Hastings 8.1

(a) $\frac{dH}{dt} = rH - bf(P)PH$

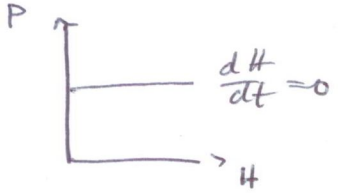
$\frac{dP}{dt} = cf(P)PH - kP$

Let $f(P) = aP$

(b) $\frac{dH}{dt} = 0$; $Hr = bf(P)PH$

$Hr = baP^2H$

$P = \sqrt{\frac{r}{ab}} = \text{constant}$

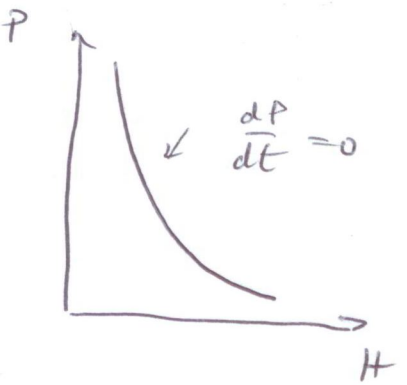


Prey isocline does <u>not</u> depend on # of prey

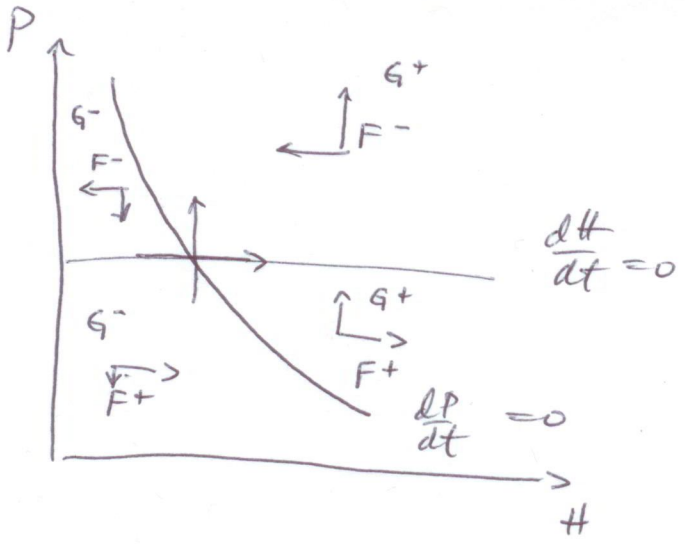
(c) $\frac{dP}{dt} = 0$; $cf(P)PH = kP$

$caP^2H = kP$

$P = \frac{k}{ca} \cdot \frac{1}{H} = \text{constant} \cdot \frac{1}{H}$



(d)



$$F = \frac{dH}{dt} > 0 \quad \text{if} \quad P < \sqrt{\frac{r}{ab}}$$

$$G = \frac{dP}{dt} > 0 \quad \text{if} \quad P > \frac{k}{acH}$$

$$J = \begin{pmatrix} \frac{\partial F}{\partial H} & \frac{\partial F}{\partial P} \\ \frac{\partial G}{\partial H} & \frac{\partial G}{\partial P} \end{pmatrix}$$

$\frac{\partial F}{\partial H} \Rightarrow 0$ $\quad \frac{\partial F}{\partial P} \Rightarrow -$

$\frac{\partial G}{\partial H} \Rightarrow +$ $\quad \frac{\partial G}{\partial P} \Rightarrow +$

$$= \begin{pmatrix} 0 & - \\ + & + \end{pmatrix}$$

Trace is sum of $\emptyset$ and $+$,
System is not stable.

For any form of $f(P)$, $\frac{\partial F}{\partial H}$ will always be zero

as long as P decreases with H, $\frac{\partial G}{\partial P} > 0$

f) this predator/prey association makes sense! Looking at the graph we can see that if the population of the predator (Insects) gets high enough, it will decrease the prey population drastically. For example, locusts swarm in very large groups and destroy entire fields of the plant prey. Their efficiency as predators can be said to be linked to how many there are in a group. At low population sizes of the predators the prey thrives. Not knowing the exact form of the predation rate does not matter. All we changed was its dependence on the # of predators, generally, so the exact function we chose does not influence our conclusions. Regardless, changing the predation rate to be dependent on the number of predators will make the system unstable.

e) This model makes sense to me. If the insect density is above a certain point, it will drive the plant abundance down. It is as if there is a slight time lag built into this model; plant abundance decreases while insect abundance increases (insects eating plants), after a while plant abundance decreases so much that insect abundance also starts to decline (insects running out of food), as insect abundance declines the plants have a chance to regrow and plant abundance increases, until there are enough plants for insect abundance to start increasing again, and the whole cycle starts again. The exact form of the predation rate won't matter to the conclusions drawn from this model, for it will only change the positions of the 2 isoclines while their relative positions will remain the same.

(f) LIKE WE LOOK AT WHERE $dH/dt = 0$ INTERSECTS $dP/dt = 0$, AND IMAGINE THAT DIFFERENT FACTORS COULD SHIFT THIS POINT, WE WOULD SEE THAT IN ORDER FOR H TO DO THE # INSECTS WOULD HAVE TO DRASTICALLY INCREASE. THIS IS WHY INSECT OUTBREAKS DO NOT OCCUR FOR SUSTAINED AMOUNTS OF TIME - THE EQUILIBRIUM IS UNSTABLE. THE FACT THAT THE ROLE OF THE PREDATION EQUATION IS UNIMPORTANT SAYS TO ME THAT NO MATTER HOW THE PER-PREDATION-RATE IS MODELED, AN UNSTABLE EQUILIBRIUM RESULTS.

f.) The assumption that predators help each other such as insects overwhelming the defenses of a plant when the density of insect is sufficiently high does make sense. An example of this would be the effect of locus on crops where multiple locus attack plants. Another example of this would be ants. Ants attack a plant with a high number of individuals. The exact form of the predation rate is not critical here because the number of predators will never reach zero and will increase infinitely with a constant prey population. If you were to change the number of prey it would have no real effect on the predator population.

8.3 Hastings

8.22 $\frac{dH}{dt} = rH\left(1 - \frac{H}{k}\right) - bHP$

8.23 $\frac{dP}{dt} = cHP - kP$

Add to each equation a term representing the effect of the pesticide. Assume this additional mortality term is density-independent. Assume H = pest

$$\frac{dH}{dt} = rH\left(1 - \frac{H}{k}\right) - bHP - dH$$

$$\frac{dP}{dt} = cHP - kP - eP$$

here e, d are positive constants

Solve for isoclines

$$\frac{dH}{dt} = 0 = rH - \frac{rH^2}{k} - bHP - dH = (r-d)H - \frac{rH^2}{k} - bHP$$

$$\frac{dP}{dt} = 0 = cHP - (k+e)P$$

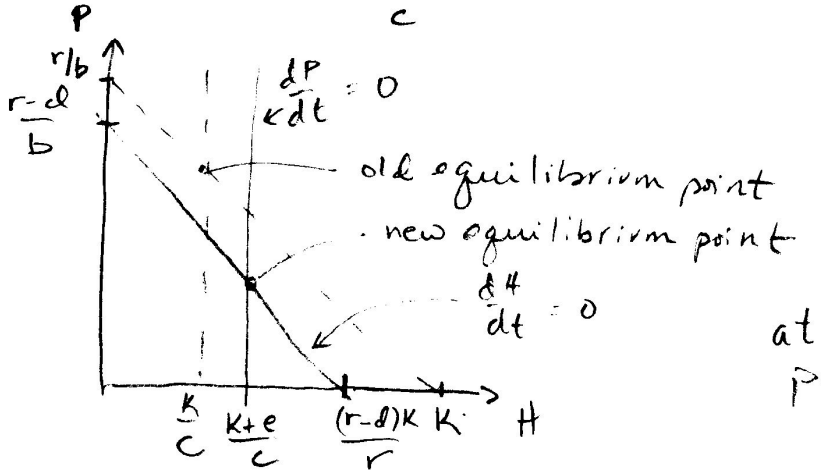
$$cH = k+e$$

$$H = \frac{k+e}{c}$$

$$r - d - \frac{rH}{k} - bP = 0$$

$$bP = r - d - \frac{r}{k}H$$

$$P = \frac{r-d}{b} - \frac{r}{kb}H$$



when P = 0

$$H\frac{r}{kb} = \frac{r-d}{b}$$

$$H = \frac{(r-d)k}{r}$$

at new equilibrium point, pest abundance (H) has increased

From Lacie: The pesticide negatively effects the predator more than the prey. When sprayed not only do the predator numbers drop, but their food supply falls as we're putting more pressure on the predator population. While some of the prey do die after spraying, the predators that kill them also die, which ultimately helps the prey population.

Hastings 8.4

Our original Lotka-Volterra model is

$$\frac{dH}{dt} = rH - bHP$$

$$\frac{dP}{dt} = cHP - kP$$

(a) Consider the case where a fraction of the prey are free from predation. Let a fraction, s, of the population be safe, and $1-s$ be available to the predator.

Our prey and predator equations now become

$$\frac{dH}{dt} = rH - b(1-s)HP$$

$$\frac{dP}{dt} = c(1-s)HP - kP$$

We can simplify and define a new $b' = b(1-s)$ and $c' = c(1-s)$ and regain the basic model

$$\left.\begin{aligned}\frac{dH}{dt} &= rH - b'HP \\ \frac{dP}{dt} &= c'HP - kP\end{aligned}\right\}$$ we have no effect on stability, so this is the same form as the original L-V model

(b) now let

$$f(H) = \begin{cases} 0 & H < H_c \\ H & H \geq H_c \end{cases}$$

where H_c is a critical prey population threshold.

For prey population densities below the threshold ($R < R_c$)

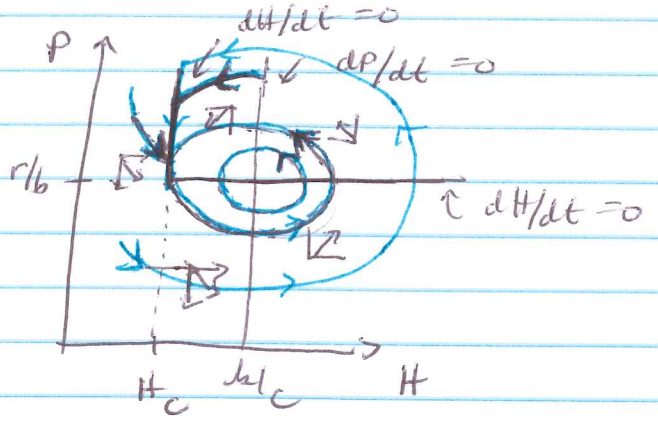
$$\frac{dH}{dt} = rH$$

$$\frac{dP}{dt} = -lP$$

Above the threshold ($R > R_c$)

$$\frac{dH}{dt} = rH - bHP$$

$$\frac{dP}{dt} = cHP - lP$$



(c) A refuge for the prey "stabilizes" the predator-prey oscillations. We now see a portion of the graph where the prey is capable of increasing even though the predator population may be very large. This is another model where predator-prey coexistence is not limited to an equilibrium.

FOR recent, formal analysis see Krivan 2011.

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On the Gause predator–prey model with a refuge: A fresh look at the history

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ABSTRACT

This article re-analyses a prey–predator model with a refuge introduced by one of the founders of population ecology Gause and his co-workers to explain discrepancies between their observations and predictions of the Lotka–Volterra prey–predator model. They replaced the linear functional response used by Lotka and Volterra by a saturating functional response with a discontinuity at a critical prey density. At concentrations below this critical density prey were effectively in a refuge while at a higher densities they were available to predators. Thus, their functional response was of the Holling type III. They analyzed this model and predicted existence of a limit cycle in predator–prey dynamics. In this article I show that their model is ill posed, because trajectories are not well defined. Using the Filippov method, I define and analyze solutions of the Gause model. I show that depending on parameter values, there are three possibilities: (1) trajectories converge to a limit cycle, as predicted by Gause, (2) trajectories converge to an equilibrium, or (3) the prey population escapes predator control and grows to infinity.

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0. Introduction

Soon after Lotka (1926) and Volterra (1926) published their theoretical treatments on prey–predator population dynamics, their predictions were experimentally tested by Gause (1934) and Gause et al. (1936). Gause focused on three experimental predator–prey systems consisting of prey (*Aleuroglyphus agilis*) and predatory (*Cheyletus eruditus*) mites, prey (*Paramecium caudatum*) and predatory (*Didinium nasatum*) protists, and yeast (*Saccharomyces exiguus*) and protists (*Paramecium bursaria*). In none of these experiments population dynamics were consistent with the Lotka–Volterra neutrally stable limit cycles. In experiments with protists and mites a prolonged coexistence of both prey and predators was obtained only when both species were regularly added to the system. In a completely homogeneous environment *Didinium* destructed all prey and it collapsed subsequently. When the environment was not homogeneous and there was a refuge for prey, prey survived in the refuge but the predator population collapsed. The situation was different when protists fed on the yeast. There was strong experimental evidence that population dynamics tended to a limit cycle that was independent from initial population numbers. These observations lead Gause et al. (1936) to search for discrepancies in assumptions of the Lotka–Volterra predator–prey model when applied to their experimental systems. First, they observed that protists were not able to feed on yeast at low densities, because at low yeast densities the prey formed into a sediment at the bottom which was not accessible to predators inhabiting the water column. Thus, prey were effectively in a refuge when at low concentrations. When prey reached above the critical density, they re-appeared in the water column and became accessible to predators. Second, they postulated that consumption of prey was a saturating function of prey density. Third, they observed that the predator population did not start to decrease until most of the predators were in the refuge. To describe their observations mathematically, Gause et al. (1936) substituted the linear consumption rate used in the Lotka–Volterra model by a saturating function that was zero below the critical prey density and had a jump (discontinuity) at the critical prey density. Hereafter I will refer to this model as the Gause model. Models with a saturating functional response were introduced to theoretical ecological literature later on by e.g., Rosenzweig and MacArthur (1963) who replaced the linear functional response in the Lotka–Volterra model by the Holling (1959) type II functional response. Besides the fact that the Rosenzweig–MacArthur model assumes negative density dependent prey growth, another difference between these two models is the jump in the functional response in the Gause model. The Rosenzweig–MacArthur model became one of the keymodels of prey–predator interactions because it documents that prey–predator coexistence is not limited to an equilibrium. So did the Gause model. However, the mechanism that leads to

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the limit cycle in the Rosenzweig–MacArthur model is entirely different from the mechanism that causes fluctuations in the Gause model. In the Rosenzweig–MacArthur model the limit cycle is caused by interaction between bottom-up and top-down prey regulation. When prey are limited by resources (i.e., when environmental carrying capacity is low), bottom-up regulation is strong and keeps predator and prey population dynamics at an equilibrium. As environmental carrying capacity increases due to enrichment, bottom-up regulation gets weaker and the population equilibrium becomes unstable due to the destabilizing Holling type II functional response. In the case of the Gause model there is no bottom-up regulation of prey growth so the nature of the limit cycle is solely due to top-down regulation and refuge presence. In fact, the Gause functional response is of the Holling type III, so their analysis clearly showed that such a functional response can lead to a limit cycle in predator–prey population dynamics.

In general, proving existence and uniqueness of a limit cycle in predator–prey population dynamics is not trivial. For example, the limit cycle in the Rosenzweig–McArthur model appears when the stable equilibrium undergoes the Hopf bifurcation and its uniqueness was proved only much later (e.g., Huang and Merrill, 1989). Certainly, these concepts were unknown when Gause et al. (1936) analyzed their system. Therefore, it is quite remarkable that using a simple geometrical argument, they were able to predict that trajectories of their model converge to a limit cycle (see their Figure 5). However, there is one crucial problem with their analysis. The Gause model was described by a differential equation with a discontinuous right-hand side (due to the jump in the functional response). Such differential equations may not have solutions in the usual sense[1] and the Gause model is an example. A concept of a "solution" for such models was introduced later on by Filippov (1960) (see also Aubin and Cellina, 1984; Filippov, 1988). No such mathematical concept existed at the time when Gause with his co-workers analyzed their model to achieve a better fit with experimental data. This is really a remarkable example of biological research that used a mathematical methodology that was not yet developed at that time.

In this article I will explain how solutions of the Gause model can be defined and will analyze this model with respect to parameter values and initial population densities. I will show that trajectories of the Gause model can converge to a limit cycle as correctly predicted by Gause et al. (1936), but they can also converge to an equilibrium, or prey can escape completely predator regulation.

1. The Gause model

Gause et al. (1936) considered the following adaptation of the Lotka–Volterra predator–prey model

$$\frac{dR}{dt} = rR - Cf(R)$$

$$\frac{dC}{dt} = (ef(R) - m)C \tag{1}$$

Here R is the prey density, C is the predator density, r is the per capita prey population growth rate, f is the Gause functional response specified below, e is the efficiency rate with which captured prey are converted to new predators, and m is the predator mortality rate. There are three distinctive features of this functional response: (1) below a critical prey population threshold (denoted by R_c) prey are not consumed, (2) the functional response has a discontinuity at the threshold, (3) consumption saturates with increasing prey density (Gause et al., 1936). The first feature corresponds to the refuge of a fixed size R_c. The jump at the critical prey density was motivated by their observation that "… predators … do not seriously decrease in concentration until the destruction of the prey down to this threshold…" (Gause et al., 1936). This suggests that the functional response in the vicinity of R_c is quite steep, and can be approximated by a functional response with a jump at R_c. In fact, as we will see below, this assumption allowed authors to analyze the predator–prey population dynamics using a simple geometric argument. Moreover these authors also asked that "the tangent to f … crosses the ordinate and not the abscissa". This latter condition excludes coexistence of prey and predators in a locally asymptotically stable equilibrium and it is interesting to note that this seems to be the first occurrence of the now well known (in)stability condition for prey–predator interactions (Murdoch and Oaten, 1975). A prototype of such a functional response is (see the solid curve in Fig. 1A)

$$f(R) = \begin{cases} 0 & R < R_c \\ \dfrac{\lambda R}{1 + h\lambda R} & R \geq R_c \end{cases} \tag{2}$$

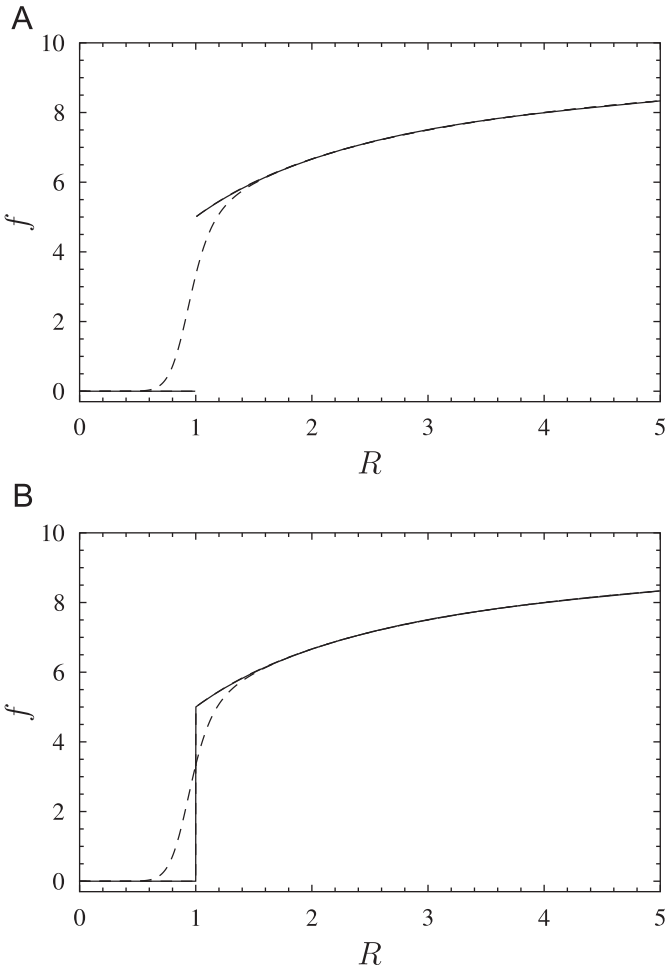

Fig. 1. Panel A (solid line) shows the Gause functional response (2). Panel B shows the Filippov regularization (7) (solid line) of the Gause functional response. The dashed line in both panels is the Holling type III functional response given by (3). Parameters: $R_c = 1$, $\lambda = 10$, $h = 0.1$, $\mu = 10$.

[1] A trivial example is the differential equation $dx/dt = 1$ when $x < 0$ and $dx/dt = -1$ when $x > 0$. Unless we set $dx/dt = 0$ when $x = 0$, this differential equation has no solution starting at $x(0) = 0$.

Here parameter λ describes the search rate of a predator and h is the handling time a predator needs to process one unit of prey. R_c is the critical prey density below which prey are not accessible to predators. Thus, for above-critical prey density, f is the Holling type II functional response. The Gause functional response (2) is a limiting case of the Holling type III functional response (dashed curve in Fig. 1A)

$$f_{III}(R)=\frac{\lambda R^{\mu}/(R^{\mu}+R_s^{\mu})R}{1+h\lambda R^{\mu}/(R^{\mu}+R_s^{\mu})R} \tag{3}$$

when the exponent μ tends to infinity. I will show now that model (1) is not well defined in the sense that solutions cannot be continued in forward time once prey density reaches the critical threshold R_c, and predator density is high enough.

For prey population densities below the threshold ($R<R_c$), prey not being eaten by predators grow exponentially while predators die exponentially, i.e., model (1) becomes

$$\frac{dR}{dt}=rR$$

$$\frac{dC}{dt}=-mC \tag{4}$$

Thus, at each point of the prey–predator density phase space to the left of the critical prey density $R=R_c$, trajectories move in the south-east direction.

For prey population densities above the threshold ($R>R_c$), population dynamics (1) are given by the Lotka–Volterra model with the Holling type II functional response

$$\frac{dR}{dt}=rR-C\frac{\lambda R}{1+h\lambda R}$$

$$\frac{dC}{dt}=\left(\frac{e\lambda R}{1+h\lambda R}-m\right)C \tag{5}$$

First, I will recall properties of model (5) when no refuge exists ($R_c=0$, e.g., Murdoch and Oaten, 1975; Svirezhev and Logofet, 1983; Křivan, 2008). For small handling times that satisfy $h<e/m$ this model has an interior, unstable equilibrium $E^{\star}=\{R^{\star},C^{\star}\}=\{m/(\lambda(e-mh)),re/(\lambda(e-mh))\}$ (Appendix A). Depending on parameters and initial conditions, model (5) has two types of solutions (Appendix A). For small handling times that satisfy $h<e/(r+m)$ all trajectories of model (5) spiral outward from the interior equilibrium with ever increasing amplitude. For larger handling times ($e/m>h>e/(r+m)$) there exist trajectories that tend to infinity without spiraling around the equilibrium (see the bottom trajectory in Fig. 2B, F). In such a case, the prey population completely escapes predator regulation.

Second, I will assume a refuge exists ($R_c>0$) and I will consider model (1) with the Gause functional response (2). For $R>R_c$ the prey isocline coincides with the prey isocline of model (5) and it is given by

$$C=\frac{r}{\lambda}(1+\lambda hR)$$

(thick dashed line in Fig. 2). For small prey densities satisfying $R<R_c$ the prey isocline is not defined. Let us consider the line $R=R_c$ in the prey–predator density phase space. The isocline analysis shows that in the vicinity and to the right of this line, the prey population decreases provided predator density is high enough, i.e.,

$$C>C_c=\frac{r}{\lambda}(1+\lambda hR_c) \tag{6}$$

Here the point (R_c, C_c) corresponds to the intersection of the prey isocline of model (5) with the critical prey line $R=R_c$ (the corner of the thick dashed line in Fig. 2). Similarly, to the left of this line, the prey population increases. It is clear that above the critical predator density C_c trajectories of the Gause model are pushed from both sides to the line $R=R_c$ and they cannot leave this line if they exist. Thus, trajectories of model (1) cannot cross the critical line $R=R_c$ above the point C_c and, by definition (the prey isocline consists of points in the prey–predator density phase space where prey population neither increases nor decreases, Rosenzweig and MacArthur, 1963), this half-line must be a part of the prey isocline of the Gause model. Thus, the prey isocline of the Gause model is L-shaped (the thick dashed line in Fig. 2).

However, the most interesting feature of the Gause model is the fact that once a trajectory falls on the vertical part of the prey isocline, it cannot be continued any further. Indeed, as the "trajectory" cannot leave the line $R=R_c$ above the point C_c it follows that $dR/dt=0$. But the right-hand side of model (1) evaluated at $R=R_c$ gives $dR_c/dt\neq 0$. In other words, the Gause model is not well posed because its trajectories are not defined when they fall on the vertical part of the prey isocline. This is a consequence of the fact that the Gause functional response has a "jump" at the critical prey density, because such models may not have solutions. In other words it is not clear how Gause et al. (1936) defined the vector field in their Figure 5, panel 1' for $R=R_c$. It took more than 20 years after Gause et al. (1936) published their article before Filippov (1960) (see also Filippov, 1988) introduced a new solution concept for such models. The crucial step is provided through suitable definition of the vector field at the critical prey density, which I briefly describe now. The Filippov solution concept applied to the Gause model defines a new vector field at the critical prey density R_c as the line segment with end points given by the two adjacent vector fields f^1 and f^2. Here $f^1=(rR_c,-mC)$ stands for the vector field defined by the right-hand side of model (4) and $f^2=(rR_c-C\lambda R_c/(1+\lambda hR_c), e\lambda R_cC/(1+\lambda hR_c)-mC)$ for the vector field defined by model (5). This new (multivalued) vector field is given by

$$F=\alpha f^1+(1-\alpha)f^2$$

where $0\leq\alpha\leq 1$. In other words, this vector field associates to every point along the vertical part of prey isocline a whole set of possible directions given by F. This definition of the Filippov field re-defines the functional response (2) at the critical prey density to

$$f(R)=\begin{cases}0 & R<R_c\\ \left[0,\dfrac{\lambda R_c}{1+\lambda hR_c}\right] & R=R_c\\ \dfrac{\lambda R}{1+h\lambda R} & R>R_c\end{cases} \tag{7}$$

It follows that under this new definition $f(R_c)$ is the line segment $[0,\lambda R_c/(1+\lambda hR_c)]$ that fills the gap in the Gause functional response (Fig. 1B). This definition is very natural as it reflects the fact that at the critical prey density the functional response does not specify exactly the prey consumption by predators. Thus, functional response (7) is a very reasonable approximation of a continuous Holling type III functional response that is steep enough at prey densities close to the critical prey density (Fig. 1B). Appendix B shows that model (1) with Filippov vector field at the critical prey density has uniquely defined trajectories for every initial condition.

To analyze the Gause model we need to know the dynamics along the vertical part of the prey isocline. When trajectory of model (1) falls on the vertical part of the prey isocline and the predator density satisfies $C>C_c$, the trajectory cannot leave the isocline and it must move vertically, i.e., $dR/dt=0$. This implies that along such a trajectory

$$\alpha=1-\frac{r(1+\lambda hR_c)}{\lambda C},$$

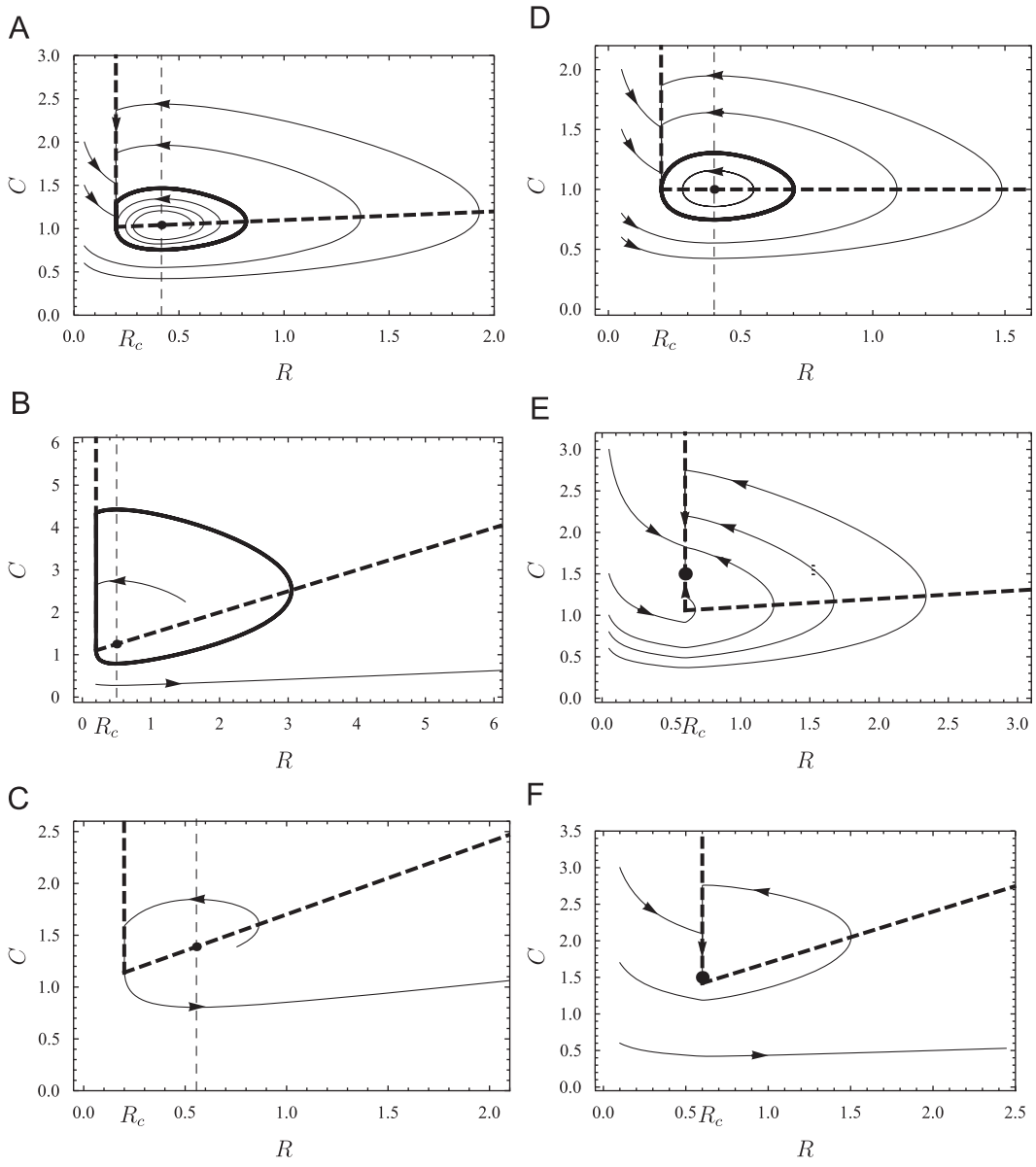

Fig. 2. Panels A–D assume that the critical prey density (R_c=0.2) is smaller than the equilibrium prey density $R^\star$ of model (5), while panels E and F assume the opposite case (R_c=0.6). Panel A shows the trajectories of the Gause model when handling time is small (h=0.1) and all trajectories converge to a limit cycle. For intermediate handling times (h=0.5, panel B) a locally asymptotically stable limit cycle exists, but there are also trajectories that tend to infinity. For yet larger handling times (h=0.7, panel C) all trajectories diverge from the equilibrium. Panel D shows the case where handling time is zero. Panel E assumes low handling time (h=0.1) in which case all trajectories converge to an equilibrium point that is located at the vertical part of the prey isocline. Panel F assumes a larger handling time (h=0.7) where the equilibrium is only locally asymptotically stable because some trajectories tend to infinity. Other parameters: r=1, λ=1, m=0.2, e=0.5.

which corresponds to the vector $f^F=(0,erR_c-mC)\in F$, and the population dynamics are

$$\frac{dR}{dt}=0$$

$$\frac{dC}{dt}=erR_c-mC \tag{8}$$

The above equation describes population dynamics along the line $R=R_c$ as long as $C>C_c$. At the point (R_c, C_c) the trajectory leaves the line $R=R_c$ and because the consumer density is low, it enters the region where $R>R_c$ (this also follows from considerations in Appendix B). Thus, model (8) provides a formal definition of the vector field along the horizontal line in Figure 5, panel 1' of Gause et al. (1936) (I remark that axes are swapped in Gause et al., 1936 so the line $R=R_c$ is horizontal there.) It is interesting to observe that there exists a new equilibrium $E^F=(R^F,C^F)=(R_c,erR_c/m)$ of model (8), provided this point is on the vertical part of the prey isocline, i.e., when $C^F>C_c$. This holds whenever the prey critical density satisfies $R_c>m/(\lambda(e-mh))$, i.e., when the predator isocline of model (5) is to the left of the prey critical line $R=R_c$, or, in other words, if the prey refuge is large enough.

Now I will analyze qualitative behavior of the solutions of the Gause model with respect to parameters and initial population densities. I will consider two cases, depending whether the critical prey population density R_c is smaller or larger than the equilibrium prey density $R^\star=m/(\lambda(e-mh))$ of model (5). First, I will assume that the critical prey density is smaller ($R_c<R^\star$, Fig. 2 A–D). Fig. 2A shows the case where the handling time is so small that all trajectories of model (5) spiral around the equilibrium. In this case, starting from any initial population densities, the corresponding trajectory must fall at certain time onto the

vertical part of the prey isocline. The trajectory will then follow dynamics described by (8) and because in this case there is no equilibrium on the vertical part of the prey isocline, the trajectory must reach at a positive time the lower end-point (R_c, C_c) (i.e., the corner of the prey isocline). At this point it will leave the isocline and a limit cycle is formed (the heavy solid cycle in Fig. 2A). All trajectories will reach this limit cycle in a finite time. This is the limit cycle predicted by Gause et al. (1936).

For larger handling times (i.e., $e/m > h > e/(r+m)$), model (5) also has trajectories that do not spiral around the equilibrium. There are two possibilities depending on the position of the critical point (R_c,C_c). The trajectory that starts at this point returns to the critical line $R=R_c$ (Fig. 2B) in which case a limit cycle exists. However, this limit cycle is not globally stable, because there are trajectories that tend to infinity (see the bottom trajectory in Fig. 2B). Numerical simulations show that for yet higher handling times the trajectory that starts at the point (R_c, C_c) tends to infinity (Fig. 2C) and predators lose control over their prey.

Second, I consider the case where $R_c > R^\star$ (Fig. 2E, F). In this case, equilibrium $E^\star$ of model (5) is located to the left of the prey critical density line in the prey–predator phase space where dynamics are described by (4). Thus, it is no longer an equilibrium of the Gause model. However, the equilibrium E^F is on the prey isocline and therefore is the only equilibrium of the Gause model. Similar to the case where the prey critical density line was to the left of the predator isocline, for small handling times all trajectories must fall onto the vertical part of prey isocline and then move along it to reach the equilibrium E^F (Fig. 2E). For higher handling times, some trajectories will escape predator regulation (Fig. 2F) so that the equilibrium is then only locally asymptotically stable.

These results are summarized in Fig. 3. This is an example of a bifurcation diagram in the (h,R_c) parameter space. The solid curve in Fig. 3 corresponds to the transition from the locally stable limit cycle in Fig. 2A to the locally stable equilibrium (panel E). This curve is given by equation $R_c=R^*$, i.e., $R_c = m/(\lambda(e-mh))$. Such bifurcation was called the boundary-focus bifurcation (see the case BF_3 in Figure 5 in Kuznetsov et al., 2003). The dashed line in Fig. 3 corresponds to transition between Fig. 2B and C, i.e., disappearance of the limit cycle in Fig. 2B, and was obtained by numerical simulations.

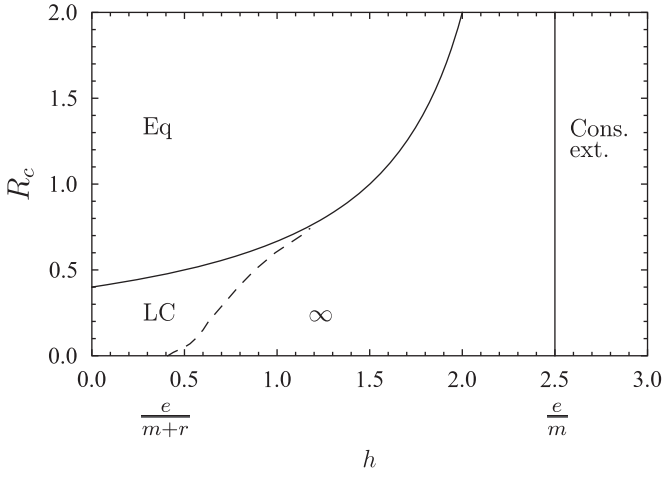

Fig. 3. A bifurcation diagram for the Gause model with respect to handling times and the refuge size. The solid curve corresponds to the transition from the limit cycle (Fig. 2A) to the stable equilibrium (Fig. 2E) and is given by $R_c = m/\lambda(e-mh)$. The dashed curve corresponds to the set of parameters where the limit cycle disappears. LC stands for the limit cycle, Eq stands for the interior equilibrium, ∞ means that no bounded attractor exists, and the right region (Cons. ext.) denotes the set of parameters for which consumers extinct and prey grow exponentially.

2. Discussion

In this article I have analyzed a prey–predator model introduced by Gause et al. (1936) to explain cycles in prey–predator population dynamics which they observed in some experiments. They replaced the linear functional response used by Lotka and Volterra by a functional response that was zero below some critical prey threshold density, had a "jump" at this threshold, and was saturating at high prey densities. Such a functional response is an extreme form of the Holling type III sigmoid functional response that was introduced later on (Holling, 1959). Using this functional response Gause et al. (1936) was able to show that predators and prey can coexist along a limit cycle. This is perhaps the very first models in ecology that shows species coexistence is possible not only at an equilibrium. They also showed that a prey refuge can lead to predator–prey coexistence as the same model without a refuge does not predict such a coexistence. Moreover, they derived a general stability condition for predator–prey models. Thus, this article was really fundamental for further development of population ecology. Although their analysis is very elegant, there is one crucial problem because their model is not well defined as there are initial population densities for which no solutions exist. The question then is whether and in which sense the predictions these authors obtained from the model are correct. In this article, using the approach developed by Filippov (1960), I showed how trajectories in the Gause model can be defined. I also analyzed this model with respect to parameters and initial population densities. Gause et al. (1936) predicted that the model has a globally stable limit cycle (see their Figure 5). I showed that predators and prey can coexist along a limit cycle provided that the refuge size and handling times are not too high (see the region LC in Fig. 3). In fact, for small handling times this limit cycle is globally stable, but for slightly higher handling times there are also trajectories along which both populations tend to infinity (Fig. 2B). Along such trajectories prey "escape" predator control due to low predator efficiency to handle prey items. For yet higher handling times the limit cycle disappears and predators cannot control prey growth (see the region in Fig. 3 denoted as ∞).

A different situation occurs when prey critical density is high enough, i.e., when the prey refuge is large. Then, for small handling times a globally asymptotically stable equilibrium point exists. As handling time increases, there will be again some trajectories that will tend to infinity.

As prey below the critical density are effectively in a refuge, this analysis nicely demonstrates the stabilizing role of refugia that protect a fixed number of prey (e.g., Rosenzweig and MacArthur, 1963; Hassell and May, 1973; Maynard Smith, 1974; Murdoch and Oaten, 1975; Hassell, 1978; Sih, 1987; Ives and Dobson, 1987; Ruxton, 1995; Hochberg and Holt, 1995; Křivan, 1998). When the refuge is large enough, i.e., when the critical prey density below which prey are protected is high, provided predators are able to regulate the prey population (i.e., when handling times are small) then the two species coexist at an equilibrium. As refuge size decreases, less prey are protected and prey–predator coexistence occurs along a limit cycle.

Because the Gause functional response is of the Holling type III, analysis of the Gause model shows that such a functional response can lead to predator and prey coexistence without any prey density dependence. In fact, the effect of a refuge on predator–prey stability is somewhat parallel to the effect of enrichment on predator–prey stability in the Rosenzweig–MacArthur model. While in the Rosenzweig–MacArthur model a higher environmental capacity weakens the bottom-up control and leads to population oscillations, the effect of refuge is just opposite. A larger refuge stabilizes otherwise unstable population equilibrium.

A similar analysis for the Lotka–Volterra model where functional response is linear with a "jump" at the critical prey density shows the

same pattern (Fig. 2D). Here all trajectories converge to a global attractor which is bounded by the largest Lotka–Volterra cycle that is to the right of the vertical part of the prey isocline. For larger R_c the population dynamics converge on an equilibrium (results not shown) exactly as in the case with the Holling type II functional response.

The ultimate reason for species coexistence at an equilibrium or along a limit cycle in the Gause model is the fact that the prey isocline has a vertical segment. This vertical segment then limits the amplitude of species fluctuations as suggested by Rosenzweig and MacArthur (1963) in their seminal work. Since then it was shown that such isoclines arise when prey–predator models consider optimal foraging of predators (Křivan, 1996; van Baalen et al., 2001), optimal activity level of prey and/or predators (Křivan, 2007) or optimal use of refuges (Křivan, 1998). All these models show that prey isoclines with vertical segments (or equivalently predator isoclines with horizontal segment) can arise naturally in real populations. In fact, using models with discontinuous right-hand sides is a very natural methodology for analyzing such systems and can lead to a much simpler analysis when compared to models with strong non-linearities. Such an approach was used for example by Crowley (1981) to demonstrate an existence of a limit cycle in Rosenzweig and MacArthur (1963) model where prey isocline was assumed to be vertical at the prey carrying capacity. This corresponds to strong density dependence that operates only when prey density reaches the carrying capacity.

It is likely that the Gause predator–prey model was one of the first models in biology where discontinuous differential equations were used. It is remarkable that this happened even before mathematicians provided a formal definition of a solution for such systems. Although it does not seem that Gause work motivated some research in this field, this example clearly shows that biology can be a source of interesting problems for mathematicians. Since that time, similar models were used in ecology (e.g., Křivan, 1996, 2007; Meza et al., 2005; Dercole et al., 2007), and in gene networks (e.g., Edwards, 2000; de Jong et al., 2004; Gouzé and Sari, 2003; Casey et al., 2006). Moreover, a complete qualitative theory was developed for two-dimensional models with a discontinuity (Kuznetsov et al., 2003), and a systematic approach (called "the puzzle method") for analyzing such models was developed (Dercole et al., 2007). Moreover, software for numerical analysis of such models is also available (Dercole and Kuznetsov, 2004, 2005; Piiroinen and Kuznetsov, 2008; Thota and Dankowicz, 2008). These methodological advances open further possibilities to study models with discontinuities that were not available at times when Gause et al. (1936) wrote their pioneering treatise.

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Appendix A. Analysis of model (5)

At the interior equilibrium $E^\star=\{R^\star,C^\star\}=\{m/(\lambda(e-mh)),\ re/(\lambda(e-mh))\}$ of model (5), the Gause criterion

$$\frac{df(R^\star)}{dR}=\frac{\lambda}{(1+\lambda hR)^2}<\frac{\lambda}{1+\lambda hR}=\frac{f(R^\star)}{R^\star}$$

implies instability. Moreover, because

$$\frac{\partial}{\partial R}\left(\frac{1}{RC}(rR-f(R)C)\right)+\frac{\partial}{\partial C}\left(\frac{1}{RC}(ef(R)C-mC)\right)=\frac{1}{R}\left(\frac{f(R)}{R}-\frac{df(R)}{dR}\right)>0$$

the Dulac (1937) criterion (see also Hofbauer and Sigmund, 1998) with the Dulac function $1/(RC)$ excludes existence of a limit cycle for model (5).

When prey density is high, model (5) asymptotically tends to

$$\frac{dR}{dt}=rR-\frac{C}{h}$$

$$\frac{dC}{dt}=\left(\frac{e}{h}-m\right)C \tag{9}$$

The prey isocline is the line $C=hrR$. Below this isocline prey population increases while above it decreases. Let us consider a line given by $C=kR$ with $0<k<hr$ (i.e., this line is below the prey isocline in the prey–predator density phase space). Trajectories of model (9) cross this line upwards provided $k>h(m+r)-e$ because under this condition $dC/dR>k$ at every point of this line. It follows that when $h<e/(m+r)$ then $k>h(m+r)-e$ for every $0<k<hr$ and trajectories of model (9) will reach the prey isocline, i.e., trajectories of model (5) will spiral around its equilibrium. However, when $h>e/(m+r)$ there will be trajectories of model (9) that never reach the prey isocline (see bottom trajectories in Fig. 2B and F). Along such trajectories prey population escapes predator control.

Appendix B. Existence and uniqueness of trajectories of the Gause model

Let $n=(n_R,n_C)=(1,0)$ be the vector perpendicular to the line $R=R_c$ in the prey–predator density phase space. Projection of the two vector fields given by the right-hand sides of (4) (denoted as f^1) and (5) (denoted as f^2) are $\langle n,f^1\rangle=rR_c$ and $\langle n,f^2\rangle=rR_c-\lambda R_cC/(1+\lambda hR_c)$. If $\langle n,f^1\rangle>0$ and $\langle n,f^2\rangle<0$ trajectories are pushed from both below and above to the line $R=R_c$. These conditions hold when predator population density is large enough, i.e., $C>C_c$.

The existence of trajectories for the Gause model follows from general existence theorems that can be found in Filippov (1988) (see also Colombo and Křivan, 1993). Uniqueness of trajectories for the Gause model follows from the fact that $\langle n,f^1\rangle=\langle n,f^2\rangle+\lambda R_cC/(1+\lambda hR_c)$. Thus, it is impossible that at some points of discontinuity $\langle n,f^1\rangle<0$ and $\langle n,f^2\rangle>0$ which would imply non-uniqueness of trajectories (Filippov, 1988; Colombo and Křivan, 1993).

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