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Biological control does not imply paradox

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Abstract

Is the classical predator–prey theory inherently pathological? Defenders of the theory are losing ground in the debate. We will demonstrate that detractors' main argument is based on a faulty model, and that the conceptual and predictive bases of the theory are fundamentally sound.

Keywords: Biological control; Holling Type II functional form; Ratio-dependent predation; Intraspecific interference predator–prey model

Biological control is to introduce natural predators or parasites to ecosystems for pest control. It works when the pest populations are either eradicated or kept at a stable and low density equilibrium or low density cycle. There are numerous examples of successful biological control. A paradox was raised in [18] when a classical predator–prey model was used to solve the biological control problem. The model assumes the logistic growth for the prey (the pest), the Holling Type II predation for the predator (the control agent), and a *constant* per-capita death rate of the predator. Such a model cannot have a stable and arbitrarily low prey density equilibrium [21]. Attempts have been made to find alternative models that can exhibit controlled stable and low

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density states [4,6]. One proposal is to replace the prey-dependent Holling Type II predation form [14] by the ratio-dependent functional form [4]. It has been controversial ever since largely because the ratio-dependent form lacks a mechanistic derivation at a level comparable to Holling's work [5,1,3]. Nevertheless it has been steadily gaining acceptance in the field of population ecology [4,2,16,8,15]. The key justification by its proponents lies in a perceived predictive attribute that the ratio-dependent model is able to *phenomenologically* produce arbitrarily small and stable equilibrium for both predator and prey [4]. Hence, 'in a biological sense, it is the prey-dependent model that shows pathological behavior' [2]. Without its predictive power, the usefulness and consequently the validity of the classical theory are seriously diminished. Traditionalists are losing ground in the debate and the predator–prey research is seemingly back to the days prior to Holling's 1959 seminal work on predation.

We argue that the suggestion that the classical theory is in a fundamental crisis is unfounded for a reason that its detractors' argument is based on a faulty model. The root of problem lies in the overly simplistic assumption that the predator dies at a constant per-capita rate. In theory it is in consistent with Verhulst's logistic modelling principle [24,17,25] that species per-capita death rate should be density dependent. As a consequence it produces a vertical per-capita nullcline for the predator. It in turn leads to an absurdity that if one can fix, at least conceptually, the prey density at a level greater than the nullcline, the predator population would grow exponentially to infinity. The fundamental contribution of Lotka, Volterra, and Verhulst from the first quarter of last century taught us that one should always take into account the effect of intraspecific interference at high density levels unless such a consideration is justifiably not warranted. This complete version of the classical theory was used by Rosensweig [19, pp. 564–565] to settle yet another well-known 'paradox' – the paradox of enrichment [11,21]. He called such predators 'squabbling'. The logistic predator model can exhibit enrichment paradox as he originally raised in [21], not as a case against the classical theory, rather as a case for caution out of a biologically counterintuitive yet mathematical valid principle, though not biologically as universal as the first impression his original work was likely to leave. More specifically, for a large squabbling coefficient, the enrichment paradox phenomenon does not occur, thus accommodating the class of ecosystems for which enrichment does not lead to destabilizing cycles. This latter part of his work from [19] is commonly overlooked, especially when the enrichment paradox is cited in the literature as another pathology of the classical theory, in addition to the biological control paradox.

The difference between the exponential predator–prey model, which the proponents of ratio-dependent predation use to question the mechanistic theory, and the logistic predator–prey model, which they overlooked, cannot be greater [7]. For the former, there can be no more than one non-trivial equilibrium which cannot be small for both predator and prey and stable at the same time. For the latter, there can be 1, 2, and 3 non-trivial equilibrium points, with the 2-equilibrium state being a bifurcation state between the 1-equilibrium and 3-equilibrium states. If there is only one equilibrium point, it can be stable independent of where it is. If there are three, then the right most and the left most equilibrium points can be stable, one sits on each side of the prey nullcline hump. All that the stability requires is to have a sufficiently large reproductive ratio of the predator over the prey (the predator-to-prey prolific ratio). Furthermore a small cycle can be born from the left most equilibrium point if the point is to the left of the prey nullcline hump and the ratio decreases and crosses a Hopf bifurcation point. The amplitude of the limit cycle increases with decrease in the ratio, giving way to the destabilizing regime of 'enrichment paradox'.

See Appendixes A and B, and Fig. 1(a). These predictions are perfectly consistent with what is expected for an effective biological control. In addition, when the left most equilibrium is near the origin, the prey density is in the order of $d/[c(b - hd)]$, and the predator density is in the order of r/c, where r is the intrinsic prey growth rate, b is predator's birth-to-consumption ratio, c is predator's rate of discovery, d is predator's intrinsic death rate, h is predator's handling time, all measured in per-capita rate. As an example, a high predator discovery rate, c, can result in low equilibrium density for both species. Other combinations of the parameters can result in the same phenomenon of low density and stable states. Those are self-evident from the formula.

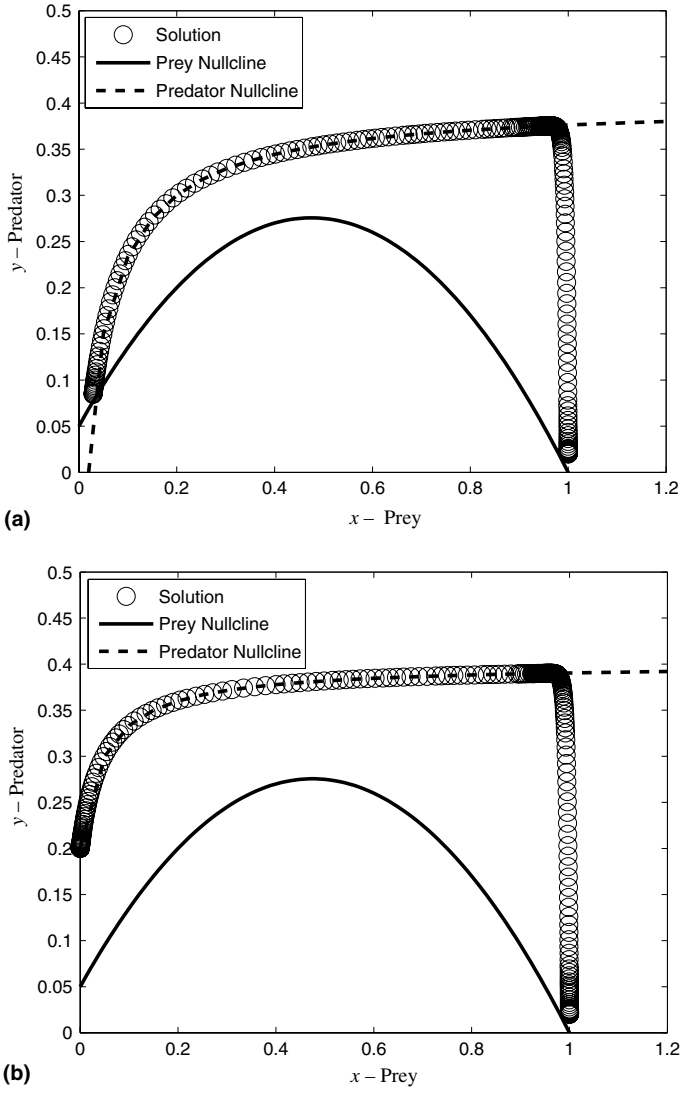

Fig. 1. (a) Parameter values for Eq. (2) $\epsilon = 100$, $\beta = 0.05$, $\delta = 0.2$, $\sigma = 0.2$. The solution starts near the scaled prey capacity (1, 0). (b) Same as (a) except that $\alpha = 0.3$ in Eq. (5).

The exponential predator–prey model is a poor choice for biology and for control. It is a reasonable model only if the prey out-reproduces the predator, presumably the case for many natural systems. It is not an appropriate model if the predator must dominate the prey as in pest control situations we typically envision. The logistic predator–prey model on the other hand is the basis for both. With respect to control in practice, management strategies can be modelled and incorporated into the model *mechanistically*. One can supply a system with a constant influx of predator, or artificially sustain a high level of predator density by supplying it with an alternative food source. In the latter case, assuming the predator consumes the pest prey and the alternative prey or nutrient indiscriminately, then it can be shown that for high enough densities of the alternative prey the per-capita nullclines of the pest prey and the controlling predator do not intersect. Instead, a globally attracting, non-vanishing, predator-only equilibrium appears that is supported by the alternative prey with the pest prey extinct. See Appendix C and Fig. 1(b). In a true control sense the predator can be eliminated, if desired, by withdrawing the alternative prey once the pest prey is eradicated.

In conclusion, the classical theory is not intrinsically pathological. Its conceptual and predictive bases remain fundamentally sound. Justification for replacing Holling's Type II predation form in predator–prey modelling by ad hoc and phenomenological forms cannot be made on the ground that it leads to paradoxes.

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Appendix A. The logistic predator–prey model

The classical predator–prey model based on the logistic growth principle and Holling's predation theory is as follows:

$$\begin{aligned} \dot{X} &= X\left[r\left(1-\frac{X}{K}\right)-\frac{pY}{H+X}\right], \\ \dot{Y} &= Y\left[\frac{bpX}{H+X}-(d+sY)\right], \end{aligned} \tag{1}$$

where r is prey's intrinsic growth rate, K is prey's carrying capacity, p is predator's per-capita saturation capture rate, H is the semi-saturation density, b is predator's birth-to-consumption ratio, d is predator's intrinsic death rate, s is predator's intraspecific death rate coefficient, the'squabbling' coefficient. Also $p = 1/h$, $H = 1/(ch)$ with h being predator's per-prey handling time and c predator's per-capita prey-discovery rate.

Using the following rescaling [10] for the variables:

$$x = X/K, \quad y = Y/(rK/p), \quad t := bpt$$

and parameters

$$\epsilon = bp/r, \quad \beta = H/K, \quad \delta = d/bp, \quad \sigma = rKs/p$$

we get the following dimensionless form:

$$\begin{aligned} \epsilon\dot{x} &= x\left[1 - x - \frac{y}{\beta + x}\right], \\ \dot{y} &= y\left[\frac{x}{\beta + x} - (\delta + \sigma y)\right]. \end{aligned} \tag{2}$$

For $\sigma > 0$, predator's per-capita nullcline has the same predation disc form: $y = [x/(\beta + x) - \delta]/\sigma$, Fig. 1(a). It is monotonically increasing in x with saturation $(1 - \delta)/\sigma$. It represents the prey-supported carrying capacity for the predator. One of the important features of this form is that it can segregate the system into different time scales. Specifically, if the predator-to-prey prolific ratio ϵ is sufficiently large, then the temporal change in x is negligible if y is not near its capacity nullcline state and becomes significant if and only if y is so. Therefore solutions will first quickly converge to the y-capacity nullcline and then slowly develop along the capacity curve to the non-trivial equilibrium, wherever it locates. If on the other hand ϵ is small, the time scales for the species are reversed: x changes faster than y does. In such a case solutions congregate near the x-nullclines. It matters little whether or not the per-capita y-nullcline is vertical or monotonically increasing in x. Consequently, the exponential model works as well as the logistic model.

The same results apply to models based on various Holling Type II expanded functional forms [9,23] for which predator's intraspecific interference is considered in the context of spacial interference of predation. All these forms will generate a non-vertical prey-supported capacity nullcline for the predator whether or not the death rate coefficient, s, of the predator is zero. Consequently, the non-trivial equilibrium point near the origin is always generically stable for sufficiently large predator-to-prey prolific parameter ϵ.

On the magnitude of the xy-equilibrium point for the logistic predator–prey model above, we see that when the equilibrium is near the origin it is near both axes. Specifically, the x-intercept of the y-nullcline is $x_* = \beta\delta/(1 - \delta)$, and the y-intercept of the x-nullcline is $y_* = \beta$. In the dimensional form, the corresponding equilibrium point $(\overline{X}, \overline{Y})$ is approximately $\overline{X} \approx Kx_* = d/[c(b - hd)]$ and $\overline{Y} \approx rKy_*/p = r/c$. This approximation is better if the squabbling coefficient s is small since the y-nullcline will become more vertical.

Appendix B. Proof of stability

The stability of the equilibrium point can be established alternatively. Let f and g be the right hand of the x-equation and y-equation respective in Eq. (2) and let

$$\begin{aligned} \epsilon\dot{u} &= f_1 u + f_2 v, \\ \dot{v} &= g_1 u + g_2 v, \end{aligned} \tag{3}$$

where $f_1 = \partial f/\partial x$ evaluated at an/the xy-equilibrium point and so on. The corresponding u-nullcline $f_1 u + f_2 v = 0$ is the tangent line to the humped x-nullcline at the equilibrium point. Thus

we always have $f_2 < 0$, and $f_1 > 0$ if the equilibrium point is left of the hump and $f_1 < 0$ if it is right of the hump. Similarly, $g_1 > 0$, $g_2 < 0$ no matter where the equilibrium is. These conditions imply that the equilibrium point right of the hump is *always* stable. For the equilibrium point left of the hump and closest to the origin, the slope of the v-nullcline is greater than the slope of the u-nullcline: $-g_1/g_2 > -f_1/f_2$. Using this property it is straightforward to show that for sufficiently large ϵ the eigenvalues of Eq. (3) always have a negative real part. Moreover, by decreasing ϵ the eigenvalues will cross the imaginary axis so that the stable equilibrium point will give way to a stable limit cycle via Hopf bifurcation. This result is not model-specific. It applies to systems with qualitatively similar per-capita nullclines of prey and predator.

We note that the stabilizing effect of such predator interference has been fully explored for discrete-time models, the so-called Nicholson–Bailey type for biological control as well. See [12], also [22,13,20] for the modelling of intraspecific interference functional forms.

Appendix C. Biological control with alternative prey

Let Z be the alternative prey of predator Y, and assume Y feeds on X, Z indiscriminately. Then Holling's disc functions for the per-predator capture rates are $cX/(1 + chX + c_z h_z Z)$ on X and $c_z Z/(1 + chX + c_z h_z Z)$ on Z, with c_z, h_z being predator's discovery rate and handling time on Z respectively. The per-capita birth rate is $bcX/(1 + chX + c_z h_z Z) + b_z c_z Z/(1 + chX + c_z h_z Z)$ with b_z being predator's birth-to-consumption ratio on Z. Assuming Z is maintained at a constant level, then with proper regroup of parameters Eq. (1) takes the following form:

$$\begin{aligned} \dot{X} &= X\left[r\left(1 - \frac{X}{K}\right) - \frac{pY}{H + X}\right], \\ \dot{Y} &= Y\left[\frac{A + bpX}{H + X} - (d + sY)\right]. \end{aligned} \tag{4}$$

It is the same form as Eq. (1) except that the composition of H is expanded accordingly and that the inclusion of A is due to the constant supply from prey Z. The Y-intercept of the per-capita Y-nullcline is $\overline{Y} = (A/H - d)/s$. For $A/H - d > 0$, a Y-equilibrium point $(0, \overline{Y})$ is born. For $\overline{Y} = (A/H - d)/s > r/c$, the Y-intercept of the per-capita prey nullcline, the $(0, \overline{Y})$ equilibrium is always stable. Under the same condition and for small enough s, the per-capita nullclines for both prey and predator do not intersect, thus all solutions converge to $(0, \overline{Y})$, eliminating the prey. Also, using the same scalings as for Eq. (2), the equations are cast in the same form except for the y-equation:

$$\dot{y} = y\left[\frac{\alpha + x}{\beta + x} - (\delta + \sigma y)\right], \tag{5}$$

with $\alpha = A/(bpK)$.

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