < 0$ (in fact, the trajectory cannot cross the curve A-B, which is a trajectory itself, nor segment A-D because $\dot{c} < 0$, nor the nullcline $\dot{c} = 0$ where the derivative vector is vertical, pointing upward).

The fact that the trajectory gets arbitrarily close to D follows from its continuous dependence on the initial conditions, since the trajectory converges to D for $\epsilon = 0$. □

VI. Numerical Simulations

We illustrate here the concept of spiking system and our results through numerical simulations.

Consider system (4)-(5) with parameters $\alpha = \beta = 1$, $\gamma = \delta = 0.1$, normalised so that the nontrivial equilibrium at which caterpillars and virus coexist is $\bar{c} = 1$ and $\bar{v} = 1$. Take $p = 2$.

At the initial time, assume we have a sudden decrease in the virus population, so that the initial conditions are $c(0) = 1$ and $v(0) = 0.1$. Then, the time evolution of the system, shown in Fig. 5, exhibits a spike with a peak that is about 3.5 times the equilibrium population density. The spike peak becomes higher when the initial virus population is smaller: for instance, $v(0) = 0.01$ yields a peak that is almost 7 times the equilibrium population density (see Fig. 6). Quite surprisingly, the height of the spike peak is decreasing with p. For instance, when $p = 3$ and $v(0) = 0.1$, the height of the peak, as shown in Fig. 7, is reduced with respect to that in Fig. 5.

The time evolution of the system with $p > 1$ is considerably different from the one achieved when $p = 1$: in the latter case, corresponding to the classic Lotka-Volterra predator-prey system, the trajectories exhibit persistent oscillations associated with a periodic solution, as shown in Fig. 8.

In the case of system (6)-(7), with parameters $\alpha = \beta = 1$, $\gamma = \delta = 0.1$, $d = 10$, the equilibrium at which caterpillars and virus coexist is $\bar{c} = 1$ and $\bar{v} < 1$. As expected, a spiking behaviour occurs, as shown in Fig. 9 for $p = 2$ and in Fig. 10 for $p = 3$, but the spike peak never exceeds the threshold $d = 10$. Again, the height of the spike peak is decreasing not only with the initial condition $v(0)$, but also with p.

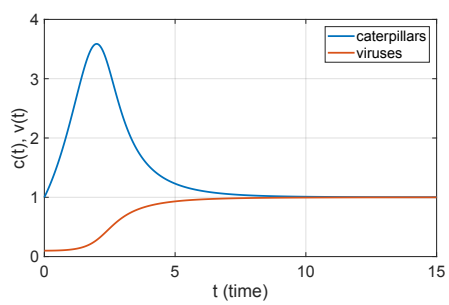

Fig. 5. Time evolution of the spiking system (4)-(5) with $\boldsymbol{p} = \mathbf{2}$ and initial conditions $(\mathbf{1}, \mathbf{0.1})$.

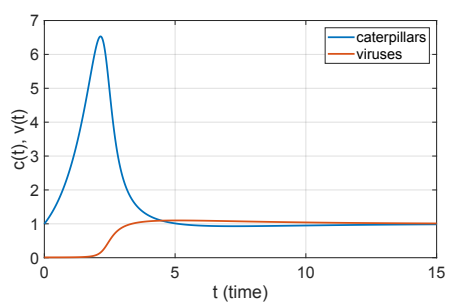

Fig. 6. Time evolution of the spiking system (4)-(5) with $\boldsymbol{p} = \mathbf{2}$ and initial conditions $(\mathbf{1}, \mathbf{0.01})$.

VII. Conclusion

We have introduced the new concept of *spiking system*, inspired by the peculiar dynamics of caterpillar-virus interactions. A spiking system has a locally asymptotically stable

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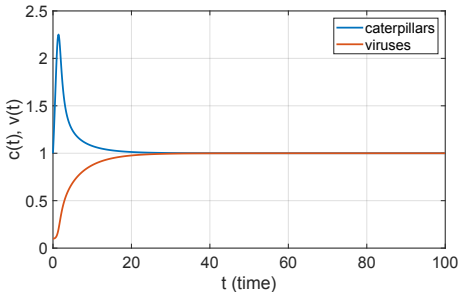

Fig. 7. Time evolution of the spiking system (4)-(5) with $p = 3$ and initial conditions $(1, 0.1)$.

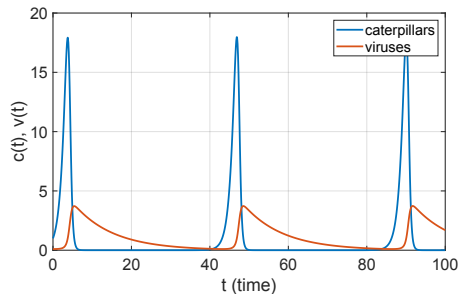

Fig. 8. Time evolution of the oscillatory system (4)-(5) with $p = 1$ and initial conditions $(1, 0.1)$.

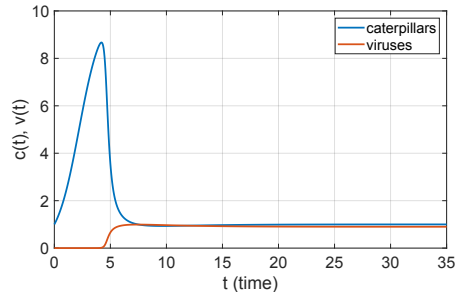

Fig. 9. Time evolution of the spiking system (6)-(7) with $p = 2$ and initial conditions $(1, 10^{-7})$.

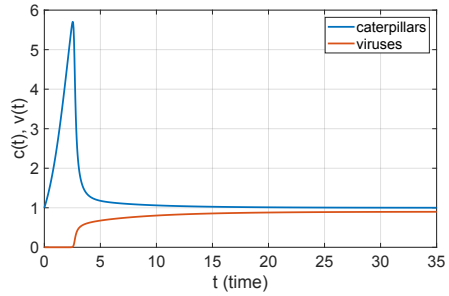

Fig. 10. Time evolution of the spiking system (6)-(7) with $p = 3$ and initial conditions $(1, 10^{-7})$.

equilibrium but, for initial conditions in a compact region including the equilibrium, its output can become arbitrarily large before the state returns within the region. To exemplify spiking dynamics in a simple but meaningful case study, we have considered a model that captures the population-infection dynamics of caterpillars and viruses, well described in the ecological literature, and proven that it is a spiking system according to our mathematical definition. For a variation of the model where the caterpillar population is bounded, and hence it cannot become arbitrarily large, we have shown that it can still get arbitrarily close to the maximum achievable in the absence of virus. More complex models that exhibit a spiking behaviour are possible and can be the object of future investigation; for instance, we believe that the idea of spiking system applies to other natural phenomena that exhibit sudden short-lived outbursts, e.g. in the population of jellyfish, insects, parasites (locust infestation), but also flames and fires.

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