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The maintenance of polymorphism and divergence in sexually selected traits affected by frequency-dependent predation and consistent mating advantages

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Running heads
Left = Ponkshe and Endler
Right = Joint effects of frequency-dependent predation and mate choice

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ABSTRACT

Questions: How does negative frequency-dependent natural selection (NFD) affect the maintenance and divergent fixation of polymorphic traits subject to directional mate preferences? What role does directional natural selection (DNS) on mate preferences play in these conditions when DNS is sex-limited versus when it is not?

Negative frequency-dependent selection: NFD is characterized by a decline in the relative fitness of genotype with an increase in its relative frequency or abundance in a population.

Models: We build sets of population genetic models based on the classical haploid version of the null model of sexual selection and use numerical simulations of deterministic recursions to analyse them.

Conclusions: (1) Environments with NFD weaker than mate preferences of individual female types promote the fixation of sexual traits in opposite directions than environments with NFD stronger than mate preferences. (2) Strong NFD prevents the fixation of sexual traits in opposite directions despite the strong differences in mate preferences of individual female types. (3) The Fisher process interacts with NFD identically whether DNS is sex-limited or not.

Keywords: Fisher process, frequency-dependent selection, mate choice, null model, polymorphic sexual traits, polymorphism maintenance, population divergence.

INTRODUCTION

Sexually selected traits can evolve in an ecological context where mate choice is based on traits that are simultaneously under negative frequency-dependent natural selection (NFD). Colour polymorphisms are ideal systems to look for such interactions (Gray and McKinnon, 2007; Wellenreuther *et al.*, 2014). For example, colour polymorphisms can be under NFD due to frequency-dependent predation (Allen, 1972; Cooper, 1984; Endler, 1988; Bond and Kamil, 1998; Punzalan *et al.*, 2005; Bond, 2007; Ishii and Shimada, 2010). Furthermore, colour polymorphic species can show variation in mate preferences and consequently non-random mating based on colour pattern components. For example, colour morphs in male guppies are under NFD (Olendorf *et al.*, 2006) and female preference for colour patterns vary within (Brooks and Endler, 2001b) as well as between (Endler and Houde, 1995) guppy populations.

On its own, NFD is a powerful mechanism to maintain polymorphism within populations (Clarke and O'Donald, 1964; Ayala and Campbell, 1974). Ecologically driven NFD can arise through frequency-dependent predation (Clarke, 1962; Allen and Greenwood, 1988) and/or competition for resources (Ayala and Campbell, 1974). Additionally, we know that on their own, mate preferences can promote the maintenance of sexual trait polymorphism (Servedio and Bürger, 2014; Ponkshe and Endler, 2018) and could also contribute to divergence of sexual traits and evolution of reproductive isolation (van Doorn *et al.*, 2004). However, we know little about how NFD might interact with variation in mate preferences. Given that these processes can frequently operate simultaneously in colour polymorphic species (Gray and McKinnon, 2007), it is important to examine under which conditions their interaction promotes the maintenance of polymorphism versus divergence in sexual traits. Here we identify conditions that promote versus constrain the maintenance of polymorphism and divergent fixation in sexual traits. We also ask what role directional natural selection (DNS) on mate preferences plays in these conditions. We use Kirkpatrick's standard haploid version of the classical model of sexual selection as a foundation to address these questions.

Kirkpatrick's haploid model (Kirkpatrick, 1982) is based on the Fisher process, which is considered to be a null process of trait-preference co-evolution (Prum, 2010). The Fisher process can produce a stable line of equilibria or a Fisherian runaway process. The line of equilibria is interpreted as evidence for traits-preference variation among populations. The presence of a stable line of equilibria implies design, meaning, and honesty in sexual signals (Fuller *et al.*, 2005; Prum, 2010). Populations can move along the line of equilibria, can diverge by chance, or speciation can occur due to differences in parameters and starting conditions (Kirkpatrick, 1982; Houde, 1993; Uyeda *et al.*, 2009; Prum, 2010).

Maintenance of sexual trait polymorphism becomes complicated when mate preferences are under directional selection (DNS) because even weak DNS on mate preferences is expected to deplete variation in sexual traits (Kirkpatrick and Ryan, 1991; Kokko *et al.*, 2006). The stable line of equilibria collapses to a single equilibrium point when mate preferences are under even weak DNS (Grafen, 1990; Kirkpatrick and Ryan, 1991; Prum, 2010), implying that even weak DNS on mate preferences can erode variation in sexual traits. In such cases, the interaction of NFD with mate preferences can possibly rescue the maintenance of sexual trait polymorphism when mate preferences are under DNS. However, we know little about how exactly NFD interacts with mate preferences when mate preferences are under DNS. Here we identify conditions under which the interaction of NFD

with mate preferences promotes versus restricts the maintenance of sexual trait polymorphism when mate preferences are affected by DNS.

Kirkpatrick's original model and subsequent extensions typically include DNS on sexual traits and/or on mate preferences (Seger, 1985; Seger and Trivers, 1986; Bulmer, 1989; Takahasi, 1997; Kuijper *et al.*, 2012) but do not include NFD. Here we build on Kirkpatrick's classical model and explore two sets of models. In the first set, we examine the interaction between mate preferences and NFD in the absence of DNS on mate preferences. In the second set, we examine the interactions between NFD and mate preferences when preferences are under DNS independent of mate choice. In this set of models, we explore two cases: when the machinery underlying mate preferences is sex-limited versus when it is not.

MODEL 1: ONLY NFD

Consider a haploid population exhibiting polymorphism in sexual traits and mating preferences. Assume that sexual traits are expressed in males and are controlled by locus T, whereas unlinked locus P controls mate preferences in females. Let locus T have two alleles T_1 and T_2 in males, which correspond to different sexual traits, and locus P have two alleles P_1 and P_2 in females, which correspond to different mate preferences (Ponkshe and Endler, 2018). The standard null model of selective mating (Kirkpatrick, 1982; Prum, 2010) normally assumes directional viability selection on sexual traits. Instead, here we assume sexual traits are affected by ecologically driven negative frequency dependence. Such scenarios can arise in natural populations of polymorphic species. For example, guppies show heritable variation in sexual traits (Brooks and Endler, 2001a) and mate preferences within populations (Brooks and Endler, 2001b). In addition, guppy populations also show frequency-dependent survival (Olendorf *et al.*, 2006). In such cases, sexual traits can be simultaneously under NFD and mate preferences.

Let m_1, m_2, m_3, and m_4 represent the starting frequencies of T_1P_1, T_1P_2, T_2P_1, and T_2P_2 zygotes in males and f_1, f_2, f_3, and f_4 be their founding frequencies in females. Let β be the strength of NFD. Consequently, since NFD works only on T in males, male genotype fitness measures are:

$$W_{T_1P_1} = 1 - \beta(m_1 + m_2); W_{T_1P_2} = 1 - \beta(m_1 + m_2);$$
$$W_{T_2P_1} = 1 - \beta(m_3 + m_4); W_{T_2P_2} = 1 - \beta(m_3 + m_4).$$

Let NFD on sexual traits occur before mating; this alters the frequencies of males available for selective mating. The frequencies of male genotypes available for selective mating after NFD are:

$$m_1{}' = \frac{m_1\big(1-\beta(m_1+m_2)\big)}{\overline{W}}; m_2{}' = \frac{m_2\big(1-\beta(m_1+m_2)\big)}{\overline{W}};$$
$$m_3' = \frac{m_3\big(1-\beta(m_3+m_4)\big)}{\overline{W}}; m_4' = \frac{m_4\big(1-\beta(m_3+m_4)\big)}{\overline{W}}.$$

where $\overline{W} = [m_1(1-\beta(m_1+m_2)) + m_2(1-\beta(m_1+m_2)) + m_3(1-\beta(m_3+m_4)) + m_4(1-\beta(m_3+m_4))]$.

Let P_1 females prefer T_1 males with the relative preference 1 and let their preference for T_2 males be $1-\alpha_1$. Similarly, let P_2 prefer T_2 males with the relative preference 1 and let their preference for T_1 males be $1-\alpha_2$. α_1 and α_2 are mate choice coefficients, where $\alpha_1 = \alpha_2 = 0$ means no choice with respect to male traits, and $\alpha_1 = \alpha_2 = 1$ means both females mate only with their preferred males. We assume discrete and non-overlapping generations. We obtained next-generation zygote frequencies by substituting male and female haplotype frequencies into the recurrence equations:

$$\begin{aligned} T_1P_{1(t+1)} = {} & f_1\left[\frac{m_1'}{z_1} + \frac{m_2'}{2z_1} + \frac{m_3'(1-\alpha_1)}{2z_1} + \frac{m_4'(1-\alpha_2)}{4z_1}\right] \\ & + f_3\left[\frac{m_1'}{2z_1} + \frac{m_2'}{4z_1}\right] + f_2\left[\frac{m_1'(1-\alpha_2)}{2z_2} + \frac{m_3'}{4z_2}\right] \\ & + f_4\left[\frac{m_1'(1-\alpha_2)}{4z_2}\right] \end{aligned} \quad (1)$$

$$\begin{aligned} T_1P_{2(t+1)} = {} & f_2\left[\frac{m_1'(1-\alpha_2)}{2z_2} + \frac{m_2'(1-\alpha_2)}{z_2} + \frac{m_3'}{4z_2} + \frac{m_4'}{2z_2}\right] \\ & + f_4\left[\frac{m_1'(1-\alpha_2)}{4z_2} + \frac{m_2'(1-\alpha_2)}{2z_2}\right] \\ & + f_1\left[\frac{m_2'}{2z_1} + \frac{m_4'(1-\alpha_1)}{4z_1}\right] + f_3\left[\frac{m_2'}{4z_1}\right] \end{aligned} \quad (2)$$

$$T_2P_{1(t+1)} = f_1\left[\frac{m_3'(1-\alpha_1)}{2z_1} + \frac{m_4'(1-\alpha_1)}{4z_1}\right]$$

$$+f_3\left[\frac{m_1'}{2z_1}+\frac{m_2'}{4z_1}+\frac{m_3'(1-\alpha_1)}{z_1}+\frac{m_4'(1-\alpha_1)}{2z_1}\right] \quad (3)$$
$$+f_2\left[\frac{m_3'}{4z_2}\right]+f_4\left[\frac{m_1'(1-\alpha_2)}{4z_2}+\frac{m_3'}{2z_2}\right]$$

$$T_2P_{2(t+1)} = f_2\left[\frac{m_3'}{4z_2}+\frac{m_4'}{2z_2}\right]$$
$$+f_4\left[\frac{m_1'(1-\alpha_2)}{4z_2}+\frac{m_2'(1-\alpha_2)}{2z_2}+\frac{m_3'}{2z_2}+\frac{m_4'}{z_2}\right] \quad (4)$$
$$+f_1\left[\frac{m_4'(1-\alpha_1)}{4z_1}\right]+f_3\left[\frac{m_2'}{4z_1}+\frac{m_4'(1-\alpha_1)}{2z_1}\right]$$

Here, $z_1 = m_1' + m_2' + (1 - \alpha_1)(m_3' + m_4')$, and $z_2 = (1 - \alpha_2)(m_1' + m_2') + m_3' + m_4'$.

We investigated the model behaviour in MATLAB 2015a by numerically computing equilibrium T_1 frequencies until there was no difference in allele frequencies among consecutive generations starting with T_1–P_1 frequencies that covered the entire parameter space.

For a given combination of NFD strength (β) and mate preferences (α_1 and α_2), joint trait-preference starting frequencies that will maintain polymorphism in T at equilibrium form a zone in the trait-preference frequency space (Fig. 1A). Following Ponkshe and Endler (2018), we refer to this zone as the polymorphic zone. The polymorphic zone has two distinct boundaries: the upper (U) and lower (L) boundaries based on their intersection on the axis of P_1 starting frequencies (Fig. 1A). U and L separate distinct evolutionary outcomes. If populations fall inside the polymorphic zone (between U and L inclusive), they remain polymorphic. Populations can either fix or lose the same trait allele if populations fall outside of the polymorphic zone (above U or below L). However, if populations happen to fall on the opposite sides of the polymorphic zone, then different sexual trait alleles will go to fixation.

We followed Ponkshe and Endler (2018) to identify the polymorphic zone and boundaries. First, for a given α_1–α_2 combination, we computed the equilibrium T_1 frequency for all possible combinations of T_1–P_1 starting frequencies. The polymorphic zone includes all T_1–P_1 starting frequencies that produce the equilibrium T_1 frequency between 0.001 and 0.999 ($0.001 < T_{1\,(\text{equilibrium frequency})} < 0.999$). To compute U, we identified a threshold P_1 for all T_1 such that any change in P_1 above U will give T_1 fixation, i.e. $T_{1\,(\text{equilibrium frequency})} > 0.999$ (see U in Fig. 1A). Similarly, to compute L, we identified a threshold P_1 for all T_1 such that any change in the starting frequency of P_1 below L will result in T_1 loss, i.e. $T_{1\,(\text{equilibrium}}$

$_{\text{frequency}}) < 0.001$ (see L in Fig. 1A). We will now discuss how NFD and mate preferences interact to affect the shape and size of the polymorphic zone located between U and L.

Effects of NFD and mate preferences on the shape and size of the polymorphic zone

For a given α_1–α_2 combination, the polymorphic zone is narrower when NFD is weak than when it is strong (Fig. 1B). The polymorphic zone gradually increases in size as β increases (note the systematic expansion between U and L with a gradual increase in β in Fig. 1B). Note that Figs. 1A and 1B include a scenario where NFD is weaker than both mate preferences (i.e. $\beta < \alpha_1$ and α_2). Consequently, as NFD strength increases, more trait-preference starting frequency combinations maintain sexual trait polymorphism and populations are less likely to fall on the opposite sides of U and L, which decreases the overall likelihood of fixation of different trait alleles.

Both U and L disappear as soon as NFD becomes stronger than mate preferences (i.e. when $\beta > \alpha_1$ and α_2). Figure 1D shows this general result (note the absence of U and L in Fig. 1D when $\beta > \alpha_1$ and α_2). When NFD is stronger than both mate preferences, populations remain polymorphic and neither can go to fixation or lose either trait allele or fix different trait alleles.

One of the threshold boundaries (either U or L) disappears when $\alpha_1 > \beta > \alpha_2$ or $\alpha_2 > \beta > \alpha_1$. L disappears when $\alpha_1 > \beta > \alpha_2$, and U disappears when $\alpha_2 > \beta > \alpha_1$. Figure 1C shows this result for $\alpha_2 > \beta > \alpha_1$. In such cases, the Fisher process overwhelms the effects of NFD in females showing stronger mate preference than NFD, leading to fixation of the matching trait allele. However, the Fisher process cannot overpower NFD in females showing weaker mate preference than NFD and consequently cannot lead to fixation of the matching trait allele (note that Appendix Fig. S1 shows equilibrium P_1 frequency when $\beta < \alpha_1$ and α_2, $\alpha_2 > \beta > \alpha_1$, and $\beta > \alpha_1$ and α_2; see evolutionary-ecology.com/data/3223Appendix.pdf).

These results show that sets of polymorphic populations can fix both T_1 and T_2 if, and only if, NFD is weaker than both mate preferences (i.e. $\beta < \alpha_1$ and α_2). In all other cases, populations either remain polymorphic or can fix or lose the same trait allele. Note that qualitative results described here hold true for the entire ranges of α_1, α_2 and β.

For a given NFD strength (β), and for a given set of T_1–P_1 starting frequencies, there is a polymorphic zone for combinations of α_1 and α_2 that maintain sexual trait polymorphisms. Figure 2 shows this general result under different NFD regimes for two sets of T_1–P_1 starting frequencies. For a given set of T_1–P_1 starting frequencies, the polymorphic

zone increases in size when NFD is strong than when it is weak (see the expansion in U and L when NFD increases from $\beta = 0$ to $\beta = 0.1$ in Figs. 2A and 2B). When NFD is strong, one of the thresholds disappears (note that U is absent in Fig. 2A and L is absent in Fig. 2B when NFD is strong, i.e. when $\beta = 0.7$). This result suggests that under strong NFD, sets of populations cannot fix sexual traits in opposite directions despite the strong differences in α_1 and α_2. This qualitative result holds true for all combinations of trait-preference starting frequencies (Fig. S2 in the Appendix shows this general result for more T_1–P_1 starting frequency combinations).

MODEL SET 2: SEXUAL TRAITS ARE UNDER NFD AND MATE PREFERENCES ARE UNDER DNS

Model 2.1: Males are affected by NFD; females are affected by DNS independent of mate choice

As before, we assume sexual traits are affected by NFD. In addition, we assume female preference is under the directional natural selection (DNS) caused by the environment independent of mate choice. Aside from this modification, the model is identical to model 1.

As in previous models, let m_1, m_2, m_3, and m_4 represent the starting frequencies of T_1P_1, T_1P_2, T_2P_1, and T_2P_2 zygotes in males and f_1, f_2, f_3, and f_4 be their founding frequencies in females. NFD on traits (T) in males occurs before mating, as in model 1. Let P_2 females have a disadvantage such that the P_2 viability is $1 - \gamma$ relative to P_1 females; γ is the viability selection coefficient ($0 \leq \gamma \leq 1$). Let DNS on females occur before mating. The frequencies of females available for mating after DNS are:

$$f_1' = \frac{f_1}{\overline{W}_{\text{females}}}; f_2' = \frac{f_2(1-\gamma)}{\overline{W}_{\text{females}}}; f_3' = \frac{f_3}{\overline{W}_{\text{females}}}; f_4' = \frac{f_4(1-\gamma)}{\overline{W}_{\text{females}}}$$

where $\overline{W}_{\text{females}} = [f_1 + f_2(1-\gamma) + f_3 + f_4(1-\gamma)]$. Female preferences are implemented as in model 1. Zygote frequencies in the next generation can be obtained by substituting f_1', f_2', f_3', and f_4' for f_1, f_2, f_3, and f_4 in equations 1–4. The computation procedure to solve recurrence equations and obtain U and L is the same as for model 1. Figure 3 illustrate the results of this model under weak ($\gamma = 0.05$) and strong ($\gamma = 0.3$) DNS for different NFD regimes. The majority of DNS estimates on morphological and phenological

traits reported in natural populations are less than 0.15 and few are greater than 0.5 (Hoekstra *et al.*, 2001).

Combinations of α_1 and α_2 that maintain sexual trait polymorphisms form a polymorphic zone in the α_1–α_2 state space (Fig. 3A). The lower threshold (L) of the polymorphic zone disappears when mate preferences are under DNS. This is because under DNS, the disadvantageous preference allele (P_2) cannot fix its matching trait allele (T_2) and thus L disappears. Consequently, in such cases, differences in mate preferences can produce two outcomes: populations can either remain polymorphic if they fall anywhere inside the polymorphic zone (i.e. below U), or the advantageous trait allele (T_1) is fixed (i.e. populations above U).

Figures 3B and 3C show the permissiveness of polymorphism (size of the polymorphic zone) in different NFD regimes under strong ($\gamma = 0.3$) and weak ($\gamma = 0.05$) DNS respectively. It is important to note that differences in DNS strength (γ) have very little effect on the position, shape, and size of the polymorphic zone [(note that the shape and position of U for different values of β remain unchanged under strong (Fig. 3B) and weak (Fig. 3C) DNS regimes], whereas NFD and α_1 have a far stronger effect on the position and size of the polymorphic zone; the relative strengths of NFD (β) and α_1 determine the position of U.

For a given DNS regime, populations can maintain polymorphism in T if, and only if, the strength of NFD is greater than the preference strength of the advantageous preference allele, P_1 (i.e. $\beta > \alpha_1$; compare the position of U with respect to α_1 for different values of β in Figs. 3B and 3C). As soon as the preference strength exceeds the strength of NFD (i.e. $\beta < \alpha_1$), the corresponding trait T1 is fixed, regardless of the strength of the disadvantageous preference allele α_2 (horizontal nature of U for different NFD strengths in Figs. 3B and 3C shows that α_2 does not affect the polymorphism maintenance of T under strong and weak DNS regimes respectively). Note that the Fig. S3 in the Appendix shows the corresponding P_1 equilibrium frequency.

Model 2.2: Males are affected by NFD and DNS; females are affected by DNS (machinery underlying mate preferences is not sex-limited)

In many cases, factors affecting mate preference are not sex-limited, such as when mate preferences are co-opted from other traits and sensory system properties determine mate preferences (Ryan, 1990; Endler, 1992; Endler and Basolo, 1998). In such cases, if sensory systems are under DNS as a result of their physical environment, then both males and females can be

under DNS independent of mate choice. Here we model this scenario. Aside from this modification, the model is identical to model 2.1.

As in previous models, let m_1, m_2, m_3, and m_4 represent the starting frequencies of T_1P_1, T_1P_2, T_2P_1, and T_2P_2 zygotes in males and f_1, f_2, f_3, and f_4 be their founding frequencies in females. For this model, the fitness measures of male genotypes are:

$$W_{T_1P_1} = 1 - \beta(m_1 + m_2); W_{T_1P_2} = 1 - \beta(m_1 + m_2) - \gamma;$$
$$W_{T_2P_1} = 1 - \beta(m_3 + m_4); W_{T_2P_2} = 1 - \beta(m_3 + m_4) - \gamma.$$

Let NFD and DNS occur before selective mating; this alters the frequencies of males available for mating. The frequencies of males available for mating after NFD and DNS are now:

$$m_1{}' = \frac{m_1(1 - \beta(m_1 + m_2))}{\overline{W}_{\text{males}}}; m_2{}' = \frac{m_2(1 - \beta(m_1 + m_2) - \gamma)}{\overline{W}_{\text{males}}};$$
$$m_3' = \frac{m_3(1 - \beta(m_3 + m_4))}{\overline{W}_{\text{males}}}; m_4' = \frac{m_4(1 - \beta(m_3 + m_4) - \gamma)}{\overline{W}_{\text{males}}}.$$

where $\overline{W}_{\text{males}} = \big[\big(m_1\,(1 - \beta(m_1 + m_2)\big) + \big(m_2\,(1 - \beta(m_1 + m_2) - \gamma)\big) + \big(m_3(1 - \beta(m_3 + m_4)\big) + \big(m_4\,(1 - \beta(m_3 + m_4) - \gamma)\big)\big]$. Here DNS on preference (P) in females occurs before mating, as in model 2.1.

When both sexes are affected by DNS, the effective DNS intensity (γ) is doubled. However, similar to model 2.1, differences in DNS strength do not alter the shape and position of U. The shape and position of U for different values of β remain unchanged under model 2.1 (Fig. 3C) and model 2.2 (Fig. 3D).

DISCUSSION

To what extent does the interaction of mate preferences and mating-independent negative frequency-dependent selection (NFD) contribute to the maintenance and divergence of polymorphic sexual traits? Our results show that environments with mate preferences stronger than NFD are more likely to permit the fixation of sexual traits in opposite directions (divergent fixation) than environments with mate preferences weaker than NFD. We also

show that strong NFD prevents the divergent fixation of sexual traits despite the strong differences in mate preferences.

Selection via survival tends to be weaker than selection via mating success in natural populations (Hoekstra *et al.*, 2001; Kingsolver *et al.*, 2001). This also holds for polymorphic traits (Endler, 1986). Given this, conditions in which NFD is weaker than mate preferences may occur frequently in natural populations. Our results suggest that in such environments, isolated polymorphic populations with different starting trait-preference allele frequencies can fix sexual traits in different directions. Consequently, frequency fluctuations in starting trait-preference alleles by chance can lead populations to fixation of different sexual trait alleles in such environments.

In contrast, the risk of loss of polymorphism is significantly lower in environments with mate preferences weaker than NFD. Such conditions prevent the divergent fixation of sexual traits. These results suggest that in the face of trait-preference allele frequency fluctuations, sets of polymorphic populations with NFD stronger than mate choice parameters are less likely to produce divergent fixation by frequency fluctuations alone. The early stages of sexual trait divergence could stall under such scenarios, and further parameters may need to change or evolve before divergence can be completed.

Colour polymorphic species affected by NFD can show variation in female preferences for male traits. For example, guppy populations are under NFD (Olendorf *et al.*, 2006) and also show geographic variation in mate preferences (Endler and Houde, 1995). Our results suggest that in such cases, strong NFD can prevent the divergent fixation of sexual traits despite the strong differences in mate preferences among populations (note that either U or L disappears when NFD is strong in Fig. 2). Sets of populations strongly differing in mate preferences are more likely to fix sexual traits in opposite directions under weak NFD regimes.

Effect of DNS on polymorphism maintenance and the divergence in NFD-affected polymorphic sexual selected traits

In many species, mate preferences can be under direct selection independent of mate choice. Such conditions can arise in polymorphic species where female preferences have pleiotropic effects that affect female survival via effects unrelated to mate preferences (Kirkpatrick and Ryan, 1991; Maan and Seehausen, 2011). Strong directional viability selection is relatively less common in natural populations than weak selection, but strong selection still occurs

frequently (for further discussion, see Hoekstra *et al.*, 2001). Oure results suggest that DNS on mate preferences in the presence of NFD can stall the divergent fixation of sexual traits when DNS is unidirectional.

Interestingly, we show that the Fisher process interacts with NFD in an identical way under weak and strong DNS regimes. In many cases, factors affecting mate preference are not sex-limited. Such a scenario can arise in various contexts, such as when mate preferences are co-opted due to sensory bias. For example, if the visual system that affects guppy mate preferences is under environmental selection, independent of mate choice (Endler, 1991; Sandkam *et al.*, 2016), then both females and males will also be affected by the DNS caused by the sensory environment; the senses are used for functions in addition to mate choice. Our results show that in such cases, the Fisher process will interact with NFD in ways similar to when DNS is sex-limited.

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AUTHOR CONTRIBUTIONS

Models were designed and analysed by A.P. The manuscript was prepared by A.P. and was revised by J.A.E.

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Fig. 1. Effects of selection parameters (α_1, α_2, and β) on polymorphic zones. (A) Phase map showing attraction basin of polymorphic equilibria (polymorphic zone), delimited by two thresholds, U and L, when $\beta < \alpha_1$ and α_2 for $\beta = 0.3$, $\alpha_1 = 0.4$; $\alpha_2 = 0.7$. (B) Changes in U and L as a function of NFD strength (β) when $\beta < \alpha_1$ and α_2 for $\alpha_1 = \alpha_2 = 0.8$. (C) Phase map showing polymorphic zone when $\alpha_2 > \beta > \alpha_1$ for $\beta = 0.8$, $\alpha_1 = 0.2$, $\alpha_2 = 0.9$ (note that U is absent). (D) Phase map showing polymorphic zone when $\beta > \alpha_1$ and α_2 for $\beta = 0.8$, $\alpha_1 = 0.2$, $\alpha_2 = 0.6$.

Fig. 2. Consequences of interaction between the ecologically driven NFD and mate preferences in the absence of DNS on mate preferences. (A) Changes in U and L as a function of NFD strength (β) – no NFD ($\beta = 0$), weak NFD ($\beta = 0.1$), strong NFD (β=0.7) – when starting T_1 frequency = 0.1, starting P_1 frequency = 0.1. Note that U disappears when NFD is strong ($\beta = 0.7$). (B) Changes in U and L as a function of NFD strength (β) – no NFD ($\beta = 0$), weak NFD ($\beta = 0.1$), strong NFD ($\beta = 0.7$) – when the starting T_1 frequency = 0.4, starting P_1 frequency = 0.9. Note that L disappears when NFD is strong ($\beta = 0.7$).

Fig. 3. Joint effects of NFD, DNS, and mate preferences on the maintenance of polymorphism. (A) Phase map showing polymorphic zone, delimited by threshold U under weak NFD and weak DNS ($\beta = 0.05$, $\gamma = 0.05$) when DNS is sex-limited (model 2.1). (B) Changes in threshold boundaries as a function of β under strong DNS ($\gamma = 0.3$) when DNS is sex-limited (model 2.1). (C) Changes in threshold boundaries as a function of β under weak DNS ($\gamma = 0.05$) when DNS is sex-limited (model 2.1). (D) Changes in threshold boundaries as a function of β under weak DNS ($\gamma = 0.05$) when DNS is not sex-limited (model 2.2). This figure shows results for starting T_1 frequency = 0.1, starting P_1 frequency = 0.1. However, note that these results hold true for the entire range of T_1–P_1 starting frequencies.

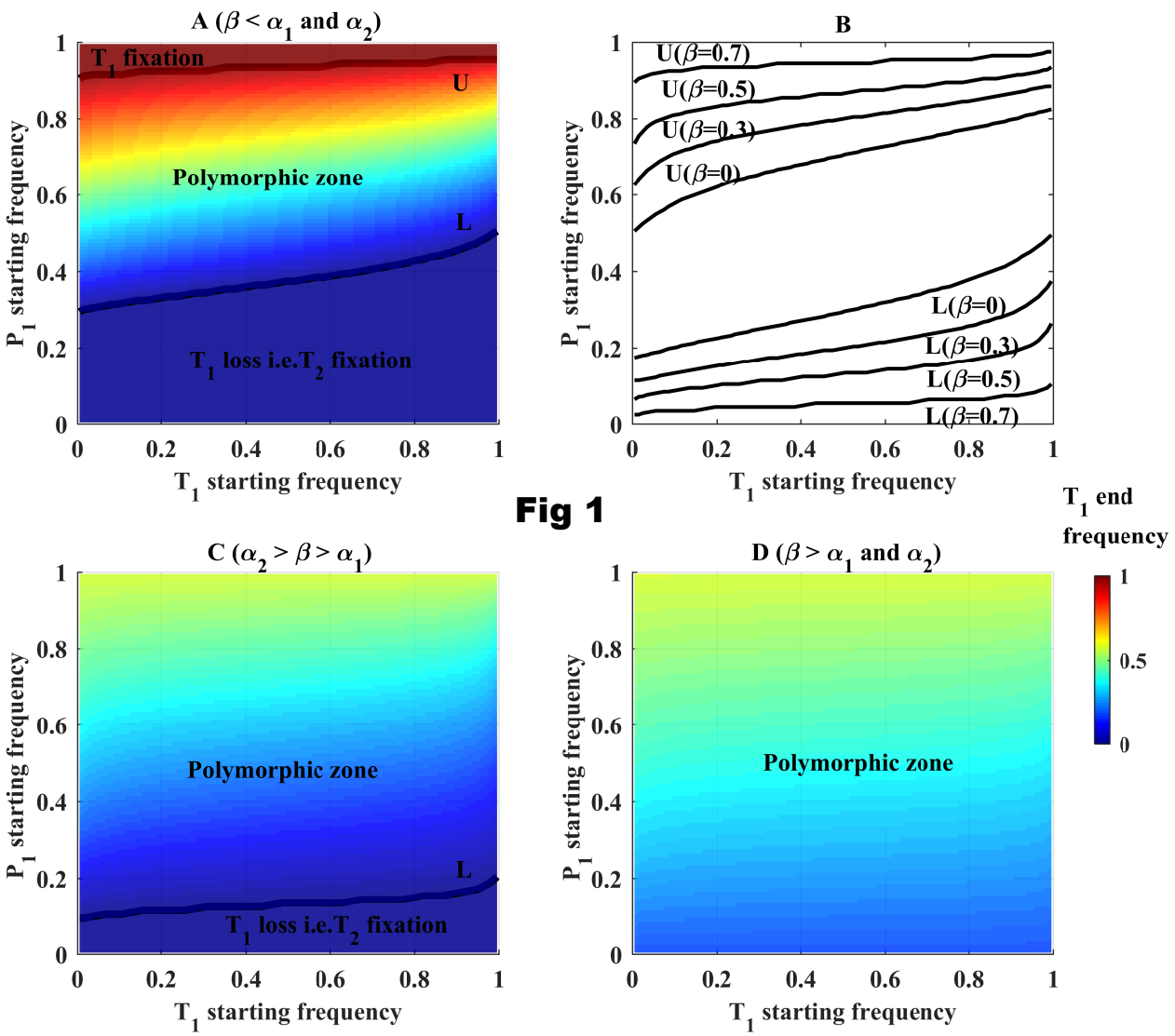

A ($\beta < \alpha_1$ and α_2)
T_1 fixation
U
Polymorphic zone
L
T_1 loss i.e.T_2 fixation
P_1 starting frequency
T_1 starting frequency
B
U(β=0.7)
U(β=0.5)
U(β=0.3)
U(β=0)
L(β=0)
L(β=0.3)
L(β=0.5)
L(β=0.7)
P_1 starting frequency
T_1 starting frequency
C ($\alpha_2 > \beta > \alpha_1$)
Polymorphic zone
L
T_1 loss i.e.T_2 fixation
P_1 starting frequency
T_1 starting frequency
D ($\beta > \alpha_1$ and α_2)
Polymorphic zone
P_1 starting frequency
T_1 starting frequency
T_1 end frequency
1
0.5
0

Fig 1

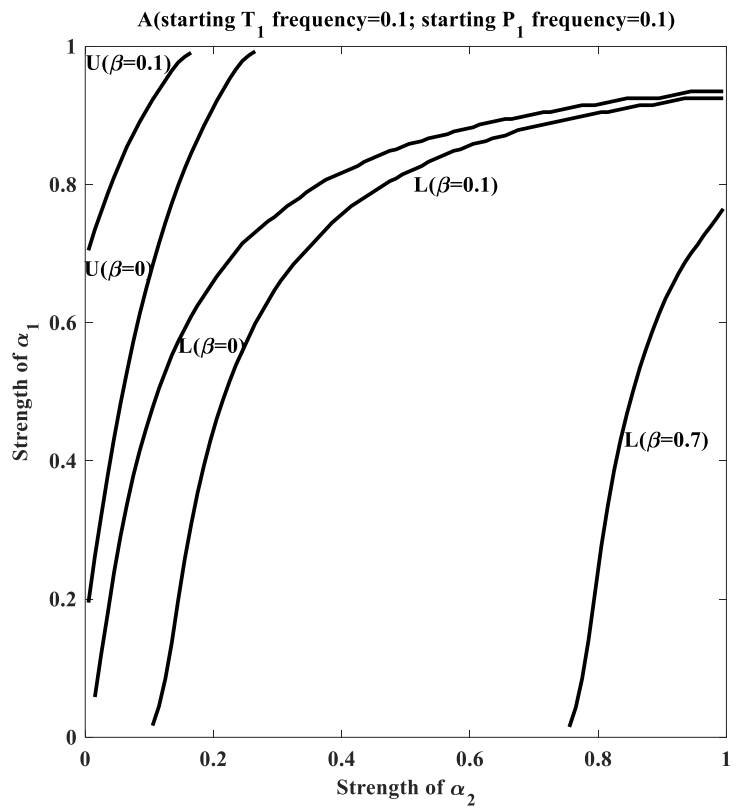

A(starting T1 frequency=0.1; starting P1 frequency=0.1)
U(β=0.1)
U(β=0)
L(β=0.1)
L(β=0)
L(β=0.7)
Strength of α1
Strength of α2
0
0.2
0.4
0.6
0.8
1

Fig 2

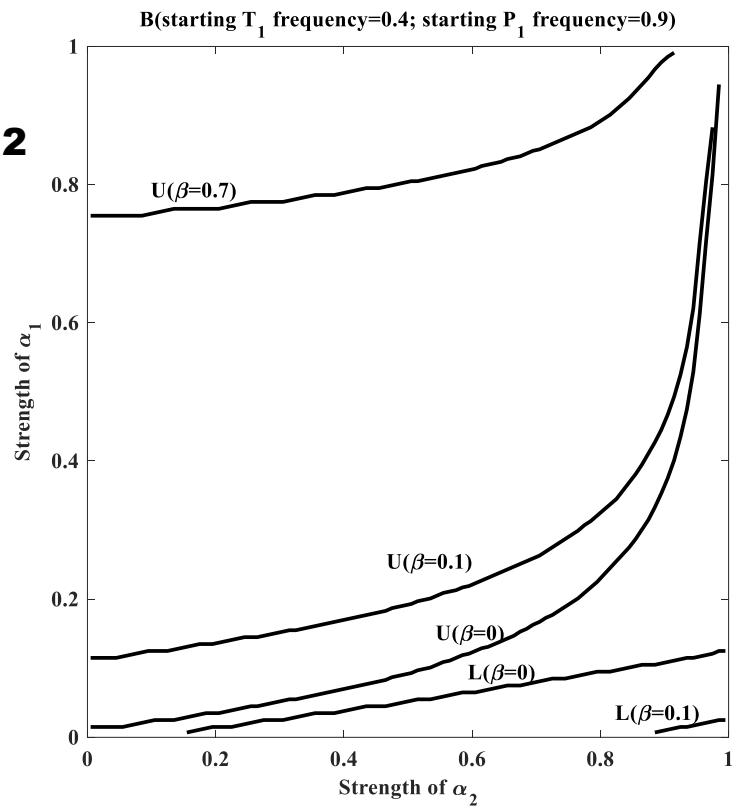
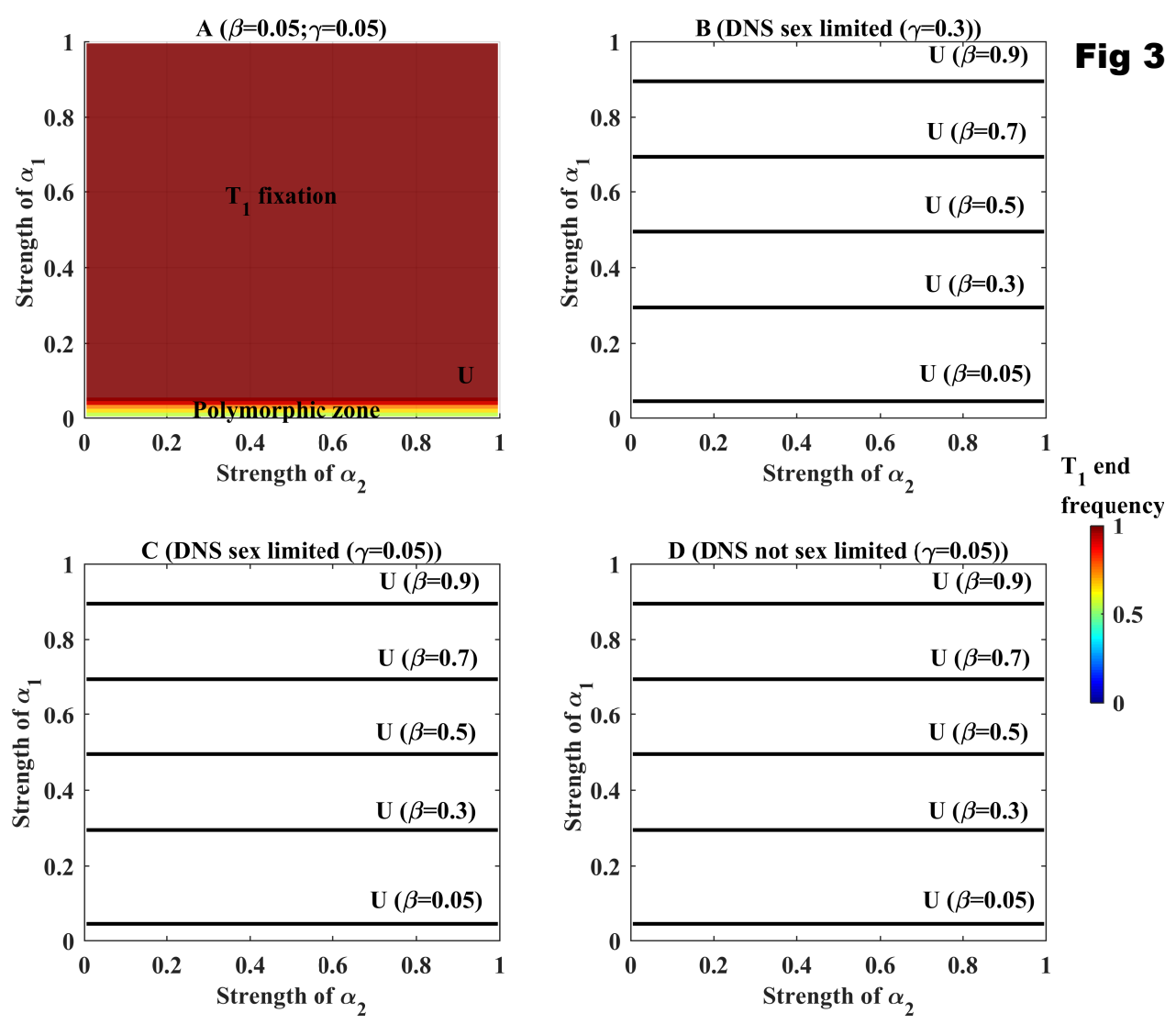

B(starting T1 frequency=0.4; starting P1 frequency=0.9)
U(β=0.7)
U(β=0.1)
U(β=0)
L(β=0)
L(β=0.1)
Strength of α1
Strength of α2
0
0.2
0.4
0.6
0.8
1

Fig 3


A (β=0.05;γ=0.05)
T1 fixation
U
Polymorphic zone
Strength of α1
Strength of α2
B (DNS sex limited (γ=0.3))
U (β=0.9)
U (β=0.7)
U (β=0.5)
U (β=0.3)
U (β=0.05)
C (DNS sex limited (γ=0.05))
D (DNS not sex limited (γ=0.05))
T1 end frequency
1
0.5
0