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New Idea

Do gynogenetic species escape evolving enemies?

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The problem

Gynogenesis is a form of 'sexual parasitism' wherein one sex (the female) attracts a heterospecific mate for the purposes of mating, but not for reproduction (Hubbs 1964; Lehtonen et al. 2013). The heterospecific sperm are used to initiate embryogenesis of an unreduced egg after plasmogamy, but the sperm's genetic component is coalesced and rendered dysfunctional thus skipping the fusion of the female and male nuclei or kariogamy (Schlupp 2005; Lehtonen et al. 2013). In this scenario, reported among 7 metazoan phyla (Beukeboom and Vrijenhoek 1998) including vertebrates like poeciliid fishes (Schultz 1967; Schultz and Kallman 1968) and *Ambystoma* salamanders (Bi and Bogart 2010), the 'asexual' females exist in the same 'population' as males and females of the sexual species. It is better to refer to this 'population' as a mixed species assemblage with males of one species mating with females of two species.

One key question that arises is why all the gynogenetic females do not just reproduce entirely asexually through parthenogenesis, i.e., evolve to forgo egg development that is dependent on attracting a heterospecific male? This question might be important to informing ideas about the evolution of (a)sex because it brings to the forefront a troubling situation: the gynogenetic mode of reproduction apparently combines disadvantages of both sexual and asexual strategies (Beukeboom and Vrijenhoek 1998; Schlupp 2005; Lamatsch and Stöck 2009). Intriguingly, gynogenetic lineages escape extinction longer than predicted based on their asexuality (Spolsky et al. 1992; Schartl et al. 1995). Here, we propose a novel mechanism by which gynogenetic populations might achieve long-term stability in spite of their asexuality. We argue that sperm-dependency prevents the asexual species from outcompeting the sexual species, as previously suggested by Beukeboom and Vrijenhoek (1998), and furthermore that it is this obligate formation of mixed-species assemblages that prevents population crashes of asexuals (gynogens) by slowing down or averting parasite adaptation.

Costs and benefits of sex—the Red Queen Hypothesis

Before attempting to explain the evolutionary persistence of gynogenetic lineages, it might be useful to review the proposed costs and benefits of sex. Sexuality involves the reduction of ploidy through meiosis, the recombination and independent segregation of chromosomes, and fertilization by kariogamy. It evolved simultaneously with eukaryotes or soon after them (Cavalier-smith 2002). As a consequence, sexuality is the most prevalent mode of reproduction in metazoans (Bell 2007) and eukaryotic asexuals have evolved from alterations of the regular meiotic cycle (Bengtsson 2009). However, it is still unclear which are the main mechanisms that provide an advantage to sexual organisms over that small fraction of metazoans that reproduce entirely asexually (<0.1% -Vrijenhoek 1998).

Sexuality and recombination have been considered troubling issues for a long time (Van Valen 1973). One of the main problems of sex is that investment in males

could have a two-fold cost relative to investment in only asexual individuals, which instead just produce female offspring that contribute directly to population growth rates. This two-fold cost is the upper limit of the theoretical difference in reproduction for completely outbreeding individuals (Bell 1982) and is only expected under the unrealistic condition of "all else being equal" (i.e. niche, fitness, reproductive output) between the sexual and asexual lineages. Another issue with sexuality, known commonly as the problem of recombination, states that sex can be detrimental to organisms that are adapted to their environment because it can break up beneficial gene associations (Nei 1967). Yet through this reshuffling, recombination allows sexual individuals to eliminate organisms riddled with deleterious mutations (Muller 1932) and to adapt more rapidly than asexuals to changes in their environment through the production of novel gene associations and increased genetic variance (but see Agrawal (2006)). Asexuals adapt to changes at a slower rate than do sexuals and predominantly do so through the occurrence of new beneficial mutations (Fisher 1930; Muller 1932). But put simply, sexuals are thought to have an advantage over asexuals as long as their environment constantly changes making yesterday's adaptive traits today's maladaptive ones.

There has been much debate as to what could generate sufficient change in the direction and strength of selection to maintain an advantage for sexuality. Antagonistic interactions, particularly those caused by parasites and disease agents, could generate the continually changing environment that would provide sexual organisms with an advantage over asexuals, even given the costs of sex, meiosis and mating (Maynard-Smith 1978; Bell 1982; Hamilton 1982; Jaenike 1978; McKinnon et al. 2004). Inspired by Lewis Carol's Alice in Wonderland, this hypothesis was dubbed the Red Queen Hypothesis (RQH), and alludes to sexual species constantly having to evolve adaptations and counter-adaptations to keep the *status quo*. The thinking is as follows: parasite fitness hinges on their ability to have increased transmission, establishment and reproduction through the use of host resources, which leads to negative impacts on host fitness and selection for traits that allow the host to control or eliminate parasite infection, which in turn feeds back to selection on the parasites. Other proposed advantages to sex include the elimination of individuals that accumulate deleterious mutations (Muller 1932), a faster rate of integration of favorable mutations that originated in different individuals (Burt 2000; Colegrave 2002), and/or increased ecological opportunities of variable offspring (Bell 1982). It would thus seem reasonable that the combination of these factors produces a sizeable advantage to sexuals over asexuals (e.g. Howard and Lively 1994). Notwithstanding, we focus on co-evolutionary dynamics between parasites and their hosts in our treatment of the evolution of gynogenetic reproduction.

Can insights offered by the RQH inform ideas about the persistence of gynogenetic lineages? As mentioned, gynogenetic asexuals seem to be combining disadvantages of sexual and asexual reproductive strategies. Even if they do not produce males, gynogenetic females spend time and energy in mating, with all the potential risks that mating entails (e.g. aggression, predation risk, disease transmission, Daly 1978). Gynogens pay these costs or entail these risks while also experiencing the costs of asexuality such as the accumulation of deleterious mutations within the genome or lower genetic diversity than sexuals, which is thought to compromise the asexual's ability to evolve counter-adaptations such as resistance to parasites and disease agents. Therefore, the reality that gynogenetic lineages tend to persist longer than other asexual lineages poses a paradox.

Tests of premises of the RQH have been done on gynogenetic species in mixed species assemblages with sexual species. In 1990, Lively and his colleagues did a study on closely related fishes (*Poeciliopsis* spp.): they compared parasite levels on assemblages of both abundant and less frequent asexual (female) lineages coexisting with sexual females. Their finding confirmed that abundant asexuals had higher parasitemia than less abundant asexuals or sexuals, suggesting that parasites were tracking the most common asexual host genotypes. This is the sort of dynamic that is required by models on parasite-mediated selection to explain sex (Hamilton 1982) and highlighted the fact that outbreeding/ recombination might provide sexuals with a moving target for parasite adaptation while asexuals appear to be trapped within a fixed recognition or resistance profile that provides a static target for parasite adaptation. Asexuality should be particularly deleterious when the asexuals are in low-diversity assemblages.

Since the study by Lively et al. (1990), a number of other studies have compared frequent and less abundant asexuals (and sexuals) of the same species often finding that abundant asexuals with low genotypic diversity have higher parasitism, again supporting premises of the RQH (although there are nuances, cf. Morran et al. 2011). It would seem that gynogens behave like pure parthenogens and thus might be expected to suffer a population crash in low-diversity assemblages and in the relative absence of sexuals. This interpretation opens the door to a novel insight regarding gynogenetic individuals: could their presence in two-species assemblages, where one species is sexual, provide gynogens with some of the benefits of pure sexual groups in terms of escape from evolving enemies? Testing this idea requires more than simply comparing levels of parasiteism experienced concurrently by asexual and sexual individuals.

Experimental approaches and key insights

We propose experimental approaches to test our verbal model. We argue it is worth asking if the population dynamics of gynogens would differ if they were alone versus when they are in association with sexual individuals, in the presence of parasites. It is also worth addressing the fitness of parasites attacking both gynogenetic and sexual females, given the different population contexts. In our reasoning and descriptions, it is assumed that both sexuals and asexuals are attacked by the same parasite species.

Experimental tests of parasite evolution on gynogenetic versus sexual hosts are needed as a baseline, where host and parasite fitness and genotypes are tracked over many generations. These types of experiments can be done with sexuals and gynogens in separate artificial populations. The predicted outcome of such an experiment is that initially the gynogen-alone population will increase much faster than the sexual-alone population. Beyond a certain time step determined by the rate of evolution of the parasite, we would predict dynamics in the gynogen-alone population to be characterized by boom and bust cycles of particular genotypes if such populations were initially diverse, or a sustained decrease in population size through time if such populations initially have low genetic diversity. In comparison, we might predict much more consistent stable population dynamics in the sexual host population as different host-defense and parasite-virulence genotypes cycle.

One particular challenge of using gynogenetic hosts is that experimental females in gynogen-alone assemblages would have to be periodically 'mated' with sexual males just to keep the population from crashing because of their sperm dependency (an effect unrelated to parasite-mediated selection). Importantly, keeping gynogens alone also would reduce the associated costs of mating paid by gynogens when in constant association with a sexual population. One control for such effects would be to expose sexual females to males for the same duration as gynogenetic females are exposed to males. The first prediction then is that populations composed only of asexual individuals will crash more frequently than populations composed only of sexual individuals (Figure 1).

Measures of parasite fitness should also be taken in these experiments. All trials should start with the same pool of parasites, to control for initial differences in parasite traits/strains potentially explaining any differences across treatments. Parasite mean abundance or prevalence could be measured on their contemporary hosts and be used to infer changes in parasite adaptation to hosts through time, although the specific type of parasite used (e.g. Lafferty and Kuris 2002) is likely to determine the choice of fitness correlate. Based on the above gynogen-alone and sexual-alone treatments, one prediction is that parasites will increase in abundance/ prevalence with consecutive generations of hosts when those hosts are unable to counter parasite adaptation. Thus, low diversity assemblages of gynogens should have more parasites with increasing generation-distance from the ancestral hosts. The prediction for sexual-alone assemblages is that there will be no change, or only transient changes, in parasite fitness when comparing parasites from different time periods to parasites from the ancestral population. Care should be taken to match parasite and host at the appropriate temporal scales (e.g. Lively 1999). If parasite adaptation is happening and accounting for the demise of gynogens, the prediction here is that parasites should have higher establishment, transmission and growth rates (i.e. fitness) on those gynogenetic hosts in which they evolved rather than on other (allopatric) gynogenetic hosts or sexual hosts (Figure 2). One important caveat is that gynogens might be suffering from mutation accumulation and other consequences of low genetic diversity that have an impact on host vigor, in which case we might expect to see higher parasite fitness on gynogens irrespective of the parasites' origin (for example, using parasites evolved on sexuals in studies involving cross-infection of asexuals). If crashes of artificial populations of gynogens (and not sexuals) occur using parasites collected from mixed assemblages in nature, the inference is that the gynogens in mixed species assemblages are somehow protected in nature from evolving enemies targeting them.

It would be worthwhile to assess further any proposed benefits of being in mixed species assemblages. Another prediction is that most parasite strains, independent of their co-evolutionary history, will have lower fitness on mixed-species groups (combined gynogenetic and sexual hosts) than on gynogen-alone groups because of the increased diversity of host recognition or

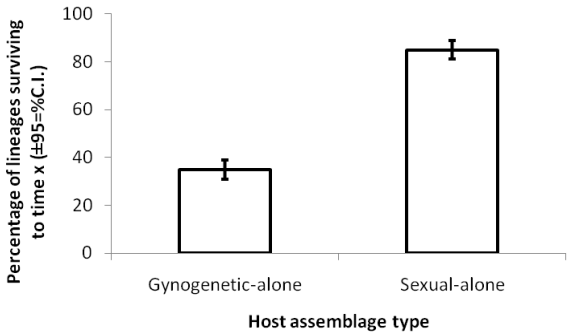

Figure 1. Expected difference in percentage of surviving gynogenetic-alone and sexual-alone assemblages after multiple generations of host-parasite coevolution. The actual values are arbitrary.

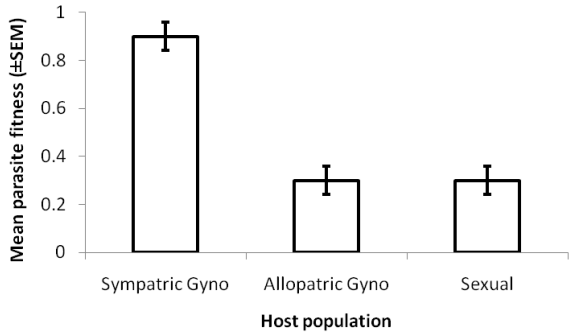

Figure 2. Expected differences in mean parasite fitness on hosts with different reproductive modes for parasites that co-evolved with sympatric gynogenetic-alone assemblages. The actual values are arbitrary.

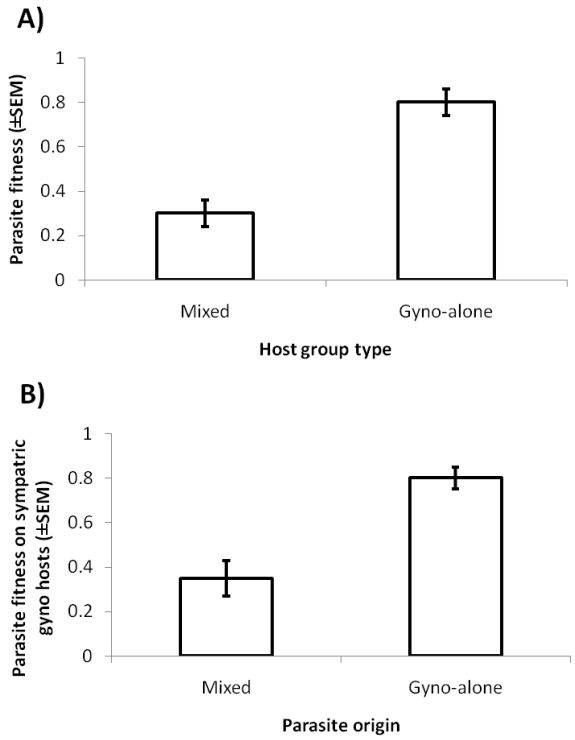

Figure 3. Expected differences in parasite mean fitness for parasites derived from all hosts when infecting mixed assemblages of sexual and gynogenetic hosts or gynogen-alone assemblages (A); and expected differences in parasite mean fitness on sympatric gynogenetic hosts for parasites that have either evolved on mixed or gynogen-alone assemblages (B). Actual vallues are arbitrary.

defense genotypes in mixed-groups (Figure 3) which will interfere with parasite transmission and growth rates (e.g. Altermatt and Ebert 2008). Also, parasite strains that have co-evolved with hosts in mixed-species groups will have lower fitness on gynogenetic hosts than parasite strains that have co-evolved with hosts in gynogen-alone assemblages, provided the initial gynogenetic host strain is derived from the same ancestor in both mixed- and alone- assemblages (Figure 3). This prediction is based on the assumption that mixed-species groups provide a larger diversity of targets and thus slows down the rate of parasite adaptation. Such a result would be evidence in support of benefits of sperm dependency of gynogenetic species. One possible alternative outcome is that parasites that have co-evolved in groups of hosts with higher diversity might themselves be more diverse, than parasites that have been evolving with low diversity (gynogenetic) hosts, and therefore have a greater adaptive potential when faced with abundant novel hosts.

In the paragraphs above, two essential points are made with respect to attempting to understand the persistence of gynogenetic lineages. The first is that parasite evolution has to be more thoroughly investigated in asexual gynogenetic species over several to many generations. The second is we need to perturb the natural stable-state to produce scenarios that do not exist in nature (cf. Travis et al. 2014), such as populations with only gynogens. Natural situations probably have already reached a certain degree of stability and comparing, for example, naturally co-occurring asexuals and sexuals with respect to parasite loads will just tell us what is currently happening and not what is possible, or not possible.

Gynogenetic females might persist in mixed assemblages because their greater reproductive capacity allows them a short-term competitive advantage over sexuals, but also specifically *because* they are obligate in assemblages with sexuals and this interferes with the parasites adapting to abundant asexual clones. We might even expect abundant asexual clones to be more parasitized than sexuals, but far away from the point of risking a population crash because the parasites have not evolved to exploit the asexual hosts as strongly as they would have if only asexuals were in the population (a finding that is consistent with previously reported empirical evidence). One could ask whether the proportion of gynogens found in mixed species assemblages is explained by a combination of selection imposed by parasites and selection imposed by sperm dependence where lack of males leads to a decline in the gynogenetic population and some recovery of the sexual population (Beukeboom and Vrijenhoek 1998). Alternatively, changes in the relative abundance of sexuals could modify male behavior and lead to frequency-dependent mating success of the gynogenetics (e.g. Moore 1975). Both instances could then lead to some stable coexistence between the two species. But one question is whether or not this stable coexistence is at the same level as when parasites are

present, i.e. whether the relative fitness benefits and/or costs of forming mixed-assemblages changes with parasitism for each reproductive strategy.

In summary, we suggest that sperm dependency would stabilize sexual populations from being out-competed by rapidly increasing gynogens and place a limit on the upper boundary of gynogenetic populations. We argue that by forcing gynogens to be in close association with sexual species, sperm dependency produces a diverse assemblage of hosts and slows parasite adaptation to gynogens. We further argue that by holding gynogenetic populations in check, sperm dependency allows gynogens to escape evolving parasites that would otherwise rapidly evolve increased virulence to them. Having smaller gynogenetic populations could then lead to lower parasite abundances, and thus reduced adaptive potential of parasites, further stabilizing the gynogenetic species in the assemblage.

Co-evolutionary dynamics: a closer look

Our argument explores the extent to which tests of some of the premises of the RQH can inform our understanding about the persistence of gynogenetic lineages. Intriguingly, selection is envisioned by the RQH to operate in two different ways. Selection can be negative frequency dependent (i.e. balancing selection) where there is *rapid* and *continuous* fluctuation of the alleles, already present in the population, that are adaptive to both host and parasites. Selection is also envisioned to operate at times through an arms-race dynamic (i.e. directional selection) where hosts and parasites express increasing levels of defense and virulence through novel allele or gene combinations. In this sense, sexual hosts are able to keep up with parasite evolution by presenting them with "novel" genetic combinations acquired through recombination and segregation, while the asexual hosts are more limited in the rate at which they can produce novel combinations mainly through mutations, mitotic recombination or in some instances automixis, and might not be able to do so fast enough to control the parasite population. But what happens when selection imposed by parasites is diffused across two host species (one sexual and one asexual)? It is quite possible that asexuals could be considered just as different host strains and incorporated into negative frequency dependent models (such might be especially relevant when the parasite is specialized more so on a host tissue than a host species *per se*). It is more difficult to envision arms races with two host species against a single parasite species. Performing direct tests of these dynamics by cross-infecting hosts from different generations with co-evolved parasites from those same generations (e.g. past, present, future - cf. Decaestecker et al. 2007) could be informative, yet would require both hosts and parasites with resting stages. We currently do not know of such associations in nature involving gynogenetic species. What we do expect is that gynogens alone could be in either arms races or negative frequency dependent selection, which would ultimately lead to selective sweeps of some genotypes leading to population reductions and possible extinction. In mixed species assemblages, we expect dampening of selective sweeps and/or disruption of parasite-led arms races.

The generality of the protective effect of mixed-species assemblages

Comparisons of the gynogen-alone versus mixed-species assemblage can serve as a proxy for the difference in dynamics that could be expected between true parthenogens that do not require sperm and gynogenetic species. This comparison could provide a coherent explanation as to why gynogenetic asexual lineages are more stable than generally predicted by theory. One could ask how successful an asexual (and not necessarily gynogenetic) clone is with respect to population persistence over time when in a simplified assemblage of only asexuals versus a mixed assemblage with sexual species, when parasites are present. In nature, the point is often moot as associations of sexual and asexual lineages might often be only transient.

Notwithstanding, the same tests we propose above could be performed in true parthenogenetic species. Such tests could inform our hypothesis on the importance of sperm dependency, but without the associated costs of mating for the asexual (non-gynogenetic) species and the extent to which mixed-species associations are a more general defensive mechanism.

We hypothesize that mixed-assemblages would stabilize not only gynogenetic populations but also true parthenogenetic individuals, but only as long as they actively form mixed assemblages with sexuals. Although multiple species form stable heterospecific assemblages to avoid predation (Thiollay 1999), increase foraging efficiency (Hino 2000) or avoid parasitism (Dargent et al. 2013), population stability may not be a deterministic outcome of mixed assemblages. True parthenogens do not require sexuals for their short-term survival, and therefore could outcompete sexuals locally (if they are initially diverse or parasitism is low), thereafter these parthenogen-alone assemblages will be left to selective sweeps and population crashes expected by the logic of the RQH. One relevant question is to what extent do true parthenogenetic asexuals exist in nature with sexual species and share the same parasites, or is this a phenomenon restricted to species assemblages involving gynogens?

Concluding remarks

The above paragraphs make the point that sperm dependency forces gynogenetic individuals into a tight association with sexual species and supposes that this reduces the ability of parasites to evolve increased virulence on gynogens. This evolutionary trajectory might explain the longer than predicted duration of gynogenetic lineages relative to other asexual lineages. An alternative explanation for the stability of gynogenetic lineages is sporadic "leakage" of paternal chromosomes which might provide genetic novelty necessary for defense against parasites and to escape the accumulation of deleterious mutations (Beukeboom and Vrijenhoek 1998; Schlupp 2005; Lamatsch and Stöck 2009). Or, both mechanisms could be operating. That is, presence of sexual individuals allows gynogens to escape or at least delay the population decline caused by co-evolving parasites. Such a delay in population decline could provide an opportunity for leakage to intervene or novel mutations against parasites to arise.

Taking this view of assessing the population dynamics and stability of sperm-dependent asexuals and sexuals in single- and mixed-species assemblages does not seem particularly parsimonious. However, it rests on the premise of parasite adaptation being faster and more deleterious to 'asexual-only' lineages over multiple host generations (which can be tested). Furthermore, it leads to some relevant predictions for the evolution of gynogenetic species and provides insights into the maintenance of asexuality, at least for asexuality that has evolved from tinkering with sexual machinery. More default comparisons of sexual and asexual hosts within single contexts that replicate natural assemblages are unlikely to be particularly informative. Finally, understanding the direction of evolutionary change will be important: that is, whether gynogenetic asexuals evolve from sexuals and can be considered as an endpoint and a radically different strategy than parthenogenesis.

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References

Agrawal, A.F. 2006. Evolution of sex: Why do organisms shuffle their genotypes? Current Biology 16: R696–R704. CrossRef

Altermatt, F. and D. Ebert. 2008. Genetic diversity of *Daphnia magna* populations enhances resistance to parasites. Ecology Letters 11:918–928. CrossRef

Bell, G. 1982. The masterpiece of nature: The evolution and genetics of sexuality. Croom Helm.

Bell, G. 2007. Selection: The Mechanism of Evolution. Oxford University Press, Oxford. CrossRef

Bengtsson, B. 2009. Asex and evolution: A very large-scale overview. Pages 1–19 *in* I. Schön, K. Martens, and P. Dijk, eds. Lost Sex. Springer Netherlands. CrossRef

Beukeboom, L.W. and R.C. Vrijenhoek. 1998. Evolutionary genetics and ecology of sperm-dependent parthenogenesis. Journal Evolutionary Biology 11:755–782. CrossRef

Bi, K. and J. Bogart. 2010. Time and time again: unisexual salamanders (genus *Ambystoma*) are the oldest unisexual vertebrates. BMC Evolutionary Biology 10:1–14. CrossRef

Burt, A. 2000. Perspective: sex, recombination, and the efficacy of selection: was Weismann right? Evolution 54:337–351.

Cavalier-smith, T. 2002. Origins of the machinery of recombination and sex. Heredity 88:125–141. CrossRef

Colegrave, N. 2002. Sex releases the speed limit on evolution. Nature 420:664–666. CrossRef

Daly, M. 1978. The cost of mating. The American Naturalist 112:771–774. CrossRef

Dargent, F., Torres-Dowdall, J., Scott, M.E., Ramnarine, I., and G.F. Fussmann. 2013. Can mixed-species groups reduce individual parasite load? A field test with two closely related poeciliid fishes *Poecilia reticulata* and *Poecilia picta*. PLOS ONE 8:e56789. CrossRef

Decaestecker, E., Gaba, S., Raeymaekers, J.A.M., Stoks, R., Van Kerckhoven, L., Ebert, D., et al. 2007. Host-parasite `Red Queen' dynamics archived in pond sediment. Nature 450:870–873. CrossRef

Fisher, R.A. 1930. The genetical theory of natural selection. Oxford University Press. CrossRef

Hamilton, W. 1982. Pathogens as causes of genetic diversity in their host populations. Pages 269–296 *in* R.M. Anderson, and R.M. May, eds. Population biology of infectious diseases. Springer. CrossRef

Hino, T. 2000. Intraspecific differences in benefits from feeding in mixed-species flocks. Journal of Avian Biology 31:441–446. CrossRef
Howard, R.S. and C.M. Lively. 1994. Parasitism, mutation accumulation and the maintenance of sex. Nature 367:554–557. CrossRef
Hubbs, C. 1964. Interactions between a bisexual fish species and its gynogenetic sexual parasite. Bulletin of the Texas Memorial Museum 8:1–72.
Jaenike, J. 1978. An hypothesis to account for the maintenance of sex within populations. Evolutionary Theory 3:191-194.
Lafferty, K.D. and A.M. Kuris. 2002. Trophic strategies, animal diversity and body size. Trends in Ecology & Evolution 17:507–513. CrossRef
Lamatsch, D. and M. Stöck. 2009. Sperm-dependent parthenogenesis and hybridogenesis in teleost fishes. Pages 399–432 *in* I. Schön, K. Martens, and P. Dijk, eds. Lost Sex. Springer Netherlands. CrossRef
Lehtonen, J., Schmidt, D.J., Heubel, K., and H. Kokko. 2013. Evolutionary and ecological implications of sexual parasitism. Trends in Ecology & Evolution 28:297–306. CrossRef
Lively, C.M. 1999. Migration, virulence, and the geographic mosaic of adaptation by parasites. The American Naturalist 153:S34–S47. CrossRef
Lively, C.M., Craddock, C., and R.C. Vrijenhoek. 1990. Red Queen hypothesis supported by parasitism in sexual and clonal fish. Nature 344:864–866. CrossRef
Maynard-Smith, J. 1978. The evolution of sex. Cambridge University Press.
McKinnon, J.S., Mori, S., Blackman, B.K., David, L., Kingsley, D.M., Jamieson, L., et al. 2004. Evidence for ecology's role in speciation. Nature 429:294–298. CrossRef
Moore, W.S. 1975. Stability of small unisexual-bisexual populations of *Poecilipsis* (Pisces: Poeciliidae). Ecology 56:791–808. CrossRef
Morran, L.T., Schmidt, O.G., Gelarden, I.A., Parrish, R.C., and C.M. Lively. 2011. Running with the Red Queen: Host-parasite coevolution selects for biparental sex. Science 333:216–218. CrossRef
Muller, H.J. 1932. Some genetic aspects of sex. The American Naturalist 66:118–138. CrossRef
Nei, M. 1967. Modification of linkage intensity by natural selection. Genetics 57:625–641.
Schartl, M., Wilde, B., Schlupp, I., and J. Parzefall. 1995. Evolutionary oprigin of a parthenoform, the Amazon molly *Poecilia formosa*, on the basis of a molecular genealogy. Evolution 49:827–835. CrossRef
Schlupp, I. 2005. The evolutionary ecology of gynogenesis. Annual Review of Ecology, Evolution, and Systematics 36:399–417. CrossRef
Schultz, R.J. 1967. Gynogenesis and triploidy in the viviparous fish *Poeciliopsis*. Science 157:1564–1567. CrossRef
Schultz, R.J. and K.D. Kallman. 1968. Triploid hybrids between the all-female Teleost *Poecilia formosa* and *Poecilia sphenops*. Nature 219:280–282. CrossRef
Spolsky, C.M., Phillips, C.A., and T. Uzzell. 1992. Antiquity of clonal salamander lineages revealed by mitochondrial DNA. Nature 356:706–708. CrossRef
Thiollay, J-M. 1999. Frequency of mixed species flocking in tropical forest birds and correlates of predation risk: An intertropical comparison. Journal of Avian Biology 30:282–294. CrossRef
Travis, J., Reznick, D., Bassar, R.D., López-Sepulcre, A., Ferriere, R., and T. Coulson. 2014. Chapter one - Do eco-evo feedbacks help us understand nature? Answers from studies of the trinidadian guppy. Pages 1–40 *in* J. R. Jordi Moya-Laraño, and W. Guy, eds. Advances in Ecological Research. Academic Press.
Van Valen, L. 1973. A new evolutionary law. Evolutionary Theory 1:1–30.
Vrijenhoek, R.C. 1998. Animal clones and diversity. BioScience 48:617–628. CrossRef