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Commentary

Building a synthetic basis for kin selection and evolutionary game theory using population genetics

Jeremy Van Cleve

Department of Biology, University of Kentucky, Lexington, KY 40506, USA

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1. Introduction

Following the work of Hamilton on kin selection (Hamilton, 1964) and Maynard Smith and Price on game theory in evolutionary biology (Maynard Smith and Price, 1973), mathematical analyses of the evolution of social behavior exploded in number. Interest in the topic bubbled over into popular articles and books (e.g., Wilson, 1975; Dawkins, 1976) that celebrated the ability of these new models to provide evolutionary explanations for the patterns of cooperation and conflict observed in humans, animals, and even single-celled organisms. At the time, the enthusiasm for the results of these models quickly outpaced the formal connections between kin selection and evolutionary game theory approaches on the one hand and population genetics models on the other. Population genetic models seek to capture the full genotype frequency dynamics of a population using explicit assumptions about the mode of reproduction (e.g., random mating among diploids), selection (e.g., differential viability or fertility), mutation, and recombination (if multiple loci are involved). In contrast, kin selection and evolutionary game theory models sidestep these biological details and thus do not capture the full dynamics; instead, they claim that the phenotypes or trait values that optimize individual fitness are the same phenotypes that are stably maintained by selection in the long run. Individual fitness is represented with a "fitness function" $w_i(z_i, z_j)$ that measures the expected number of surviving offspring of a single focal individual i (over a single generation) as a function of a heritable trait value of the focal individual z_i (e.g., amount of investment in cooperative hunting) and the trait values of other individuals in the social group (average value z_j). Moreover, kin selection introduced "inclusive fitness" as an optimized fitness function that captures the effect of population structure on selection due to individuals living in social or family groups through a single "relatedness coefficient" r (Hamilton, 1964); this coefficient is also an essential component of the well-known "Hamilton's rule" for the evolution of altruism (Hamilton, 1964; Lehmann and Rousset, 2014).

It was initially an open question whether there were circumstances under which one could justify the fitness optimization or inclusive fitness approaches using population genetics methods. Moreover, early work by P.A.P. Moran proved that fitness optimization could fail if traits are determined by more than one locus with recombination between them (Moran, 1964). Classic population genetic models that justified kin selection and evolutionary game theory approaches under specific assumptions began to appear in the late 1970s in *Theoretical Population Biology* (*TPB*) and other journals. These results established that specific mappings between genotype and phenotype are required for kin selection and game theoretic analyses to yield the same predictions as population genetics models. In *TPB* for example, Cavalli-Sforza and Feldman (1978) showed that kin selection, via a calculation of inclusive fitness, predicts invasion of mutant alleles for a one-locus diploid population when fitness is additive, and Eshel (1982) showed that the game theoretic concept of "evolutionarily stable strategies" (ESSs; Maynard Smith and Price, 1973; Maynard Smith, 1974) is sustainable in the sense that ESSs are locally stable in one-locus diploid populations when the ESS is accessible by the genotype–phenotype map.

However, these early population genetic analyses separately addressed the role of fitness optimization (via the ESS concept) and genetic relatedness (via inclusive fitness). Thus, they did not provide a single unified population genetic approach that justified

E-mail address: jvancleve@uky.edu.

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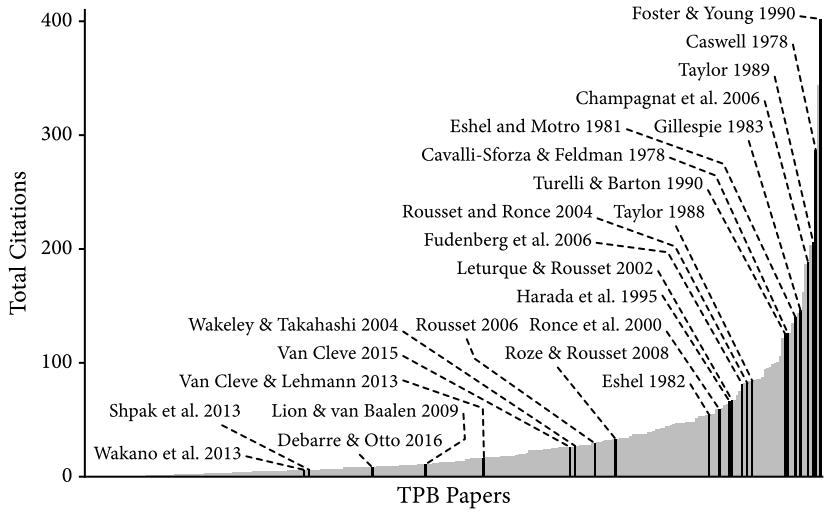

Fig. 1. Total citations in the Web of Science database (https://www.webofknowledge.com/) for *TPB* publications cited in this manuscript (labeled black bars). Other *TPB* papers mentioning social evolution, kin selection, or evolutionary game theory (unlabeled gray bars) were retrieved using the following "Advanced Search" query: `SO = Theoretical Population Biology AND TS = ((evol* NEAR/0 game*) OR (game NEAR/0 theor*) OR cooperat* OR altruis* OR (social* (evol* OR behav*)) OR ''kin selection'' OR ''group selection'' OR ''inclusive fitness'' OR (evol* NEAR/1 stab*) OR (adaptive NEAR/0 dynamic*) OR ''Hamilton's rule'')`. The search was performed in February 2020 and resulted in 287 articles.

the simultaneous use of ESS and inclusive fitness concepts in studying the evolution of social traits. A unified approach did emerge by the early 2000s (first fully elucidated by François Rousset; Rousset and Billiard, 2000; Rousset, 2004), and it leveraged several important insights and conceptual advances published in *TPB*. These concepts include weak selection induced quasi-equilibria (Turelli and Barton, 1990; Rousset, 2006), selection in class structured populations (Taylor, 1988; Rousset and Ronce, 2004), weak mutation induced long-term evolution and stochastic stability (Foster and Young, 1990; Van Cleve, 2015), and convergence stability (Eshel and Motro, 1981; Taylor, 1989). I call this unified population genetic approach the "quasi-equilibrium long-term evolution" (QELTE) approach due to its reliance on these concepts. The *TPB* papers that present the building blocks of the QELTE approach are among some of the most highly cited *TPB* papers that deal with social evolution (see Fig. 1). In what follows, I briefly outline the QELTE approach to analyzing the evolutionary equilibria of a model of a single social trait in a subdivided population and then describe the important concepts used in this approach in more detail through the *TPB* papers that helped to introduce them.

2. A (very) brief outline of the QELTE approach

This outline follows prior work by Rousset and others (Rousset and Billiard, 2000; Rousset, 2004; Wakano et al., 2013; Lehmann and Rousset, 2014; Van Cleve, 2015). Suppose that a mutant allele at a single haploid locus arises in a population of total size N with some spatial structure or subdivision where individuals are more likely to mate and interact with other individuals in their own patch or group than they are with individuals in different patches. Patches are the same size, individuals disperse or migrate between patches at equal rates, and individuals belong to the same demographic or reproductive class. Assume that a wild-type or resident allele produces a phenotype with trait value z and a mutant allele produces a phenotype with value $z+\delta$. Then, the phenotype of any individual j is $z_j = z + \delta p_j$ where p_j is the frequency of the mutant allele in individual j. The first major assumption in the QELTE approach is weak selection: $|\delta|$ is assumed to be small (sometimes called "δ-weak" selection; Wild and Traulsen, 2007), which means that the effect of the mutant allele on individual fitness is small. Mathematically, this means that the expected fitness of focal individual i with the mutant allele in population with the resident allele z will be

$$\begin{aligned} w_i(z_1,\ldots,z_N) &= 1+\delta\frac{\mathrm{d}w_i}{\mathrm{d}\delta}+O\left(\delta^2\right) \\ &= 1+\delta\left(\frac{\partial w_i}{\partial z_i}p_i+\sum_{j\neq i}^{N}\frac{\partial w_i}{\partial z_j}p_j\right)+O\left(\delta^2\right) \end{aligned} \tag{1}$$

where the derivatives are evaluated at $\delta = 0$. Given the mutant allele frequency distribution in the population (i.e., $p_1,\ldots,p_N$), the right-hand side in Eq. (1) for w_i is a function of only the mutant trait deviation δ and the resident trait value z; thus, the expected fitness of the mutant individual is often written for simplicity as a function of only the mutant and average resident trait values, $w_i(z+\delta, z)$. Using the Price equation (Price, 1970) for expected gene frequency change over a single generation and Eq. (1), one can show that the fixation probability $\pi(z+\delta, z)$ of a single mutant allele with trait value $z+\delta$ in a population with the resident trait value z is

$$\pi(z+\delta,z) = \frac{1}{N}+\delta\, S(z)+O\left(\delta^2\right) \tag{2}$$

where $1/N$ is the fixation probability of the mutant allele under neutrality (which is simply the initial frequency of the mutant allele) and the "selection gradient" $S(z)$ is given by

$$S(z) \propto \frac{\partial w_i}{\partial z_i}+\sum_{j\neq i}^{N}\frac{\partial w_i}{\partial z_j}Q_{ij} \tag{3}$$

and is evaluated at the resident trait value z with derivatives in the expression evaluated at $\delta = 0$. The first term in the selection gradient is the "direct effect" of the expression of the trait on the focal individual's fitness. The second term in (3) is the sum of the "indirect effects", which are the effects of trait expression in other individuals on the fitness of the focal individual i multiplied by the probability (under selective neutrality) that individual i and j are identical by descent (IBD) at the trait locus, Q_{ij}. The IBD probabilities Q_{ij} are examples of "quasi-equilibrium" values, which are the values of genetic associations under weak selection (see Section 3). In effect, the selection

gradient can be interpreted as the derivative (with respect to δ) of a certain fitness function (e.g., "lineage fitness"; Akçay and Van Cleve, 2016; Lehmann et al., 2016); thus, traits with value z^* where $S(z^*) = 0$ are candidates for an ESS (Ronce et al., 2000; Rousset and Billiard, 2000) since one condition for an ESS is that it has higher fitness against itself than any possible mutant. The selection gradient in Eq. (3) is also a measure of the effect of the trait z on inclusive fitness (Rousset and Billiard, 2000). When the population structure is given by an island-model (Wright, 1931), the selection gradient simplifies to the classic Hamilton's rule, $S(z) \propto -c(z) + rb(z)$, where the cost (direct effect) and benefit (indirect effect) are functions of the resident trait value z and relatedness r is equal to F_{ST} (Wright, 1951; Hamilton, 1964). Finally, the derivation of the selection gradient can be generalized to populations with class structure (Taylor, 1988, 1990; Leturque and Rousset, 2002; Rousset and Ronce, 2004) using the concept of reproductive value (Fisher, 1930) where individuals of the same genotype are in different classes if they systematically exhibit different rates of survival or reproduction.

The second major assumption in the QELTE approach is that the mutation rate is weak relative to total population size N, which means that there will be at most one mutation segregating in the population at a time (Champagnat, 2006). The evolutionary process then consists of single mutant genotypes either invading the population and going to fixation or not invading and going to extinction. This is sometimes called a "long-term" evolutionary process (Eshel, 1996; Hammerstein, 1996; Weissing, 1996). Long-term evolution through a succession of mutant invasions and fixations will approach the ESS phenotype z^* if additionally $S'(z^*) < 0$. This latter condition is called "convergence" stability (Eshel, 1983; Christiansen, 1991) and was developed in an *TPB* paper by Eshel and Motro (1981) and connected to inclusive fitness theory in *TPB* by Taylor (1989). The upshot is that the QELTE approach shows how measures of evolutionary stability from evolutionary game theory and inclusive fitness from kin selection both arise from a single population genetic analysis.

3. Weak selection

One problem for analyzing the evolution of social behavior among kin using explicit population genetic methods is that the population must be structured or compartmentalized and this entails tracking the frequency of each genotype in each compartment, which leads to unwieldy analyses when the number of compartments is larger than a handful. This issue is very similar to the issue that arise in the analysis of models of multiple loci under selection where the number of genotypes to track rapidly becomes very large as the number of loci increases. One way to avoid this complexity is to assume that selection is weak relative to non-selective forces that change genotype frequencies like recombination and migration (so-called "ω-weak" selection, which is less restrictive than δ-weak selection; Wild and Traulsen, 2007). For multiple loci, recombination causes genetic associations like linkage disequilibrium (LD) to quickly reach so-called "quasi-equilibrium" values (Kimura, 1965; Nagylaki, 1976) while selection causes allele frequencies at individual loci to change very slowly in comparison; once at quasi-equilibrium, selection changes LD and other quasi-equilibrium values very slowly compared to the rate it changes allele frequencies. Turelli and Barton (1990) in *TPB* did early work on the quasi-equilibrium approach for multiple loci and later reformulated their analysis in a framework (Barton and Turelli, 1991) that has been used in social evolution models with multiple traits (e.g., Roze and Rousset, 2005; Gardner et al., 2007; Roze and Rousset, 2008).

Rousset noted in *TPB* (2006) how weak selection and the quasi-equilibrium concept also apply to subdivided populations when selection is weak relative to migration and local genetic drift within demes. Given these assumptions, genetic associations within and between demes (e.g., F_{ST}) reach quasi-equilibrium values quickly compared to the slow change of average allele frequency across the whole population, which can change due to weak selection for or against an allele increasing investment in some social behavior. This difference in rates is sometimes called a "separation of timescales" in subdivided populations (Wakeley, 2003; Roze and Rousset, 2003; Rousset, 2006). Rousset (2006) described how this separation of timescales works in a lattice model of population structure where there is genetic isolation by distance, and Wakeley and Takahashi (2004) in *TPB* demonstrated it for an island model with overlapping generations via a Moran model (Moran, 1958) of reproduction. Roze and Rousset showed in *TPB* (2008) how to use the quasi-equilibrium approach for both multiple loci and multiple demes when the population structure is given by an infinite island model. This involved extending the quasi-equilibrium approach to include genetic associations that are nonzero under selective neutrality: equilibrium LD is zero under neutrality when recombination is nonzero but F_{ST} is nonzero even under neutrality and even for strong migration rates. Detailing how exactly the combined multi-locus multi-deme quasi-equilibrium approach connects to ESS concepts remains an open problem.

4. Class structured models

Another difficulty for measuring inclusive fitness using population genetics is that kin selection is often studied in populations with different demographic classes, such as workers and queens in a social insect colony. As is the case for population structure, class structure increases the dimensionality of the population genetic model and weak selection can simplify the analysis. What emerges from a δ-weak selection approximation is an expression for the selection gradient $S(z)$ that is a weighted sum of the effects of the mutant allele on w_{ij}, which is the number of class i offspring produced by an individual in class j. The weights are the product of the reproductive value of class i and the proportion of individuals in class j obtained from the left and right eigenvectors, respectively, of the matrix $W = (w_{ij})$ (Taylor, 1988, 1990; Rousset and Ronce, 2004). These weights are also proportional to the sensitivity (derivative) of the growth rate (eigenvalue) of a population with projection matrix W; Caswell helped introduce this way of calculating growth rate sensitivities to population biology in *TPB* in 1978. The effect of the mutant allele on w_{ij} can further be expressed in terms of inclusive fitness with direct and indirect effects where the relatedness terms in the indirect effects now measure genetic identity between individuals in different classes. In essence then, a δ-weak selection analysis of the population genetics model yields a measure of inclusive fitness appropriate for class-structured populations. Special cases of this type of result had been derived in models of eusocial insects and other species, but Taylor in *TPB* (1988) was the first to sketch the generality of the matrix population model approach using two sexes as example classes. Taylor later generalized the approach for arbitrary classes (Taylor, 1990) and wrote a very influential paper (Taylor and Frank, 1996) describing "How to Make a Kin Selection Model" using this approach.

Most applications of the class-structured approach assume (at least implicitly) that the demography of the population is constant or that it is unaffected by expression of the mutant alleles. For example, this assumption holds if one requires that group or deme size is constant or variations in group size are independent of mutant allele frequency. However, if expression of mutant alleles affects group productivity or carrying capacity, then selection will change the frequency of the mutant allele in

part because of this effect. Rousset and Ronce described this effect of selection on demography in *TPB* (2004) and showed how the total effect of selection under δ-weak selection as measured by the selection gradient $S(z)$ is the sum of two effects: the first is the normal inclusive fitness effect and the second is the mutant's effect on fitness through its effect on demography. For example, when dispersal rates evolve and local deme sizes are allowed to vary, the second effect measures how mutant dispersal rates affect fitness through their effects on local deme size. Rousset and Ronce (2004) showed the importance of both effects through an example of a metapopulation experiencing periodic local extinction where dispersal is costly and juvenile survival is positive frequency dependent (i.e., the Allee effect). The cost of dispersal causes selection for lower dispersal values and can result in "evolutionary suicide" (for further discussion of this topic, see Kisdi (2020)) where dispersal evolves below a value necessary for metapopulation persistence (Gyllenberg et al., 2002). However, local competition with genetic kin, which is captured with the inclusive fitness effect, selects for increased dispersal. Thus, these two forces can balance one another, and this balance would be missed without including either the explicit effect of genes on demography or the effect of interactions with genetic kin.

5. Weak mutation and stochastic stability

A general complexity of population genetic models is the possibility of polymorphisms generated by balancing or negative frequency dependent selection or by strong mutation rates. These forces are very important at some loci and in some organisms: for example, balancing selection in primates due to pathogen evolution has been implicated in polymorphisms at immune system genes like major histocompatibility complex (Leffler et al., 2013), and strong mutation rates in microbial pathogens and viruses generate competition between multiple beneficial types (i.e., "clonal interference"; Lang et al., 2013). However, tracking the fate of more than two alleles at a locus (i.e., the mutant and resident types) and looking for polymorphisms maintained by selection introduces substantial complexity into population genetics analyses. This complexity often makes the analysis of models with population structure or social behavior difficult. Two assumptions can help eliminate this complexity and make population genetic analyses of kin selection and evolutionary game theory more tractable. First, assuming δ-weak selection can eliminate many forms of frequency-dependent selection since such frequency dependence typically occurs at second or higher order in δ. Second, if mutation is weak enough relative to population size so that mutant alleles are lost to genetic drift or selection long before a new mutation arrives, then at most one mutant allele will be present in the population at a time. In effect, these two assumptions produce an evolutionary Markov chain where single mutations arise and either go extinct or fix and shift the population from one resident allele to another (Van Cleve, 2015). This weak selection and "weaker" mutation regime comes in a variety of guises: it is the "trait substitution sequence" (Dieckmann and Law, 1996; Champagnat et al., 2006) in the adaptive dynamics literature (Metz et al., 1996) and the "sequential fixation" regime in some population genetic models (Desai and Fisher, 2007); it is a type of "strong selection weak mutation" limit introduced by Gillespie in *TPB* (1983); and it is an example of long-term evolution or an evolutionary "streetcar" process (Eshel, 1996; Hammerstein, 1996; Weissing, 1996). Kauffman and Levin (1987) model a similar process for the purpose of studying the rate of adaptation on rugged fitness landscapes. While useful, the long-term evolution perspective is certainly not a full picture of the adaptive process since it a priori excludes scenarios where polymorphisms are stably maintained by frequency dependence or mutation–selection balance.

In evolutionary game theory, the weak mutation limit was first described not as a method for simplifying population genetic analyses but as a way to provide a concept of evolutionary stability more predictive than the ESS. Even without a complex genotype–phenotype map, evolutionary game-theoretic models can produce very rich dynamical behavior since an individual's fitness can be a complex function of the phenotypes of other individuals in the group. For example, these models can have multiple equilibria or ESSs and can produce non-equilibrium behavior such as cycling (Taylor and Jonker, 1978) or even chaos (Schnabl et al., 1991). Foster and Young suggested in *TPB* (1990) an evolutionary stability concept called "stochastic stability" that could account for these scenarios by incorporating stochastic effects, such as mutation and genetic drift, into the dynamics and taking the limit as the strength of the stochastic effects goes to zero. Specifically, Foster and Young argued that the limiting distribution of the (ergodic) stochastic population process is the right measure of persistence in these cases as it doesn't depend on initial condition of the process like a purely deterministic system with multiple ESSs might. Then, as rate of mutation and other stochastic effects goes to zero, there is often one population state remaining with nonzero probability in the limiting distribution and that state is defined as stochastically stable. In the context of the trait substitution sequence described above, the stochastically stable state is the trait value fixed in the population with probability approaching one as the mutation rate approaches zero and the population size approaches infinity. As an example, consider a coordination game where there are two strategies or phenotypes, A and B, and both individuals receive a fitness of 8 when they both use A, 4 when they both use B, and 0 when they use a different strategy than their partner. Both phenotypes A and B are ESSs in this game yet only phenotype A, which generates the largest fitness, is stochastically stable (Foster and Young, 1990).

The notion of stochastic stability kicked off a number of highly-cited papers in evolutionary game theory (Ellison, 1993; Kandori et al., 1993; Young, 1993) that showed how stochastically stable states (in two-player games like those with only a resident and mutant strategy) have an intuitive property in that they are precisely the states in the deterministic dynamics whose basins of attraction are the largest (also known as risk dominance). More recently, the stationary distribution under a low mutation limit has been used to analyze models of cooperation and punishment (though with non-zero stochasticity due to finite population size and genetic drift; Nowak et al., 2004; Fudenberg et al., 2006; Hauert et al., 2007; Rand and Nowak, 2011), and the notion of stochastic stability has been extended to continuous phenotypes (Lehmann, 2012; Van Cleve and Lehmann, 2013).

6. Convergence stability

An ESS z^* can be defined as the phenotype that receives a higher fitness than any alternative phenotype when found in a population composed of itself and the alternative phenotype at sufficiently low frequency (eq. 6.9 in Hofbauer and Sigmund, 1998). This stability concept ensures that z^* cannot be invaded by an alternative phenotype. However, when phenotypes are continuous and all individuals in a population have a trait value close to but not equal to z^*, this does not guarantee that a mutant with a trait value even closer to z^* will invade. Eshel and Motro (1981) in *TPB* were the first to clearly recognize this in their analysis of a model of cooperation among genetically-related individuals. They showed that there are two types of ESSs: ones that are convergence stable (called "continuously stable" in their paper) and ones that are not. For trait values z^* that are convergence stable, Eshel and Motro proved that mutants invading a resident population fixed on a trait value close to

z^* only do so when the mutant trait value is even closer to z^* than is the resident trait value. Building on the work of Eshel and Motro (1981), Taylor published in *TPB* (1989) an influential derivation of the convergence stability condition (termed "m-stability" in the paper). Compared to Eshel and Motro (1981), Taylor's convergence stability condition is simpler since he expresses the mutant trait value as $z + \delta$, which essentially leads to the convergence stability condition $S'(z^*) < 0$ discussed above. Taylor also showed how convergence stability can be applied to a population genetic model using δ-weak selection and the Price equation (Price, 1970) and showed how the population genetic model can generate an inclusive fitness condition. Both Eshel and Motro (1981) and Taylor (1989) argued that convergence stability may be a more important stability condition than evolutionary stability; Taylor specifically noted that convergence stability is easier to check than evolutionary stability (since the former only requires calculating terms of order δ whereas the latter also requires calculating terms of order δ^2). In practice, many models with complex population structure admit practical analytical calculations for convergence stability only. Crucially, neither paper provides an example of a phenotype that is convergence stable but not evolutionarily stable. In fact, Taylor argued that such phenotypes are unlikely to be "biologically plausible", though he noted that they would generate polymorphism in the population. Such phenotypes are now known to be plausible and are called "branching points" in adaptive dynamics (Metz et al., 1996). This topic has a rich history in *TPB* that is reviewed by Kisdi (2020).

7. Conclusion

The QELTE approach to justifying concepts from evolutionary game theory and kin selection using population genetics is mature enough that there are now multiple presentations of this approach (e.g., Rousset, 2004; Wakano et al., 2013; Lehmann and Rousset, 2014; Van Cleve, 2015). As shown above, *TPB* has played a pivotal role in developing the mathematical foundations for the QELTE approach since many of the conceptual advances necessary for this approach were published in the journal. Though it does not have as rich a history in *TPB*, there is a closely related method for deriving ESS and inclusive fitness concepts from population genetic models called the "pair approximation" method (van Baalen and Rand, 1998; Lion and van Baalen, 2008). Pair approximation makes use of weak selection and large population assumptions to derive similar expressions as the QELTE approach, but it does not use the Price equation and instead directly approximates the evolution of spatial correlations in gene frequency. The few examples of this approach in *TPB* include Harada et al. (1995), Lion and van Baalen (2009) and Shpak et al. (2013).

More recently, the QELTE approach has been extended so that evolutionary stability (not just convergence stability) can be assessed in some models with demographic and population structure (Wakano and Lehmann, 2012; Wakano and Iwasa, 2013). Intriguingly, these results show how mutation rate, population size, migration rate, and spatial heterogeneity can affect whether a phenotype z is evolutionarily stable or not (Wakano and Lehmann, 2012; Wakano and Iwasa, 2013; Wakano and Lehmann, 2014; Debarre and Otto, 2016; Parvinen et al., 2018). A rich avenue for future work likely resides in determining the consequences of relaxing some of the core assumptions, such as weak selection, for analyzing models in evolutionary game theory and kin selection. The few attempts at this in a single finite population (Fudenberg et al., 2006) and in an infinite-island of dispersal (Mullon and Lehmann, 2014) suggest that the core assumptions may not be as restrictive as previously thought. Whether authors are developing new methods that relax these core assumptions or building new models that reveal previously unknown complexity, I hope that authors continue to find *TPB* a welcome home for studying social evolution using population genetics, kin selection, and evolutionary game theory.

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