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2019-11

Vitale , C & Kisdi , E 2019 , ' Evolutionary Suicide of Prey : Matsuda and Abrams' Model Revisited ' , Bulletin of Mathematical Biology , vol. 81 , no. 11 , pp. 4778-4802 . https://doi.org/10.1007/s11538-018-0

http://hdl.handle.net/10138/308749
https://doi.org/10.1007/s11538-018-0472-9

acceptedVersion

Evolutionary suicide of prey: Matsuda & Abrams' model revisited

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Abstract

Under the threat of predation, a species of prey can evolve to its own extinction. Matsuda and Abrams (1994, Theor. Pop. Biol. 45: 76-91) found the earliest example of evolutionary suicide by demonstrating that the foraging effort of prey can evolve until its population dynamics cross a fold bifurcation, whereupon the prey crashes to extinction. We extend this model in three directions. First, we use critical function analysis to show that extinction cannot happen via increasing foraging effort. Second, we extend the model to non-equilibrium systems and demonstrate evolutionary suicide at a fold bifurcation of limit cycles. Third, we relax a crucial assumption of the original model. To find evolutionary suicide, Matsuda and Abrams assumed a generalist predator, whose population size is fixed independently of the focal prey. We embed the original model into a 3-species community of the focal prey, the predator, and an alternative prey that can support the predator also alone, and investigate the effect of increasingly strong coupling between the focal prey and the predator's population dynamics. Our 3-species model exhibits (i) evolutionary suicide via a subcritical Hopf bifurcation and (ii) indirect evolutionary suicide, where the evolution of the focal prey first makes the community open to the invasion of the alternative prey, which in turn makes evolutionary suicide of the focal prey possible. These new phenomena highlight the importance of studying evolution in a broader community context.

Keywords: adaptive dynamics, evolutionary suicide, fold bifurcation of limit cycles, foraging effort, predator-prey model, saddle-node bifurcation, subcritical Hopf bifurcation

Mathematics Subject Classification: 92D15, 92D25, 92D50

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1 Introduction

In a seminal paper, Matsuda and Abrams (1994) showed that adaptive evolution of a prey species may lead to its own extinction in a process now known as evolutionary suicide (Gyllenberg and Parvinen 2001; Webb 2003). Here we revisit and generalize this model to relax some biologically restrictive assumptions, especially the assumption that the predator-prey system is isolated. Adding just one more species to the community, we find much richer dynamics and several possible routes to evolutionary suicide.

Evolutionary suicide may seem counter-intuitive; common wisdom dictates that natural selection will perfect a species rather than lead to its demise. The reason why evolution can lead to extinction lies in the interaction between the dynamics of population size and the dynamics of trait evolution. The population dynamics of a species depend on its traits (for example, on how much resources it can collect and how much it exposes itself to predators); in other words, a trait can be seen as a bifurcation parameter of population dynamics. Evolutionary suicide may happen when the evolving trait crosses a catastrophic bifurcation point of population dynamics, whereby the interior attractor of population dynamics disappears and the population goes extinct (Gyllenberg and Parvinen 2001; Webb 2003).

Matsuda and Abrams (1994) considered a population of prey consumed by a fixed number of predators with a Holling type II functional response. In this model, the key to the catastrophic bifurcation where evolutionary suicide happens is in the nonlinear dynamics generated by the functional response. The Holling II functional response derives from the saturation of the predators when the time needed to consume and digest an individual prey is non-negligible (Holling 1959). At high prey densities, capturing prey is easy, so that most predators are busy handling prey already captured; since only a few predators are searching for prey, the risk for an individual prey of being attacked is small. At low prey densities, however, capturing prey is hard, and therefore most predators are searching; for an individual prey, the risk of predation is high. Mortality from predation therefore decreases with increasing prey density. This can generate an Allee effect in the dynamics of the prey, such that for low to intermediate prey densities (i.e., before the Holling II function saturates), the *per capita* growth rate of the prey increases rather than decreases with prey density (see e.g. Thieme 2003, chapter 7).

In the model of Matsuda and Abrams (1994), the prey evolves its foraging effort (for example, the amount of time spent actively foraging). Higher foraging effort implies higher fecundity, but also higher exposure to predation. When the risk of predation is sufficiently high, a lower foraging effort is advantageous. As the prey evolves to be less active, it becomes harder for the predator to capture prey; more predators are thus searching, and this implies an even higher risk of predation. The equilibrium population density of prey thus declines. Due to the Allee effect induced by the Holling II functional response, the decline can be accelerating, and the prey can cross a fold bifurcation point of its population dynamics, whereby its population crashes to extinction.

The above scenario of evolutionary suicide is possible when the predator is a generalist, in the sense that its population is regulated by factors other than the evolving prey, so that its

density is a constant. To see this, Matsuda and Abrams (1994) formulated the model

$$\frac{dn}{dt} = \left[B(c) - d - \delta n - \frac{c\beta p}{1 + c\beta h n} \right] n \tag{1}$$

for the population dynamics of the prey, where n is its density and c is its foraging effort. The *per capita* birth rate of the prey, B, is an increasing function of its foraging effort,

$$B(c) = \frac{\rho c}{1 + \rho b c} \tag{2}$$

with positive parameters ρ and b. The prey is subject to death other than predation at a linearly density dependent rate $d + \delta n$. This implies that in absence of the predator and with a given trait value c, the prey follows the logistic model of population growth. The last term in the brackets is loss due to predation, where p is the constant density of predators, β is the capture rate and h is the predator's handling time. It is easy to see that with p and c fixed, the dynamics in (1) can have two nontrivial equilibria, i.e., a stable equilibrium and an unstable Allee threshold; the latter is due to the Allee effect induced by the Holling II functional response in the predation term (see above). The two nontrivial equilibria of the prey can disappear through a fold bifurcation when the foraging effort c varies. Matsuda and Abrams (1994) demonstrated by examples that the adaptive evolution of the foraging effort c can push the prey through the fold bifurcation point, whereupon the prey population goes extinct.

By contrast, if the predator is a specialist on the focal prey so that its reproduction solely depends on how much of the focal prey it consumes, then p is variable and the model becomes

$$\begin{aligned} \frac{dn}{dt} &= \left[B(c) - d - \delta n - \frac{c\beta p}{1 + c\beta h n} \right] n \\ \frac{dp}{dt} &= \left[\frac{\alpha c \beta n}{1 + c\beta h n} - \mu \right] p \end{aligned} \tag{3}$$

where α is the predator's conversion factor and μ is its death rate. This is the Rosenzweig-MacArthur predator-prey model, which is well known to have a unique nontrivial equilibrium (either a stable equilibrium or an unstable focus surrounded with a stable limit cycle). There is thus no fold bifurcation in this model, and evolutionary suicide is not possible (Matsuda and Abrams, 1994).

Evolutionary suicide in the prey therefore hinges upon the predator population size being "sufficiently" independent of the focal (evolving) prey. There may be several factors that keep the predator population approximately constant for a wide range of prey population sizes. In this paper, we extend the model of Matsuda and Abrams (1994) assuming that the predator has an alternative prey, which may maintain the predator population independently of the focal prey, or else may contribute less such that the focal prey becomes an important resource for the predator. In the limiting case when the predator depends only on the alternative prey, we recover the model in (1) with evolutionary suicide as described by Matsuda and Abrams (1994). By changing the relative contributions of the focal prey and of the alternative prey to the predator's birth rate, we can study a continuum of models between (1) and (3). Due to the joint dynamics of the alternative prey, we uncover also other ways to evolutionary suicide, such

as extinction via a subcritical Hopf bifurcation or a fold bifurcation of limit cycles.

In section 2, we describe our main model, which generalizes the model of Matsuda and Abrams (1994) in three ways: the birth rate need not be a hyperbolic function of the foraging effort c as assumed in (2); the dynamics of the predator need not be independent of the focal prey; and the system may exhibit nonequilibrium dynamics when evolutionary suicide happens. In section 3, we allow for an arbitrary birth rate function $B(c)$ in (1) and establish what conditions this function should fulfil to obtain evolutionary suicide at a given value of c. In section 4, we allow for nonequilibrium dynamics of the predator-alternative prey system and study evolutionary suicide in (1) with non-constant predator densities. In section 5, we explore evolutionary suicide in the full three-dimensional system of the focal prey, alternative prey and predator, assuming that the predator depends on both prey species.

2 The model

We extend the Rosenzweig-MacArthur model in (3) to include an alternative prey, assuming that the predator hunts for only one prey species at a time (see the Discussion for the opposite case). This happens if the focal and alternative prey species live in two different habitats, and the predator spends a fraction q of its time in the first habitat, searching for and handling only the focal prey, and spends the remaining fraction $(1-q)$ of its time in the second habitat, searching for and handling only the alternative prey. The same split of the predator's time results also if the two prey species have different diurnal patterns of activity, so that the focal prey is available for a fraction q of the predator's active time and the alternative prey is available during the remaining fraction $1-q$, or if individual predators have different non-heritable preferences such that a fixed fraction q of the predators hunt for the focal prey and the rest for the alternative prey. With this assumption, we obtain the system

$$\frac{dn_1}{dt} = \left[B(c_1) - d_1 - \delta_1 n_1 - \frac{c_1\beta_1 qp}{1+c_1\beta_1 h_1 n_1}\right] n_1 \tag{4a}$$

$$\frac{dn_2}{dt} = \left[\frac{\rho_2 c_2}{1+\rho_2 b_2 c_2} - d_2 - \delta_2 n_2 - \frac{c_2\beta_2(1-q)p}{1+c_2\beta_2 h_2 n_2}\right] n_2 \tag{4b}$$

$$\frac{dp}{dt} = \left[\frac{\alpha_1 q c_1\beta_1 n_1}{1+c_1\beta_1 h_1 n_1} + \frac{\alpha_2(1-q)c_2\beta_2 n_2}{1+c_2\beta_2 h_2 n_2} - \mu\right] p \tag{4c}$$

where the notation is as in the Introduction, only now specific to the focal prey species 1 and to the alternative prey species 2. For the focal prey, we shall use the hyperbolic birth rate function

$$B(c_1) = \frac{\rho_1 c_1}{1+\rho_1 b_1 c_1} \tag{5}$$

when we resort to numerical analyses (Matsuda and Abrams 1994 used the same but with $\rho_1 = 1$ fixed). For the alternative prey, we always use the hyperbolic birth rate function already substituted into equation (4b). Since we treat the foraging effort of the alternative prey c_2 as a constant model parameter, using the hyperbolic formula for the birth rate implies that the birth rate of species 2 is a constant that falls between 0 and $1/b_2$.

The population dynamics of this system have been studied by Krivan and Eisner (2006, see their "fixed preference" model) and by Vitale (2016). The system can have up to three interior equilibria (Appendix A), two of which can be asymptotically stable, as well as limit cycles. We have no evidence for chaotic dynamics, but given the high number of parameters and lack of comprehensive analysis, we cannot exclude it either. Related models that include also competition between the two prey species exhibit chaos (Bazykin 1998; Groll et al. 2017).

To investigate the evolution of the foraging effort c_1 of the focal prey, consider a rare mutant with foraging effort c_1^m. If the resident system (4) has attained a stable equilibrium $(\bar{n}_1, \bar{n}_2, \bar{p})$, then the mutant, while rare, grows or declines exponentially at the rate

$$r(c_1^m; \bar{n}_1, \bar{p}) = B(c_1^m) - d_1 - \delta_1 \bar{n}_1 - \frac{c_1^m \beta_1 q \bar{p}}{1 + c_1 \beta_1 h_1 \bar{n}_1}$$

which we refer to as the invasion fitness of the mutant. Notice the resident foraging effort c_1 in the denominator of the last term. The factor $1/(1 + c_1\beta_1 h_1 \bar{n}_1)$ in the Holling II functional response is the fraction of predators who are searching rather than handling a prey, and this fraction is determined by the resident population of the focal prey. We follow Matsuda and Abrams (1994) in assuming that mortality other than predation occurs at the same rate $(d_1 + \delta_1 \bar{n}_1)$ for the mutant as for the resident.

The invasion fitness at $c_1^m = c_1$ is the resident's growth rate, which is zero. A first-order Taylor expansion of the invasion fitness as a function of c_1^m around $c_1^m = c_1$ shows that a mutant with foraging effort c_1^m sufficiently close to c_1 will invade the resident system if the selection gradient

$$g(c_1) = \left. \frac{\partial r(c_1^m; \bar{n}_1, \bar{p})}{\partial c_1^m} \right|_{c_1^m = c_1} = B'(c_1) - \frac{\beta_1 q \bar{p}}{1 + c_1 \beta_1 h_1 \bar{n}_1} \tag{6a}$$

has the same sign as $(c_1^m - c_1)$. If the resident system has settled on a stable limit cycle, then the invasion fitness of a mutant is the time average of its growth rate over a full cycle of the resident system, and the selection gradient is

$$g(c_1) = B'(c_1) - \frac{1}{T} \int_0^T \frac{\beta_1 q p(t)}{1 + c_1 \beta_1 h_1 n_1(t)} dt \tag{6b}$$

where T is the period of the cycle.

If $|c_1^m - c_1|$ is sufficiently small and c_1 is away from evolutionary singularities (defined by $g(c_1) = 0$), invasion implies fixation, i.e., an invading mutant excludes the former resident and becomes the new resident (Geritz 2005; Dercole and Rinaldi 2008; Dercole and Geritz 2016). By repeated steps of mutation, invasion and fixation, the foraging effort c_1 evolves towards higher or lower values depending on whether the selection gradient $g(c_1)$ is positive or negative (Dieckmann and Law 1996; Geritz et al. 1998). Note that if the resident population has multiple attractors of its population dynamics, the selection gradient in (6) depends on at which equilibrium or limit cycle we evaluate it. The "Tube Theorem" of Geritz et al. (2002) ensures that with small mutations, the population tracks one branch of equilibria or limit cycles while evolving c_1, as long as no catastrophic bifurcation of the population dynamics is encountered. In all cases we consider below, the resident population has only one attractor with the focal species present at

positive population densities, but it may have another attractor on the boundary with the focal species extinct. We evaluate $g(c_1)$ at the attractor where the focal species is present without making this explicit in the notation. The adaptive dynamics of c_1 described here agree with the adaptive dynamics used by Matsuda and Abrams (1994, see their equation 6), which is based on quantitative genetics (Abrams et al. 1993 and references therein discuss the relationship between adaptive dynamics and quantitative genetic models of evolution).

The limiting case of Matsuda and Abrams

In order to recover the "generalist predator" model of Matsuda and Abrams (1994), we need to assume that the predator's dynamics are independent of the focal prey (i.e., the predator is sustained solely by the alternative prey), but the predator does impact on the focal prey. In our model, this is the case when $\alpha_1 = 0$, i.e., when consuming the focal prey does not contribute to the reproduction of the predator. Biologically this limiting case is unrealistic, but this is how our extended model connects to the original model of Matsuda and Abrams (1994). Because evolutionary suicide is a structurally robust phenomenon, our predictions hold also for small positive values of α_1. We consider the degree of robustness in section 5.

With $\alpha_1 = 0$, the 2-dimensional system (4b,c) of the predator and the alternative prey is autonomous, and it is the well-known Rosenzweig-MacArthur predator-prey model of a logistic prey and a predator with Holling II functional response. Suppose that the predator is viable, and the unique interior equilibrium $(\bar{n}_2, \bar{p})$ of the Rosenzweig-MacArthur model in (4b,c) is stable. Once the predator has equilibrated, $p = \bar{p}$ in (4a) is fixed, and for the focal prey, we recover the "generalist predator" model of Matsuda and Abrams (1994; see equation (1) above). The two nontrivial equilibria of (1) correspond to two interior equilibria of the 3-dimensional system in (4), which have the same coordinates for the predator and the alternative prey; depending on whether (4b,c) has a stable node or focus, the equilibria of (4) are a stable node or a stable focus-node and a saddle or a saddle-focus.

Figure 1 illustrates the adaptive dynamics of the foraging effort c_1 with $\alpha_1 = 0$, $p = \bar{p}$ constant and $B(c_1)$ as given in (5). In panels (a)-(e), we assume the same parameter values for the focal species as Matsuda and Abrams (1994) did, and vary the effective predator density $q\bar{p}$ experienced by the focal species via varying c_2; panels (b) and (e) are identical to Figure 1b,d in Matsuda and Abrams (1994).

As the foraging effort c_1 evolves, the system tracks the stable interior equilibrium as long as it exists (Geritz et al. 2002). Evolutionary suicide happens when c_1 evolves downwards (in the dark grey regions in Figure 1) to cross a saddle-node bifurcation point (marked with arrows in Figure 1a-c,f). When a mutant with foraging effort below the bifurcation point invades, the entire population of the focal species crashes to extinction. Note that a saddle-node bifurcation does not necessarily imply evolutionary suicide; in Figure 1g, the selection gradient is positive such that c_1 evolves away from the bifurcation point. Next to evolutionary suicide, the model can also exhibit attracting singularities (filled dots), evolutionary repellors (empty dots), and bistability of the adaptive dynamics (in Figure 1d, between two attracting singularities; in Figure 1f, between an attracting singularity and evolutionary suicide). Since $[\partial^2 r/\partial(c_1^m)^2]_{c_1^m=c_1} = B''(c_1) < 0$ with

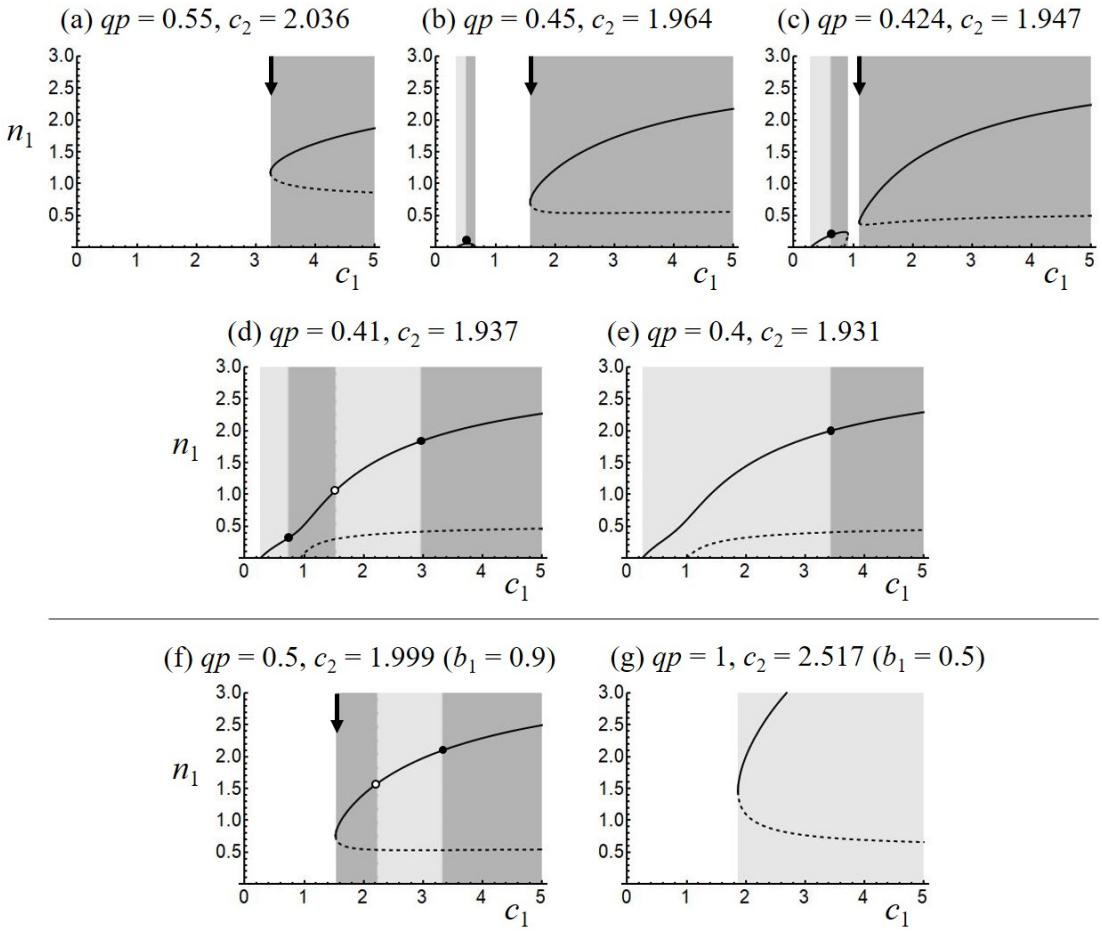

Figure 1: Bifurcation diagrams in the limiting case of Matsuda and Abrams (1994). The plots show the density of the focal species $\bar{n}_1$ as a function of its evolving trait c_1. Because $\alpha_1 = 0$, $\bar{n}_2$ and $\bar{p}$ do not depend on c_1. Thick lines: stable equilibria; dashed lines: unstable equilibria; dark grey background: the selection gradient of c_1 is negative at the stable equilibrium; light grey background: the selection gradient is positive. Filled dots mark convergence stable ESSs, empty dots are evolutionary repellors. Arrows indicate the fold bifurcations where evolutionary suicide occurs (panels (a)-(c) and (f)). $B(c_1)$ as in (5). Parameter values: $d_1 = 0.1$, $\delta_1 = 0.25$, $\rho_1 = 1$, $\beta_1 = 1$, $h_1 = 1$, $d_2 = 0.9$, $\delta_2 = 0.01$, $b_2 = 0.4$, $\rho_2 = 0.9$, $\beta_2 = 0.9$, $h_2 = 0$, $\alpha_2 = 0.6$, $\mu = 0.5$, $q = 0.9$ and (a)-(e) $b_1 = 1$, (f) $b_1 = 0.9$, (g) $b_1 = 0.5$; the value of c_2, given in the panel titles, is chosen to obtain the values of $q\bar{p}$ shown in the panels.

the birth rate function in (5), the singularities are evolutionarily stable (ESS; Maynard Smith 1982; Geritz et al. 1998).

3 Evolutionary suicide through a saddle-node bifurcation: Critical function analysis

In this section, we start with a non-specified birth rate function $c_1 \mapsto B(c_1)$ for the focal species, and ask what properties this function should have for the model to exhibit evolutionary suicide at a given trait value c_1^*. Starting with a non-specified birth rate function is motivated by the fact that the choice of this function is the least justified element of the model. The hyperbolic function in (5) is not derived from any underlying mechanism (in contrast to the Holling II functional response of the predators, which derives from switching between searching and handling), and while it is biologically realistic to assume that the birth rate is an increasing and saturating function of the foraging effort, the particular shape of this function could well be different. The following analysis is similar in spirit to the method of critical function analysis, which is used to construct trade-off functions to obtain evolutionary branching points and other types of evolutionary singularities (de Mazancourt and Dieckmann 2004; Bowers et al. 2005; Geritz et al. 2007; Kisdi 2006, 2015; Kisdi et al. 2013). Here we apply this construction method for the first time to find evolutionary suicide.

Throughout this section, we assume $\alpha_1 = 0$ so that that the dynamics of the predator and its alternative prey in (4b,c) are decoupled from the dynamics of the focal prey in (4a). Suppose that all parameters in (4b,c) are fixed such that the Rosenzweig-MacArtur system of the alternative prey and the predator attains a stable interior equilibrium $(\bar{n}_2, \bar{p})$. From (4a), the equilibrium density of the focal species is given by

$$\bar{n}_1^{\pm} = \frac{\Gamma \pm \sqrt{\Delta}}{2\delta_1 c_1 \beta_1 h_1}$$

with $\Gamma = c_1\beta_1 h_1(B(c_1) - d_1) - \delta_1$ and $\Delta = \Gamma^2 + 4\delta_1(\Gamma + \delta_1 - c_1^2\beta_1^2 h_1 q\bar{p})$. A saddle-node bifurcation of equilibria occurs when the discriminant Δ is zero, which is the case if Γ takes one of the two values $\Gamma^{\pm} = -2\delta_1 \pm 2c_1\beta_1\sqrt{\delta_1 h_1 q\bar{p}}$. Notice that the smaller root Γ^- is always negative. For the saddle-node bifurcation to happen at a given foraging effort $c_1 = c_1^*$ and at a positive density $\bar{n}_1^* = \Gamma^+/(2\delta_1 c_1 \beta_1 h_1) > 0$, it is necessary that Γ^+ is positive and $B(c_1^*)$ is such that $\Gamma = \Gamma^+$. These two conditions are equivalent to

$$(c_1^*)^2 \beta_1^2 h_1 q\bar{p} > \delta_1 \tag{7}$$

and

$$B(c_1^*) = \frac{\Gamma^+ + \delta_1}{c_1^*\beta_1 h_1} + d_1 = 2\sqrt{\frac{\delta_1 q\bar{p}}{h_1}} - \frac{\delta_1}{c_1^*\beta_1 h_1} + d_1 \tag{8}$$

respectively (note that by (7), the birth rate in (8) is always positive).

The necessary condition in (7) is independent of the choice of the birth rate function B. For fixed model parameters, it implies that evolutionary suicide is possible only if the foraging

effort c_1^* is chosen to be sufficiently high. Alternatively, one could fix c_1^* first, and choose the parameters of the alternative prey and the predator (i.e., the parameters in (4b,c)) such that $\bar{p}$ is sufficiently high to satisfy condition (7). The well-known properties of the Rosenzweig-MacArthur predator-prey model ensure that it is always possible to find parameters to get a stable equilibrium with arbitrary predator density $\bar{p}$. Biologically, (7) shows that at a given foraging effort c_1^*, evolutionary suicide is not possible if the density of predators is too low. Further, evolutionary suicide is never possible if there is no handling time for the focal prey ($h_1 = 0$). If the focal prey cannot saturate the predator, then there is no Allee effect in its dynamics and therefore bistability and evolutionary suicide are not possible.

We say that the saddle-node bifurcation opens to the right if the two interior equilibria exist for $c_1 > c_1^*$. This is the case when

$$\left.\frac{\partial \Delta}{\partial c_1}\right|_{c_1=c_1^*} > 0$$

Using (8), this condition is equivalent to

$$\frac{1}{\beta_1}\frac{\delta_1}{(c_1^*)^2 h_1} < B'(c_1^*) \tag{9}$$

If the opposite strict inequality holds, then the saddle-node bifurcation opens to the left (i.e., two interior equilibria exist for for $c_1 < c_1^*$).

Recall that to ensure a saddle-node bifurcation at c_1^*, $\Delta = 0$ is not sufficient in itself, two non-degeneracy conditions must also be satisfied (Kuznetsov 1998, Theorem 3.1). Both involve derivatives of the right hand side of (4a), evaluated at $(c_1, n_1) = (c_1^*, \bar{n}_1^*)$. First, the second derivative with respect to n_1 must not vanish; it is straightforward to show that this is ensured by (7). Second, the derivative with respect to c_1 must not be zero; this is equivalent to requiring that (9) does not hold with equality.

The selection gradient at the bifurcation point, given by (6a) evaluated at $(c_1, \bar{n}_1) = (c_1^*, \bar{n}_1^*)$, is negative if and only if

$$B'(c_1^*) < \sqrt{q\bar{p}\frac{\delta_1}{(c_1^*)^2 h_1}} \tag{10}$$

Assuming that (7) holds, one can obtain evolutionary suicide at a given trait value c_1^* by choosing the birth rate function such that its value $B(c_1^*)$ is as given in (8) and its slope $B'(c_1^*)$ satisfies (9) and (10). In this case, there is a right-opening saddle-node bifurcation at c_1^* combined with a negative selection gradient at the bifurcation point, and, by continuity, also in the vicinity of the bifurcation point. The negative selection gradient drives the evolution of c_1 downwards until evolutionary suicide occurs at c_1^* (similarly to the examples in Figure 1a-c,f). The choice prescribed by (9) and (10) is always possible because the right hand side of (10) is greater than the left hand side of (9), i.e.,

$$\sqrt{q\bar{p}\frac{\delta_1}{(c_1^*)^2 h_1}} - \frac{1}{\beta_1}\frac{\delta_1}{(c_1^*)^2 h_1} > 0 \tag{11}$$

holds precisely when (7) is satisfied.

Notice that the required slope of the birth rate function is positive, which is biologically realistic. The width of the interval of $B'(c_1^*)$ which results in evolutionary suicide is given by the left hand side of (11) (see Appendix B for details). Since evolutionary suicide is a non-degenerate phenomenon, qualitatively similar results hold by continuity also for small positive values of α_1.

The above construction method prescribes the birth rate function B only locally, up to first order at c_1^*; otherwise the function can be chosen arbitrarily, with biological realism in mind (e.g. a monotonically increasing, saturating function). For example, the two-parameter hyperbolic function in (5) is sufficiently flexible to satisfy the conditions obtained for evolutionary suicide at c_1^*, but so are many other functions.

One can easily construct a birth rate function such that the model exhibits a left-opening saddle-node bifurcation at a given foraging effort c_1^*, but evolutionary suicide is not possible at such a point. Figure 1c shows an example; there is a left-opening saddle-node bifurcation at c_1 slightly below 1, but the selection gradient is negative at this point, so that the focal prey evolves away from extinction. To prove that this is always the case, recall that for a left-opening saddle-node bifurcation at a positive density $\bar{n}_1^*$, (7) and (8) must hold together with the opposite of (9). For the selection gradient at c_1^* to be positive, which would drive the focal species through the saddle-node bifurcation to extinction, the opposite of (10) should hold. For the opposites of (9) and (10) to be true simultaneously, the opposite of (11) is necessary; but this contradicts (7). Hence in the limiting case of the "generalist predator" model of Matsuda and Abrams (1994), evolutionary suicide can never happen due to evolving higher values of the foraging effort c_1, no matter what the birth rate function is.

4 Evolutionary suicide through a fold bifurcation of limit cycles

Since evolutionary suicide is a structurally stable phenomenon, it should occur also if the density of predators is not constant but has some small temporal variation. In the context of our model, the equilibrium that undergoes a saddle-node bifurcation at the point of evolutionary suicide can be replaced with a limit cycle, so that evolutionary suicide happens through a fold bifurcation of limit cycles.

To obtain an example for this, we can use the construction method of the previous section combined with a continuity argument. Throughout this section, we assume $\alpha_1 = 0$. In the first step, we consider the autonomous planar system of (4b,c), i.e., the Rosenzweig-MacArthur model of the predator and its alternative prey, and poise it at a Hopf bifurcation point as follows. Fix all parameters in equations (4b,c) except c_2. The birth rate of the alternative prey is an increasing function of its foraging effort c_2, and hence the predator-free equilibrium density of the prey increases with c_2. The predator needs a minimum density of the prey to be viable, i.e., c_2 needs to exceed a certain threshold $\tilde{c}_2$ for a positive interior equilibrium to exist (the expression for $\tilde{c}_2$ is easy to derive but is relatively lengthy). Let $\operatorname{tr} J$ denote the trace of the 2×2 Jacobian matrix evaluated at the unique interior equilibrium of (4b,c); by the Routh-Hurwitz criteria, the

Hopf bifurcation occurs when $\operatorname{tr} J = 0$. With c_2 only slightly above the transcritical bifurcation point $\tilde{c}_2$, the equilibrium must be a stable node and therefore $\operatorname{tr} J$ is negative. Further,

$$\frac{d \operatorname{tr} J}{dc_2} = \frac{\rho_2 h_2 \mu}{\alpha_2(1-q)(1+\rho_2 b_2 c_2)^2} + \frac{\delta_2 \mu[\alpha_2(1-q)+h_2\mu]}{\alpha_2\beta_2 c_2^2(1-q)[\alpha_2(1-q)-h_2\mu]} > 0$$

since $\alpha_2(1-q) > h_2\mu$ is necessary for predator viability and

$$\lim_{c_2\to\infty} \operatorname{tr} J = \frac{h_2\mu(1-b_2d_2)}{\alpha_2 b_2(1-q)} > 0$$

whenever the handling time h_2 is nonzero since $b_2d_2 < 1$ is necessary for the viability of the prey. It thus follows that there is a unique $c_2 = c_2^H > \tilde{c}_2$ where $\operatorname{tr} J = 0$ and the system undergoes a Hopf bifurcation. The Hopf bifurcation of the Rosenzweig-MacArthur model is known to be supercritical, such that a stable limit cycle of the alternative prey-predator system is present when c_2 exceeds c_2^H.

In the second step, we take $c_2 = c_2^H$ and use section 3 to obtain evolutionary suicide of the focal prey. Let $\bar{p}^H$ denote the equilibrium predator density at $c_2 = c_2^H$. Fix the values of d_1, δ_1, β_1 and h_1 in equation (4a). Choose c_1^* such that (7) holds with $\bar{p} = \bar{p}^H$. With the parameter values used in Figure 2, $c_2^H = 2.128$, $\bar{p}^H = 1.187$, and (7) is equivalent to $c_1^* > 0.4478$; we set $c_1^* = 2.1$. From (8), we obtain the value $B(c_1^*) = 0.67$. Conditions (9) and (10) constrain $B'(c_1^*)$ to be between 0.0324 and 0.1519; we choose the slope $B'(c_1^*) = 0.1$. The birth rate function in (5) has the required value $B(c_1^*) = 0.67$ and slope $B'(c_1^*) = 0.1$ if its parameters are $\rho_1 = 1.018$ and $b_1 = 1.025$. With the parameters obtained at this point, the focal species undergoes evolutionary suicide through a collision of two non-hyperbolic equilibria.

In the last step, we increase the value of c_2 to exceed the supercritical Hopf bifurcation point c_2^H. The two interior non-hyperbolic equilibria are then replaced with two limit cycles, which are stable when n_1 is fixed, but one is attracting and the other is repelling in the n_1-direction. By continuity, these limit cycles are near the original equilibria, so that for a sufficiently small change in c_2, a fold bifurcation of limit cycles takes place near c_1^* and the sign of the selection gradient remains unchanged. Thus we obtain evolutionary suicide through a fold bifurcation of limit cycles.

In Figure 2 illustrating this result, we have also rounded off the parameters of the birth rate function to $\rho_1 = 1$ and $b_1 = 1$; due to continuity, this does not alter the outcome. The selection gradient of c_1, obtained from (6b), is negative on the stable limit cycle for all values of c_1 shown in Figure 2. Hence evolution will drive c_1 downwards until the population of the focal species collapses at the fold bifurcation of limit cycles at around $c_1 = 2.16$. By continuity, the same must happen also for small positive values of α_1, i.e., if the predator is not fully decoupled from the focal prey's dynamics.

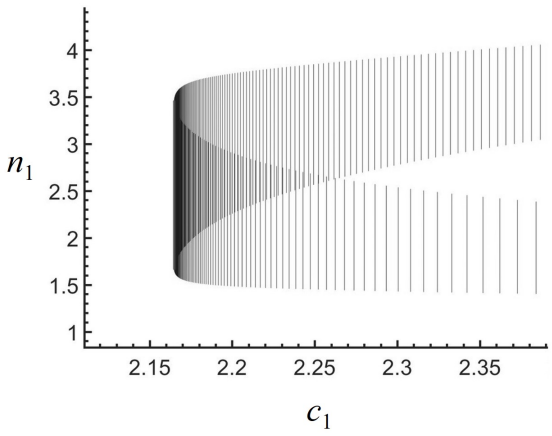

Figure 2: Evolutionary suicide through a fold bifurcation of limit cycles. Each vertical line represents the range of n_1 along a limit cycle. For each value of c_1 above the fold bifurcation of limit cycles at ca $c_1 = 2.16$, there exist a stable limit cycle (upper branch) and an unstable limit cycle (lower branch); the corresponding lines are shown at slightly different values of c_1 for readability. The selection gradient is negative on the upper branch of stable limit cycles. Parameter values: $d_1 = 0.1, \delta_1 = 0.1, b_1 = 1, \rho_1 = 1, \beta_1 = 1, h_1 = 0.7, d_2 = 0.9, \delta_2 = 0.01, b_2 = 0.4, \rho_2 = 0.9, \beta_2 = 0.9, h_2 = 0.5, \alpha_1 = 0, \alpha_2 = 0.7, \mu = 0.5, q = 0.6, c_2 = 2.13$.

5 Robustness and evolutionary suicide through a subcritical Hopf bifurcation

In this section we consider the model with $\alpha_1 > 0$, so that all three equations in (4) are coupled. The full model has very rich population dynamics (see Krivan and Eisner 2006; Vitale 2016). We do not aim at a comprehensive analysis; rather, we show two examples to illustrate robustness issues and various routes to evolutionary suicide. The numerical bifurcation analysis presented below was carried out using MatCont (Dhooge et al. 2008).

Example 1

In our first example, we assume that both prey species are associated with a positive handling time ($h_1, h_2 > 0$), but the parameters are such that the 2-species Rosenzweig-MacArthur model of the alternative prey and the predator has a stable equilibrium. We take the birth rate function of the focal prey as given in (5) and choose parameter values such that at $\alpha_1 = 0$, and by continuity also at sufficiently small values of α_1, the first prey species exhibits evolutionary suicide by evolving lower values of c_1 until the stable interior equilibrium disappears through a saddle-node bifurcation. At $\alpha_1 = 0$, this recapitulates the evolutionary suicide scenario found by Matsuda and Abrams (1994; cf. section 2).

In Figure 3, we show what happens as α_1 increases (the predator can better utilize the focal prey). In this figure, the selection gradient of the focal prey's foraging effort (evaluated numerically) is negative wherever the focal prey persists (either at a stable equilibrium or on a stable limit cycle). The evolutionary trajectory is therefore a straight line downwards to lower c_1. At

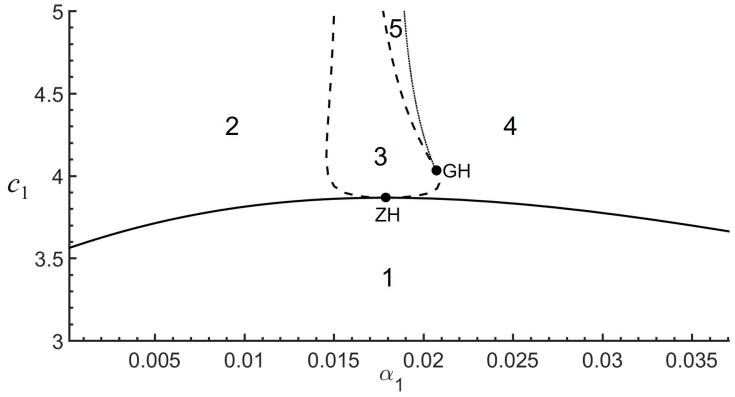

Figure 3: Bifurcation diagram for Example 1. Solid line: saddle-node bifurcation (a stable focus-node and a saddle-focus collide left to ZH, a saddle-focus and an unstable focus-node collide right to ZH); dashed line: Hopf bifurcation; dotted line: fold bifurcation of limit cycles; ZH: zero-Hopf bifurcation; GH: generalized Hopf bifurcation. Numbered regions: see text for description. Parameter values: $d_1 = 0.1, \delta_1 = 0.25, b_1 = 1, \rho_1 = 1, \beta_1 = 1, h_1 = 1, d_2 = 0.9, \delta_2 = 0.01, b_2 = 0.4, \rho_2 = 0.9, \beta_2 = 0.9, h_2 = 0.5, \alpha_2 = 0.7, \mu = 0.5, q = 0.6, c_2 = 2.06$.

low values of c_1 (in region 1 of Figure 3), there is no interior equilibrium. Given the values of the fixed parameters, the focal species cannot invade the 2-species system of the alternative prey and the predator with any $c_1 > 0$, i.e., the equilibrium on the boundary $n_1 = 0$ is always stable also transversally; and for the values of α_1 shown in Figure 3, the predator is not viable in absence of the alternative prey, such that the 2-species system of the focal prey and the predator has no positive equilibrium. In region 1, therefore, the only stable equilibrium is the boundary equilibrium where the focal species is extinct, and this equilibrium attracts all orbits. Near $\alpha_1 = 0$, evolutionary suicide happens as c_1 evolves downwards and crosses the saddle-node bifurcation line (solid line in Figure 3) to region 1.

In region 2, the model has a pair of stable and unstable interior equilibria. Near $\alpha_1 = 0$, these equlibria are a node and a saddle (all eigenvalues of the Jacobian are real), but with increasing α_1, they turn into a stable focus-node and a saddle-focus (both have two complex eigenvalues with negative real parts, the third eigenvalue is real and negative for the focus-node and positive for the saddle-focus). With increasing α_1, the saddle-focus undergoes a Hopf bifurcation (dashed line between regions 2 and 3), whereby an unstable limit cycle is born (the first Lyapunov coefficient of the Hopf bifurcation is negative, yet the limit cycle is unstable because of the positive eigenvalue of the saddle-focus). The stable focus-node is still present in region 3, along with an unstable focus-node and the unstable limit cycle.

The Hopf and saddle-node bifurcation lines are tangent at the point of the zero-Hopf bifurcation (of the kind with $s = 1$, $\theta(0) > 0$ using the notation of Chapter 8.5 in Kuznetsov 1998; see his figure 8.13). To the right of the zero-Hopf point, the subcritical Hopf bifurcation (now with a positive first Lyapunov coefficient) involves the stable focus-node of region 3, turning it into a saddle-focus (with a real negative eigenvalue and a complex conjugate pair with positive real part). Hence in region 4, there is no stable equilibrium and all orbits are attracted to the

boundary equilibrium where the focal species is extinct.

Slightly above $c_1 = 4$, the right branch of the Hopf bifurcation line turns into supercritical at the point of a generalized Hopf bifurcation. When crossing from region 3 to region 5, a stable interior limit cycle is born as the stable focus-node of region 3 is destabilized. The stable limit cycle coexists in region 5 with the unstable limit cycle already present in region 3, and they disappear through the fold bifurcation of limit cycles that connects to the generalized Hopf point (dotted line between regions 4 and 5).

Recall that the selection gradient is everywhere negative. As c_1 evolves downwards, evolutionary suicide happens either via a saddle node-bifurcation (crossing from region 2 to 1) or via a subcritical Hopf bifurcation (from region 3 to 4). Starting with a limit cycle in region 5, the evolution of c_1 first stabilizes the population dynamics (crossing to region 3), and then leads to evolutionary suicide via the subcritical Hopf bifurcation (crossing to region 4).

In this example, the scenario of evolutionary suicide found at $\alpha_1 = 0$ (which corresponds to Matsuda and Abrams 1994) is not robust. At quite small positive values of α_1, suicide via a saddle-node bifurcation is replaced by suicide via a subcritical Hopf bifurcation. The latter however exists only in a narrow range of α_1, and if α_1 is increased further, the focal species is not viable for any foraging effort c_1. Note that this non-robustness is invisible if the analysis assumes a constant predator density. With $\alpha_1 = 0$, the bifurcation diagram with respect to c_1 looks the same as Figure 1a.

Example 2

In Figure 4, we show an example where evolutionary suicide robustly extends to positive values of α_1 (the only difference to the previous example is in the choice of the fixed parameters). This example also shows a way how the "generalist predator" model of Matsuda and Abrams (1994), with evolutionary suicide, connects through a continuous change of parameters to their "specialist predator" model, where evolutionary suicide is not possible.

At $\alpha_1 = 0$, the example in Figure 4 is precisely the same as Figure 1b above and Figure 1b of Matsuda and Abrams (1994). In addition, we have $h_2 = 0$ in this example, so that the alternative prey-predator system has no limit cycle.

As before, near $\alpha_1 = 0$ and starting with high foraging effort, c_1 decreases until evolutionary suicide occurs at a saddle-node bifurcation (solid line extending to $\alpha_1 = 0$ in Figure 4). There is a narrow interval of low values of c_1, between two horizontal dotted lines of transcritical bifurcations, where the focal species is viable and has an attracting ESS (marked with a dot in Figure 1b). This interval remains the same for all α_1, because at the transcritical bifurcations n_1 becomes zero, so that the product $\alpha_1 n_1$ in equation (4c), the only equation that contains α_1, is zero independently of α_1. The position of the singularity (the borderline between areas of positive and negative selection gradients between the dotted lines) depends on α_1 too weakly to be visible in Figure 4. If the initial value of c_1 is within this narrow interval, then it evolves to the singularity within the interval and evolutionary suicide does not occur.

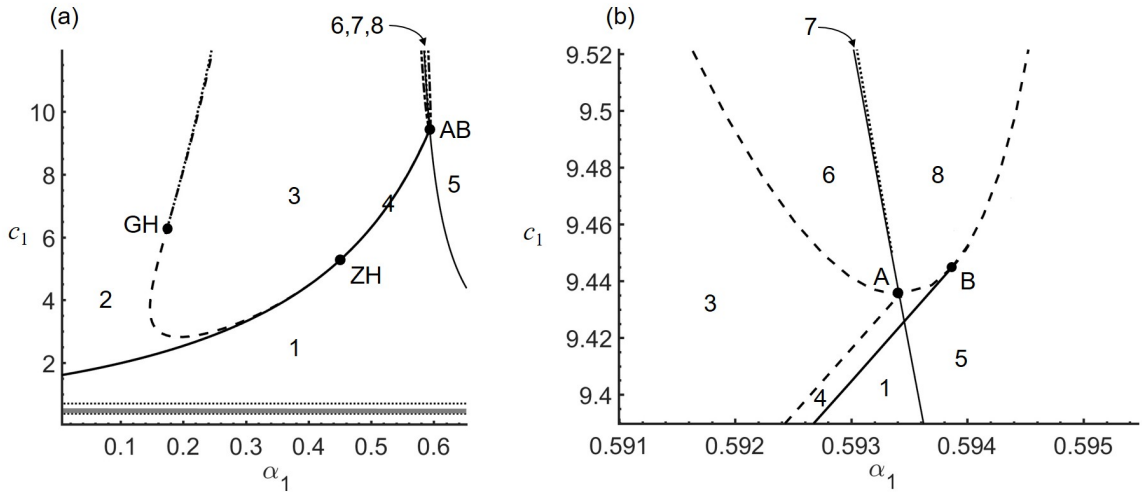

Figure 4: Bifurcation diagram for Example 2. (a) Main diagram; (b) the vicinity of AB magnified. Solid line through ZH: saddle-node bifurcation (a stable focus-node and a saddle-focus collide left to ZH, a saddle-focus and an unstable focus-node collide right to ZH); dashed line through GH: Hopf bifurcation; dotted line connecting to GH and running close above the Hopf bifurcation line: fold bifurcation of two unstable limit cycles; GH: generalized Hopf bifurcation; ZH: zero-Hopf bifurcation; horizontal dotted lines: transcritical bifurcations. Except for the grey area between the horizontal dotted lines, the selection gradient of c_1 is negative whenever the focal prey is present. Features resolved in panel (b), dashed curve delineating regions 6-8: transcritical bifurcation of interior equilibria crossing the equilibrium on the boundary $n_2 = 0$; solid line through A: supercritical Hopf bifurcation of the two-dimensional system restricted to the boundary $n_2 = 0$, with a limit cycle on the boundary on the right; dotted line connecting to A: transcritical bifurcation of the boundary limit cycle, transversally unstable on the right. Point A: the Hopf bifurcation of the interior equilibrium happens on the boundary $n_2 = 0$; B: the saddle-node bifurcation happens on the boundary $n_2 = 0$. The bifurcation lines are not drawn where the corresponding equilibria are outside the positive orthant. Numbered regions: see text for description. Parameter values: $d_1 = 0.1, \delta_1 = 0.25, b_1 = 1, \rho_1 = 1, \beta_1 = 1, h_1 = 1, d_2 = 0.9, \delta_2 = 0.01, b_2 = 0.4, \rho_2 = 0.9, \beta_2 = 0.9, h_2 = 0, \alpha_2 = 0.6, \mu = 0.5, q = 0.9, c_2 = 1.96417$ (this choice ensures $q\bar{p} = 0.45$ at $\alpha_1 = 0$).

The equilibria of regions 1-4 in Figure 4, organized by the zero-Hopf bifurcation point, are similar to the corresponding regions of Figure 3. In region 1, there is no interior equilibrium; in region 2, there is one stable and one unstable interior equilibrium; when crossing to region 3, the unstable saddle-focus of region 2 becomes an unstable focus-node, but the stable equilibrium does not change. In the example of Figure 4, the generalized Hopf bifurcation is on the left branch of the Hopf bifurcation line, so that it involves the saddle-focus of region 2, not the stable equilibrium. All limit cycles associated with the Hopf bifurcation left to the zero-Hopf bifurcation point are unstable. Region 4 is very narrow, but the subcritical Hopf bifurcation line is above the saddle-node line (see panel (b)).

Regions 2 and 3 are the parts where the focal species can be present at a stable interior equilibrium. The selection gradient of c_1 is negative everywhere in these regions, so that c_1 evolves downwards. For α_1 below ca 0.45, i.e., to the left of the zero-Hopf bifurcation, evolutionary suicide happens when c_1 crosses the saddle-node bifurcation line to region 1. For greater α_1, evolutionary suicide happens through the subcritical Hopf bifurcation between regions 3 and 4.

For higher values of α_1, the focal species alone can maintain a sufficiently large population of the predator to drive the alternative prey extinct. The line between regions 3 and 6 (see Figure 4b) is the line of a transcritical bifurcation, where the stable interior equilibrium leaves the positive orthant through the equilibrium of the focal prey and the predator on the boundary plane $n_2 = 0$. In region 6, the boundary equilibrium $(\bar{n}_1, 0, \bar{p})$ is stable both transversally and with respect to perturbations of n_1 and p. Note that since $h_2 = 0$, the boundary equilibrium with the alternative prey, $(0, \bar{n}_2, \bar{p})$, is always stable with respect to perturbations of n_2 and p; and it is also transversally stable except at very low c_1, between the two horizontal lines of transcritical bifurcations described above. Therefore in region 6, the model exhibits bistability with two stable boundary equilibria where one or the other prey is extinct.

On the boundary $n_2 = 0$, the focal prey and the predator follow the Rosenzweig-MacArthur model. The boundary equilibrium $(\bar{n}_1, 0, \bar{p})$ undergoes a supercritical Hopf bifurcation between regions 6 and 7. The limit cycle born in the bifurcation is transversally stable in the narrow region 7, but transversal stability is soon lost; in region 8, the alternative prey can invade the limit cycle of the focal prey and the predator, and drives the focal prey extinct (recall that there is no stable interior equilibrium here). Finally, the transcritical bifurcation line between regions 5 and 8 marks where the unstable focus-node, which is present in regions 3-4 and 6-8, leaves the positive orthant and makes the boundary equilibrium $(\bar{n}_1, 0, \bar{p})$ (which has lost stability through the supercritical Hopf bifurcation) also transversally unstable. The supercritical Hopf bifurcation of the boundary equilibrium $(\bar{n}_1, 0, \bar{p})$ exists independently of the transversal stability of this equilibrium, so that the bifurcation line continues also below point A. In region 5, orbits on the boundary $n_2 = 0$ go to a transversally unstable limit cycle, whereas in region 1, to a transversally unstable equilibrium. Starting with all three species present, the focal species goes extinct both in region 1 and region 5, and the only attractor is the boundary equilibrium $(0, \bar{n}_2, \bar{p})$.

The selection gradient of c_1 is negative at the stable boundary equilibrium $(\bar{n}_1, 0, \bar{p})$ in region 6 and on the stable limit cycle in region 7. As c_1 evolves downwards, this first stabilizes the boundary equilibrium (if starting from region 7), and then sufficiently decreases the equilibrium

density of the predator for the alternative prey to invade the system (when crossing from region 6 to region 3). Once the alternative prey is present (in region 3), evolutionary suicide occurs through the subcritical Hopf bifurcation as described above.

Notice that in regions 6 and 7, the focal prey and the predator behave as in the "specialist predator" model of Matsuda and Abrams (1994), where evolutionary suicide is not possible. However, the focal prey evolves such that the system becomes vulnerable to the invasion of the alternative prey, and once the alternative prey invades, the focal prey evolves to its extinction. This may be seen as an indirect way of evolutionary suicide; the evolution of the focal prey first facilitates the invasion of a new species (the alternative prey), whereby evolutionary suicide becomes possible. In this example, the evolution of the focal prey cannot drive the alternative prey extinct.

6 Discussion

Evolutionary suicide, a species evolving to its own extinction, is perhaps the most counterintuitive result of evolution by natural selection (Gyllenberg 2008). From a mathematical point of view, a slowly evolving trait may be seen as a slowly changing bifurcation parameter of the population dynamics of the evolving species. Evolutionary suicide can happen when the evolving trait crosses a point of a catastrophic bifurcation, where a population dynamic attractor disappears without leaving a new attractor in its neighbourhood (Gyllenberg and Parvinen 2001; Gyllenberg et al. 2002; Parvinen and Dieckmann 2013).

Note that crossing a catastrophic bifurcation point is generically a necessary but not a sufficient condition for evolutionary suicide, because the system may settle at another interior attractor such that no species is lost (see Dercole et al. 2002 for an example). In the model we consider in the present paper, however, the interior attractor is unique and crossing a catastrophic bifurcation does result in extinction. We note further that evolution may lead to very low equilibrium population densities (Matsuda and Abrams 1994b) or to a limit cycle along which population density becomes repeatedly very low (e.g. in the Rosenzweig-MacArthur predator-prey model with slow predator and fast prey dynamics, Rinaldi and Muratori 1992; Dercole et al. 2010) also without encountering a catastrophic bifurcation. Since there is a positive attractor, these examples are not considered to be evolutionary suicide, but may in reality lead to extinction e.g. due to demographic stochasticity. Without a catastrophic bifurcation, however, evolution of a single species generically cannot lead to zero population density of the same species (Gyllenberg and Parvinen 2001; a mathematically non-generic but biologically relevant exception is analysed by Boots and Sasaki 2003 and Boldin and Kisdi 2016).

Webb (2003) has catalogued bifurcations that potentially lead to evolutionary suicide. Since the path the evolving trait(s) generate in the parameter space of population dynamics is unlikely to go through bifurcations of codimension 2 and higher, the bifurcations where evolutionary suicide can generically happen are the codimension-1 catastrophic bifurcations. In 2-dimensional systems, these are the saddle-node bifurcation, the subcritical Hopf bifurcation, the fold bifurcation of limit cycles, and global bifurcations involving homoclinic or heteroclinic structures;

the subcritical Neimark-Sacker bifurcation of limit cycles should be added for systems in three dimensions and higher.

Matsuda and Abrams (1994) demonstrated evolutionary suicide in a one-dimensional model of a prey that evolves its foraging effort under threat from a "generalist" predator that has constant population density. With single-species population dynamics, the only possible route to evolutionary suicide is through a fold bifurcation of equilibria (the one-dimensional analogue of the saddle-node bifurcation), and this is what Matsuda and Abrams (1994) found. In the present paper, we have relaxed the assumption of constant predator density by adding an alternative prey, which can maintain the predator also if the focal prey does not, and by varying the strength of coupling between the predator's dynamics and the focal prey through the parameter α_1, the factor at which the predator converts the focal prey consumed into predator offspring ($\alpha_1 = 0$ means that the predator's dynamics are decoupled from the focal prey as assumed by Matsuda and Abrams 1994). In this augmented model, we have found evolutionary suicide through saddle-node bifurcations, subcritical Hopf bifurcations (Figures 3 and 4), and fold bifurcations of limit cycles (Figure 2; a possibility Webb (2003) did not have an example for). In our first example in section 5, evolutionary suicide is not robust when the predator's dynamics are not independent of the focal prey, because the focal prey is not viable unless α_1 is small (Figure 3). In our second example, however, evolutionary suicide through a saddle-node bifurcation occurs in a wide interval of α_1, and in another wide interval of α_1 we obtain evolutionary suicide via a subcritical Hopf bifurcation (Figure 4).

Further, our second example illustrates an indirect way of evolution leading to extinction. Evolutionary suicide is not possible without the alternative prey, so that if the 2-species system of the focal prey and the predator excludes the alternative prey, as it happens in regions 6 and 7 in Figure 4b, then the system appears to be safe. The evolution of the focal prey however makes the community vulnerable to the invasion of the alternative prey (by crossing to region 3 in Figure 4), whereupon the focal prey continues to evolve to its own extinction (crossing to region 4). A similar, but not identical, indirect evolutionary suicide occurs in the intraguild predation model of Hin and de Roos (*in prep*), who found that the evolution of the predator opens the community to the invasion of a new species that drives the predator instantly (i.e., without further trait evolution) extinct. The different routes to evolutionary suicide that open up in our 3-species model and especially the scenario of indirect evolutionary suicide highlight the importance of studying evolution in a community context.

In our model, only the special case of $\alpha_1 = 0$ is analytically tractable. $\alpha_1 = 0$ means that the predator does not benefit from capturing the focal prey, which is biologically unrealistic. Still, the analysis of this special case is useful, because, by continuity, the results hold also for small positive values of α_1. We used a construction method similar to critical function analysis (de Mazancourt and Dieckmann 2004; Bowers et al. 2005; Geritz et al. 2007; Kisdi 2006, 2015; Kisdi et al. 2013) to obtain birth rate functions such that evolutionary suicide through a saddle-node bifurcation occurs at a prescribed foraging effort c_1^* of the focal prey. Note that the function $c_1 \mapsto B(c_1)$ used for linking foraging effort to offspring production is the least justified element of the model. Our analysis in section 3 shows that a wide class of two-parameter functions can lead to evolutionary suicide. In section 4, we extended the method of section 3 to construct an example for evolutionary suicide through a fold bifurcation of limit cycles. It

follows from the autonomous dynamics of the predator and the alternative prey (which obey the Rosenzweig-MacArthur predator-prey model when $\alpha_1 = 0$) that other catastrophic bifurcations are not possible in this limiting case of the model.

For $\alpha_1 = 0$, we have also shown that evolutionary suicide can happen only when c_1 evolves downwards, not when it evolves towards higher values (with arbitrary birth rate functions, see section 3). This is so despite that the saddle-node bifurcation can open in either direction, and also the selection gradient can point in either direction (see Figure 1 for examples); but left-opening bifurcations cannot combine with positive selection gradients. Also in our numerical analysis of the full model (section 5), we find evolutionary suicide with decreasing c_1. The title of Matsuda and Abrams (1994) equates a low foraging effort c_1 with the prey being timid. We could thus conclude that evolving timidity may lead to suicide whereas evolving braveness will not. Note, however, that the trait c_1 is expressed by the prey independently of whether or not the predator presents an acute danger to the individual prey. This is different from timidity as a reaction to an encounter with the predator, for example hiding when the predator is sighted or smelled. Prey reacting to the actual presence of the predator implies a different functional response (Geritz and Gyllenberg 2012), and the evolution of timidity in the sense of hiding quicker and for longer times upon sensing a predator is different from the evolution of foraging effort we considered (Geritz and Gyllenberg 2014).

We assume that the two prey species interact only via the shared predator (apparent competition, Holt 1977). Adding resource competition or interference competition between the two prey species would make the population dynamics even more complicated, with heteroclinic bifurcations and chaos (see Bazykin 1998; Groll et al. 2017). Global bifurcations like a heteroclinic bifurcation are also possible routes to evolutionary suicide (Webb 2003), but in our model these are not found. Further, in our model the Holling type II functional response is the only source of an Allee effect in the dynamics of the prey. In reality, however, the prey may suffer an Allee effect also due to other reasons, such as due to low mating success at low population densities. In this case, heteroclinic bifurcations occur (Van Voorn et al. 2007) and also a specialist predator can drive the prey to evolutionary suicide (Berec et al. 2018).

Throughout this paper, we assumed that only the focal prey evolves, whereas the parameters pertaining to the alternative prey and the predator are fixed. This assumption ensures that we have "clean" examples of evolutionary suicide, scenarios where extinction is a direct consequence of the evolution of the focal species. Our bifurcation plots in Figures 3 and 4, however, give some insight also into the consequences of predator evolution. Given that the predator spends a fraction q of its time hunting for the focal prey, it will be under selection to utilize this prey as best as possible, and hence (everything else being the same) the conversion factor α_1 will increase. As a consequence, the predator can overhunt and drive the alternative prey extinct, but, somewhat paradoxically, the focal prey can also go extinct. The alternative prey is excluded when, by increasing α_1, the system crosses from region 3 to region 6 in Figure 4. This is an example for apparent competition between the two prey species resulting in competitive exclusion via a transcritical bifurcation; as α_1 increases, the focal prey maintains higher predator density, and therefore the alternative prey gradually declines to extinction (Holt 1977). The same scenario can not lead to the exclusion of the focal prey, because with n_1 going to zero, the dynamics in equations (4) become independent of α_1. Increasing α_1 may, however, result in a catastrophic

bifurcation, whereupon the focal prey crashes to extinction. This happens in Figure 3 when increasing α_1 takes the system from region 2 to 1 (saddle-node bifurcation), from region 3 to 4 (subcritical Hopf bifurcation) or from region 5 to 4 (fold of limit cycles). The example in Figure 4 shows a further twist; starting from region 3 at high values of c_1, increasing α_1 first leads to the exclusion of the alternative prey (in regions 6 and 7), but as the predator further increases α_1, the alternative prey can invade again and drive the focal prey extinct (when crossing to region 8). In both examples, as the focal prey becomes a valuable resource for the predator, the predator drives it extinct!

An essential assumption of our model is that the predator hunts for only one prey species at a time. Biologically, this may be the case if the two prey species live in separate habitats, or if they are active (and therefore accessible) during different times. Our model applies also if individual predators have non-heritable fixed preferences such that a fraction q of the predator population hunts for the focal prey and the remaining fraction for the alternative prey. In Appendix C, we investigate a model where all three species are present simultaneously in one well-mixed system, and the predators catch any prey without preference. We show that this alternative model, or its extension to an arbitrary number of prey species, has no saddle-node bifurcation and hence cannot underpin the model of Matsuda and Abrams (1994). To explain this result heuristically, note that at least one prey species must be common for the predator to be at a sufficiently high density to drive the evolution of its prey. In a well-mixed system, a common prey species saturates the predator and thereby lifts the predator-induced Allee effect, described in the Introduction, in the dynamics of the less common prey. Without an Allee effect, the system does not have a saddle-node bifurcation. In our main model in equations (4), the alternative prey can maintain a high predator density and yet the predator, when it hunts for the focal prey, is not saturated with the alternative prey.

We obtain evolutionary suicide assuming that the predator is constrained to spend a fraction q of its time hunting for only the focal prey, even if consumption of the focal prey contributes little or none to the predator's fitness (α_1 is small or zero). If q were free to evolve, the predator would spend its time hunting for the more profitable prey (see Krivan and Eisner 2006 for a version of this model where the predator can quickly adapt its foraging behaviour). However, there may be reasons why the predator does not evolve q to zero even if α_1 is small. If the alternative prey is not active during part of the day or its habitat is inaccessible (e.g. too dark or too cold/hot) in fraction q of the predator's active time, then even a small positive α_1 makes it worthwhile for the predator to exploit the focal prey during the time otherwise lost. If it is easier to catch the focal prey than the more profitable alternative prey, then individuals in weaker condition may prefer the focal prey even if α_1 is small. Hunting for the focal prey gives no advantage when $\alpha_1 = 0$; hence to underpin the evolutionary suicide model of Matsuda and Abrams (1994) with our model, it is important whether evolutionary suicide is robust with respect to increasing α_1 from zero to positive (as it is in Figure 4) or not (Figure 3).

A further possibility to maintain $q > 0$ as a worthwhile strategy for the predator is if the focal prey shares its habitat with a third species of prey. With only this one shared habitat, the dynamics are like in our alternative model in Appendix C, where a saddle-node bifurcation is not possible. Adding the second habitat and its alternative prey, however, changes this situation (see the equations of this three-prey model at the end of Appendix C). Continuity with our

model in (4) ensures that evolutionary suicide is now possible at least if the third prey is not too abundant and/or profitable (i.e., if $\alpha_3 n_3$ is sufficiently small). The presence of the third prey makes it worthwhile for the predator to spend any time not suitable for hunting in the second habitat with hunting in the first habitat, even if $\alpha_1 = 0$. Incidental predation on the focal prey may then drive the focal prey to evolutionary suicide.

The three-prey model suggests that extending our model to include more species would not be a futile exercise in complexity. Our main finding is that embedding the classic model of Matsuda and Abrams (1994) into our slightly larger community yields not only much richer dynamics but also new routes to evolutionary suicide, including the indirect route where the invasion of a new species is the key to evolutionary suicide. Studying evolution in a community context is an important challenge for future research (Walsh 2013; terHorst et al. 2018).

Acknowledgements

We thank Francesca Scarabel for help with MatCont, and anonymous reviewers for their comments on an earlier version of this paper. C.V. was partially supported by a Leverhulme Trust studentship as part of the Centre for Applied Biological Modelling at the University of Sheffield. E.K. was funded by the Academy of Finland through the Centre of Excellence in Analysis and Dynamics.

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Appendix A

In this appendix, we investigate the interior equilibria $(\bar{n}_1, \bar{n}_2, \bar{p})$ of the population dynamics in equations (4). Using the notation

$$G_1 = B(c_1) - d_1, \quad G_2 = \frac{\rho_2 c_2}{1 + \rho_2 b_2 c_2} - d_2$$

for abbreviating the density-independent part of the *per capita* growth rates as well as

$$\phi_1 = \frac{q c_1 \beta_1 \bar{n}_1}{1 + c_1 \beta_1 h_1 \bar{n}_1}, \quad \phi_2 = \frac{(1-q) c_2 \beta_2 \bar{n}_2}{1 + c_2 \beta_2 h_2 \bar{n}_2} \tag{A.1}$$

for the Holling II factors, the equilibrium equations (assuming nonzero population densities $\bar{n}_1$, $\bar{n}_2$, $\bar{p}$) are

$$G_1 - \delta_1 \bar{n}_1 = \phi_1 \bar{p}/\bar{n}_1 \tag{A.2a}$$
$$G_2 - \delta_2 \bar{n}_2 = \phi_2 \bar{p}/\bar{n}_2 \tag{A.2b}$$
$$\alpha_1 \phi_1 + \alpha_2 \phi_2 = \mu \tag{A.2c}$$

First, we express $\bar{n}_1$ and $\bar{n}_2$ from (A.1),

$$\bar{n}_1 = \frac{\phi_1}{c_1 \beta_1 (q - h_1 \phi_1)}, \quad \bar{n}_2 = \frac{\phi_2}{c_2 \beta_2 (1 - q - h_2 \phi_2)} \tag{A.3}$$

Next, we eliminate $\bar{p}$ by dividing (A.2a) with (A.2b), and substitute $\bar{n}_1$ and $\bar{n}_2$ with the above expressions to rewrite the equation in terms of ϕ_1 and ϕ_2,

$$\frac{G_1 - \delta_1 \frac{\phi_1}{c_1 \beta_1 (q - h_1 \phi_1)}}{G_2 - \delta_2 \frac{\phi_2}{c_2 \beta_2 (1 - q - h_2 \phi_2)}} = \frac{c_1 \beta_1 (q - h_1 \phi_1)}{c_2 \beta_2 (1 - q - h_2 \phi_2)}$$

which is rearranged into

$$[G_1 c_1 \beta_1 (q - h_1 \phi_1) - \delta_1 \phi_1] c_2^2 \beta_2^2 (1 - q - h_2 \phi_2)^2 = [G_2 c_2 \beta_2 (1 - q - h_2 \phi_2) - \delta_2 \phi_2] c_1^2 \beta_1^2 (q - h_1 \phi_1)^2 \tag{A.4}$$

Substituting $\phi_2 = (\mu - \alpha_1 \phi_1)/\alpha_2$ from (A.2c), we obtain a cubic polynomial for ϕ_1. Each root of this cubic equation yields a root of the equilibrium equations (A.2) with ϕ_2 from (A.2c), $\bar{n}_1$ and $\bar{n}_2$ from (A.3), and $\bar{p}$ from (A.2a). Figure 29 of Vitale (2016) shows an example where all three roots are real and positive, i.e., biologically admissible equilibria of the model.

If $\alpha_1 = 0$, then $\phi_2 = \mu/\alpha_2$ is a constant independent of ϕ_1. Therefore (A.4) is only quadratic in ϕ_1, yielding at most two interior equilibria.

Appendix B

In section 3, we show that evolutionary suicide occurs at a given trait value c_1^* if the birth rate function at this point has a specific value $B(c_1^*)$ given by (8) and a slope $B'(c_1^*)$ in the interval given by inequalities (9) and (10). The figure below shows the width of this interval,

$$\sqrt{q\bar{p}\frac{\delta_1}{(c_1^*)^2 h_1}} - \frac{1}{\beta_1}\frac{\delta_1}{(c_1^*)^2 h_1}$$

Evolutionary suicide is impossible if this width is negative (grey area). The width depends on the chosen value of c_1^* (panel (a)). It is easy to see also analytically that the width increases with $q\bar{p}$ (all panels) and with β_1 (panel (b)), and has a maximum as a function of the composite parameter $(c_1^*)^2(h_1/\delta_1)$ (panels (a),(c)).

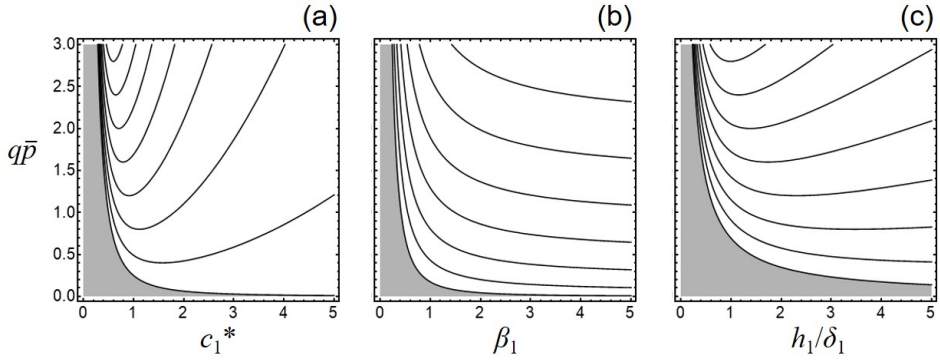

Figure 5: The width of the interval for $B'(c_1^*)$ that results in evolutionary suicide. The contour lines, starting from the bottom line, correspond to values $0, 0.1, 0.2, ..., 0.7$. In the grey area (i.e., below the 0 contour line) condition (7) is violated and hence evolutionary suicide is not possible. Parameter values: (a) $h_1/\delta_1 = 4, \beta_1 = 1$; (b) $h_1/\delta_1 = 4, c_1^* = 1.2$; (c) $\beta_1 = 1, c_1^* = 1.2$.

Appendix C

Here we consider a well-mixed system where all species are present in one habitat and the predator searches for both prey simultaneously all time. This alternative model is given by

$$\frac{dn_1}{dt} = \left[B_1 - d_1 - \delta_1 n_1 - \frac{c_1\beta_1 p}{1 + c_1\beta_1 h_1 n_1 + c_2\beta_2 h_2 n_2}\right] n_1 \quad \text{(C.1a)}$$

$$\frac{dn_2}{dt} = \left[B_2 - d_2 - \delta_2 n_2 - \frac{c_2\beta_2 p}{1 + c_1\beta_1 h_1 n_1 + c_2\beta_2 h_2 n_2}\right] n_2 \quad \text{(C.1b)}$$

$$\frac{dp}{dt} = \left[\frac{\alpha_1 c_1\beta_1 n_1 + \alpha_2 c_2\beta_2 n_2}{1 + c_1\beta_1 h_1 n_1 + c_2\beta_2 h_2 n_2} - \mu\right] p \quad \text{(C.1c)}$$

where B_1 and B_2 abbreviate the birth rates of prey species 1 and 2, respectively. Note that in contrast to equations (4), here both prey densities appear in the denominator of the Holling II

terms. To interpret this difference, recall that 1 over the denominator of the Holling II functional response gives the fraction of predators that are searching (as opposed to handling an already captured prey). In the model of (C.1), handling any of the prey in the well-mixed system removes a predator individual from the searching predators. In our main model (4), in a fraction q of its time a predator can only capture, and therefore handle, prey species 1, whereas in the remaining $1-q$ fraction of time it can handle only species 2. Thus within each time frame, only handling one prey species removes an individual from the searching predators; accordingly, only one prey species appears in the functional response in equations (4).

The model in (C.1) naturally extends to k prey species,

$$\frac{dn_i}{dt} = \left[B_i - d_i - \delta_i n_i - \frac{c_i\beta_i p}{1+\sum_{j=1}^{k} c_j\beta_j h_j n_j}\right] n_i \quad \text{for } i=1,...,k \tag{C.2a}$$

$$\frac{dp}{dt} = \left[\frac{\sum_{i=1}^{k}\alpha_i c_i\beta_i n_i}{1+\sum_{j=1}^{k} c_j\beta_j h_j n_j} - \mu\right] p \tag{C.2b}$$

To find the equilibria, let S denote the number of searching predators,

$$S = \frac{p}{1+\sum_{j=1}^{k} c_j\beta_j h_j n_j} \tag{C.3}$$

At an interior equilibrium of (C.2), we have

$$B_i - d_i - \delta_i n_i - c_i\beta_i S = 0 \quad \text{for } i=1,...,k \tag{C.4a}$$

$$\frac{1}{\mu}\sum_{i=1}^{k}\alpha_i c_i\beta_i n_i = 1 + \sum_{j=1}^{k} c_j\beta_j h_j n_j \tag{C.4b}$$

This is a linear system for the unknowns $n_1,...,n_k,S$, and hence generically has only one solution for the equilibrium. The equilibrium value of p follows directly from (C.3). Since the population dynamics in (C.1) has a unique interior equilibrium, it cannot undergo a saddle-node bifurcation for any change of the traits such as the foraging effort c_1 and the birth rate B_1.

The four-species model

$$\frac{dn_1}{dt} = \left[B_1 - d_1 - \delta_1 n_1 - \frac{qc_1\beta_1 p}{1+c_1\beta_1 h_1 n_1 + c_3\beta_3 h_3 n_3}\right] n_1 \tag{C.5a}$$

$$\frac{dn_2}{dt} = \left[B_2 - d_2 - \delta_2 n_2 - \frac{(1-q)c_2\beta_2 p}{1+c_2\beta_2 h_2 n_2}\right] n_2 \tag{C.5b}$$

$$\frac{dn_3}{dt} = \left[B_3 - d_3 - \delta_3 n_3 - \frac{qc_3\beta_3 p}{1+c_1\beta_1 h_1 n_1 + c_3\beta_3 h_3 n_3}\right] n_3 \tag{C.5c}$$

$$\frac{dp}{dt} = \left[q\frac{\alpha_1 c_1\beta_1 n_1 + \alpha_3 c_3\beta_3 n_3}{1+c_1\beta_1 h_1 n_1 + c_3\beta_3 h_3 n_3} + (1-q)\frac{\alpha_2 c_2\beta_2 n_2}{1+c_2\beta_2 h_2 n_2} - \mu\right] p \tag{C.5d}$$

unites the features of our main model in equation (4) and the alternative model in (C.1); here the focal prey 1 and prey 3 live in the first habitat where the predator spends a fraction q of its time, and the alternative prey 2 lives in the second habitat where the predator spends the remaining fraction $(1-q)$ of its time. This model is briefly mentioned in the Discussion.