A reevaluation of the Red Queen model for the maintenance of sex in a clonal–sexual fish complex (Poeciliidae: *Poeciliopsis*)

Stephen C. Weeks

Abstract: The validity of the assumptions of the Red Queen model for the maintenance of sexual reproduction was reassessed using life-history data collected from clonal and sexual fish in the genus *Poeciliopsis*. A previous study using these strains (one sexual, two clonal) indicated that sex might be maintained by coevolutionary processes outlined in the Red Queen model. However, the previous study did not test the assumption that parasitism negatively affects the host's fitness. The current study was undertaken to address this issue, as well as to reevaluate the assumptions of the Red Queen model in this complex. Data on size, fecundity, and parasitic intensity were collected from fish from seven Mexican pools. Parasitic intensity was highest for one clone, but intensity was not correlated with clonal frequency, as assumed by the Red Queen model. Variance in parasitic intensity was not reduced in the clonal strains relative to the sexual strain, and intensity was not correlated with fecundity. These data, combined with aspects of the parasite's biology, indicate that the Red Queen model is unlikely to maintain sex in this clonal–sexual complex.

Résumé : La validité des suppositions relatives au modèle Red Queen pour l'entretien de la reproduction sexuelle a été réévaluée à l'aide des données historiques recueillies à partir de poissons d'origines clonale et sexuelle du genre *Poeciliopsis*. Une étude antérieure, fondée sur ces souches (une sexuelle, deux clonales), a montré que la reproduction sexuelle pourrait être entretenue par les processus de coévolution décrits dans le modèle Red Queen. Cependant, l'étude précédente n'a pas vérifié l'hypothèse voulant que le parasitisme agissait négativement sur la bonne forme de l'hôte. L'étude actuelle a été entreprise pour examiner cette question et pour réévaluer les suppositions du modèle Red Queen relatives à ce complexe. Les données sur la taille, la fécondité et l'intensité parasitaire ont été recueillies sur les poissons provenant de sept étangs mexicains. L'intensité parasitaire était la plus élevée pour l'un des clones, mais il n'y avait pas de corrélation entre l'intensité et la fréquence clonale, comme le suppose le modèle Red Queen. La variance dans l'intensité parasitaire ne se trouvait pas réduite dans les souches clonales, comparativement aux souches sexuelles, et il n'y avait pas corrélation entre l'intensité et la fécondité. Ces données, combinées à certains aspects de la biologie des parasites, montrent qu'il est peu probable que le modèle Red Queen entretienne la reproduction sexuelle dans ce complexe clonal–sexuel.
[Traduit par la Rédaction]

Introduction

In the early 1970s, Maynard Smith (1971*a*, 1971*b*) and Williams (1975) concluded that sexual reproduction has an immediate twofold numerical cost relative to asexual reproduction. This twofold cost of sex was difficult to accept considering the prevalence of sexual reproduction among higher plants and animals (Levins 1942; Bell 1982). Since then, numerous models have been formulated proposing short-term advantages of sex that might offset this cost. Hypothesized benefits of sexual reproduction range from strictly genetic (Bernstein et al. 1981, 1984, 1985; Kondrashov 1982, 1984; Kirkpatrick and Jenkins 1989) to strictly ecological (Williams and Mitton 1973; Ghiselin 1974; Hamilton 1975, 1980, 1990; Williams 1975; Vrijenhoek 1979, 1984; Anderson and May 1982; Bell 1982, 1985; Case and Taper 1986; Weeks 1993).

One of the ecological models, the Red Queen (RQ) model, ascribes a benefit to producing variable progeny that can potentially offset this twofold cost of sex in a biotically variable environment (Hamilton 1975, 1980, 1990; Levin 1975; Glesener and Tilman 1978; Jaenike 1978; Bremmermann 1980, 1985; Anderson and May 1982; Bell 1982). Sexual reproduction is predicted to allow offspring to adapt to a changing biotic environment, in which antagonists (e.g., predators and parasites) target the most common phenotypes of a population, causing frequency-dependent selection for uncommon phenotypes (Hamilton 1980; Hutson and Law 1981). This coevolution of predator and prey or parasite and host causes the offspring's environment to be predictably different from that of the parent's, which selects for genetic rearrangement of the parental genes across generations (Bell 1982). Thus, both the target population and its associated antagonists are caught in a continual cycle of genetic rearrangement, with the target species shuffling its genes to assure moving targets for predators and parasites, and the antagonists also reshuffling to genetically track their prey or host populations (Levin 1975; Jaenike 1978; Hamilton 1980, 1990; Hutson and Law 1981; Bell 1982).

Clonal and sexual fish in the genus *Poeciliopsis* have been used successfully to address many of the ecological models for the maintenance of sex. Several studies of a related ecological model, the frozen niche variation (FNV) model (Vrijenhoek 1979, 1984; Weeks 1993), have indicated that clonal and sexual strains coexist because of phenomena associated with re-

Received July 7, 1995. Accepted November 20, 1995.
J12986

S.C. Weeks. Department of Biology, University of Akron, Akron, OH 44325–3908, U.S.A. e-mail: scweeks@uakron.edu

Table 1. Estimated fish populations collected from nine pools.

Pool	Estimated total number	Density (no./L)	Number collected				Position[a]
			P. monacha	MML/I	MML/II	ML/VII	
1	203	0.15	23	18	0	0	1 (near log pool)
2	390	1.14	40	8	2	0	1 (near log pool)
3	50	0.16	16	12	8	0	2 (near sandal pool)
4	210	0.39	40	10	0	0	2 (near sandal pool)
5	269	0.35	32	10	8	0	2 (near sandal pool)
6	200	0.10	47	2	1	0	3 (below sandal pool)
7	69	0.13	25	9	18	0	4 (200 m above bottom)
8	136	0.08	41	4	0	5	5 (bottom of Platanos)
9	75	0.07	42	8	2	0	5 (bottom of Platanos)

[a]Positions are outlined in Lively et al.

source partitioning. The FNV model assumes that the production of offspring capable of using different resources is adaptive in a heterogeneous environment (Vrijenhoek 1979, 1984; see also Bell 1982). Sexual populations are assumed to have a broader niche than a monoclonal population, which should reduce among-individual competition and increase overall productivity (Ghiselin 1974; Vrijenhoek 1979, 1984; Bell 1982, 1985; Case and Taper 1986; Weeks 1993). Clones should outcompete sexual individuals for the narrow range of resources to which the clones are best adapted, because of their twofold reproductive advantage, but sexual individuals are predicted to persist because their wider niche allows them use of resources unavailable to individual clones (Vrijenhoek 1984; Weeks 1993). Vrijenhoek (1979) has shown that sexual individuals constitute a smaller proportion of *Poeciliopsis* populations in streams where clonal diversity is high than they do in monoclonal streams. This sexual replacement is consistent with the notion that the clones are usurping a higher proportion of the niche space in populations where their combined resource breadth is largest (Vrijenhoek 1979). Schenck and Vrijenhoek (1986, 1989) found both spatial and dietary differences among sexual and clonal populations of two unisexual biotypes of *Poeciliopsis* that might facilitate coexistence in their native streams. Similarly, Weeks et al. (1992) described differences in feeding behavior between two naturally cooccurring hemiclones and their sexual progenitors, which corresponded with natural differences in the diets of field-caught fish, which may also help explain clonal and sexual coexistence. These studies provide strong evidence for genetically determined, ecologically relevant, phenotypic differences among clonal lineages and between clones and sexual individuals and have therefore verified many of the assumptions and predictions of the FNV model.

Recently, the *Poeciliopsis* system was also used to confirm assumptions of the RQ model. Lively et al. (1990) showed that clones in a three-pool system had higher parasitic intensity than similarly sized, outcrossed sexual individuals. Also, Lively et al. (1990) found that the most common clone was the most heavily parasitized, although the most common type differed among pools. These results suggest a tight tracking of the most common genotype by the parasite over a short geographic range, which would be a strong verification of the assumptions of the RQ model.

The results of the Lively et al. (1990) study are intriguing because they suggest that the maintenance of sexual reproduction in the *Poeciliopsis* system may be due, at least in part, to processes associated with the RQ model. However, that study did not establish a fitness effect of the parasites on the fish, which is necessary for operation of the RQ. In the current experiment, I sought to examine the relationship of parasitic intensity to fitness in these fish by collecting life-history data (size and fecundity) from field-collected fish. I was also able to extend the work of Lively et al. (1990) by using seven separate pools (as compared with three) to test the RQ model's assumption of higher parasitic intensity in clones relative to sexual populations. By combining the fecundity and parasitic intensity data, I was able to test for the expected negative effects of parasitism on one aspect of fitness (fecundity), which is required for the maintenance of sex under a coevolutionary scenario, as outlined by the RQ model.

Materials and methods

Fish strains and collection locations

Three *Poeciliopsis* strains were analyzed in this experiment: an outcrossed sexual strain (*Poeciliopsis monacha*) and two associated unisexual, triploid strains (*Poeciliopsis 2-monacha-lucida*: MML/I and MML/II; Vrijenhoek et al. 1977). The triploid *Poeciliopsis* strains reproduce gynogenetically, a strictly clonal mode of reproduction whereby the entire triploid genome is faithfully replicated among generations (Schultz 1967). Although sperm from a sexual species is required to activate embryogenesis in the triploid ova, paternal genes make no contribution to the genotype of the offspring (Schultz 1967; Cimino 1972; Vrijenhoek 1972). For further information on this reproductive complex, see Schultz (1969).

Five individuals of the hybridogenetic clone *P. monacha-lucida* (ML/VII; Vrijenhoek et al. 1978) were collected from one of the nine pools (Table 1). However, data from a single pool did not allow a statistical comparison; therefore, data from these five fish were not included in the figures or tables that follow.

Fish were collected from pools in the Arroyo de los Platanos region of the Rio Fuerte, Sonora, Mexico (26°30′N, 108°30′W; see Moore and Eisenbrey 1979 for further details of the region), during May 1990. During this time of year, the stream dries down to small, isolated pools that are interconnected by trickles of water. This isolation results in a number of pools that are essentially closed systems, with only a slight amount of migration among pools (Moore and Eisenbrey 1979). Nine of the most isolated of these pools were chosen to maximize the likelihood that the measured biotic and abiotic factors of each pool well reflected the major influences on recent growth and development of the collected fish.

The measures taken from each pool included relative position along the stream, dimensions of the pool (width, length, and depth), and total number of fish per pool. Most pools were fairly rectangular,

Table 2. Analysis of covariance for infections per strain.

Source	df	Mean square	F	P
Pool 1				
Size	1	24.221	39.70	0.0001
Strain	1	3.257	5.34	0.0254
Size × strain	1	5.113	8.38	0.0058
Pool 2				
Size	1	0.744	0.90	0.3487
Strain	1	6.335	7.64	0.0082
Pool 3				
Size	1	25.660	42.75	0.0001
Strain	2	1.137	1.89	0.1669
Pool 4				
Size	1	48.160	56.21	0.0001
Strain	1	3.202	3.74	0.0594
Pool 5				
Size	1	46.797	49.14	0.0001
Strain	2	3.081	3.24	0.0484
Pool 7				
Size	1	3.290	6.54	0.0138
Strain	2	0.131	0.26	0.7716
Pool 9				
Size	1	0.490	0.35	0.5568
Strain	1	6.025	4.31	0.0434

Note: Pool 1 had a significant size × strain interaction, and thus this term was included in the model. All other pools had no significant size × strain interaction and thus had size × strain dropped from the model.

with sloping bottoms. Therefore, pool volume was estimated using the dimensions above and assuming the pools were longitudinally divided half cylinders. Total number per pool was estimated using a catch per unit effort model (Ricker 1958). Pools were repeatedly sampled, using a hand-held seine, until either no fish were caught, or the number was reduced to the point at which only a few fish were caught in each haul (usually five to seven seine hauls). Fish density was calculated by dividing the estimated population size by pool volume.

A sample of 50 females (when available) was taken from each pool. Tissue samples from each female were used for starch-gel electrophoresis to determine sexual type and clonal identity. Life-history characters measured were standard length and number of eggs and embryos per female. Also, the number of parasitic cysts was determined for each female (see Lively et al. 1990). The parasite is a trematode larva (*Uvulifer* sp.) that causes spherical, black cysts in the epidermis of these fish, which are easily counted under a dissecting microscope at low magnification (Lively et al. 1990).

Statistical analysis

All statistical analyses were performed using the SAS statistical package (SAS Institute Inc. 1985). In any particular pool, a clonal strain was not included in these analyses if there were less than 5 individuals in the collection of 50 fish (Table 1). Parasitic infection was analyzed using analyses of covariance, as outlined in Lively et al. (1990). The numbers of parasitic cysts per fish were compared among strains in each pool. Only one of seven comparisons indicated that the slopes of the lines relating number of cysts to body size were significantly different among strains. In this one case, analysis was discontinued at this point (see Lively et al. 1990). In the other six cases, in which the slopes were not significantly different, an analysis of covariance was performed comparing the number of cysts per unit body size among the strains within each pool.

Variance ratios were employed to test the prediction that sexual populations have a greater among-individual difference in parasitic load than clones. Variation in parasitic intensity per strain was estimated by computing the variance of the residuals of cyst number regressed on standard length (see Lively et al. 1990), both within each pool as well as combined across all pools. For the across-pool comparison, the residuals were computed from a model including standard length, pool, and the standard length by pool interaction to account for possible differences in parasitic intensity among pools. Comparisons between strains were made using the ratios of the sexual to the clonal variance and comparing these ratios with standard F tables using $n_s - 2$ degrees of freedom for the numerator and $n_c - 2$ degrees of freedom for the denominator, where n_s is the sample size of the sexual populations and n_c is the sample size of the clones (Sokal and Rohlf 1981).

To determine whether increased parasitic intensity was associated with reduced fecundity, size-corrected fecundity was regressed on number of cysts per individual. Size-corrected fecundity was calculated by using the residuals of fecundity regressed on standard length and adding the average fecundity to each residual score. These comparisons were made within each strain, combining individuals across all nine pools.

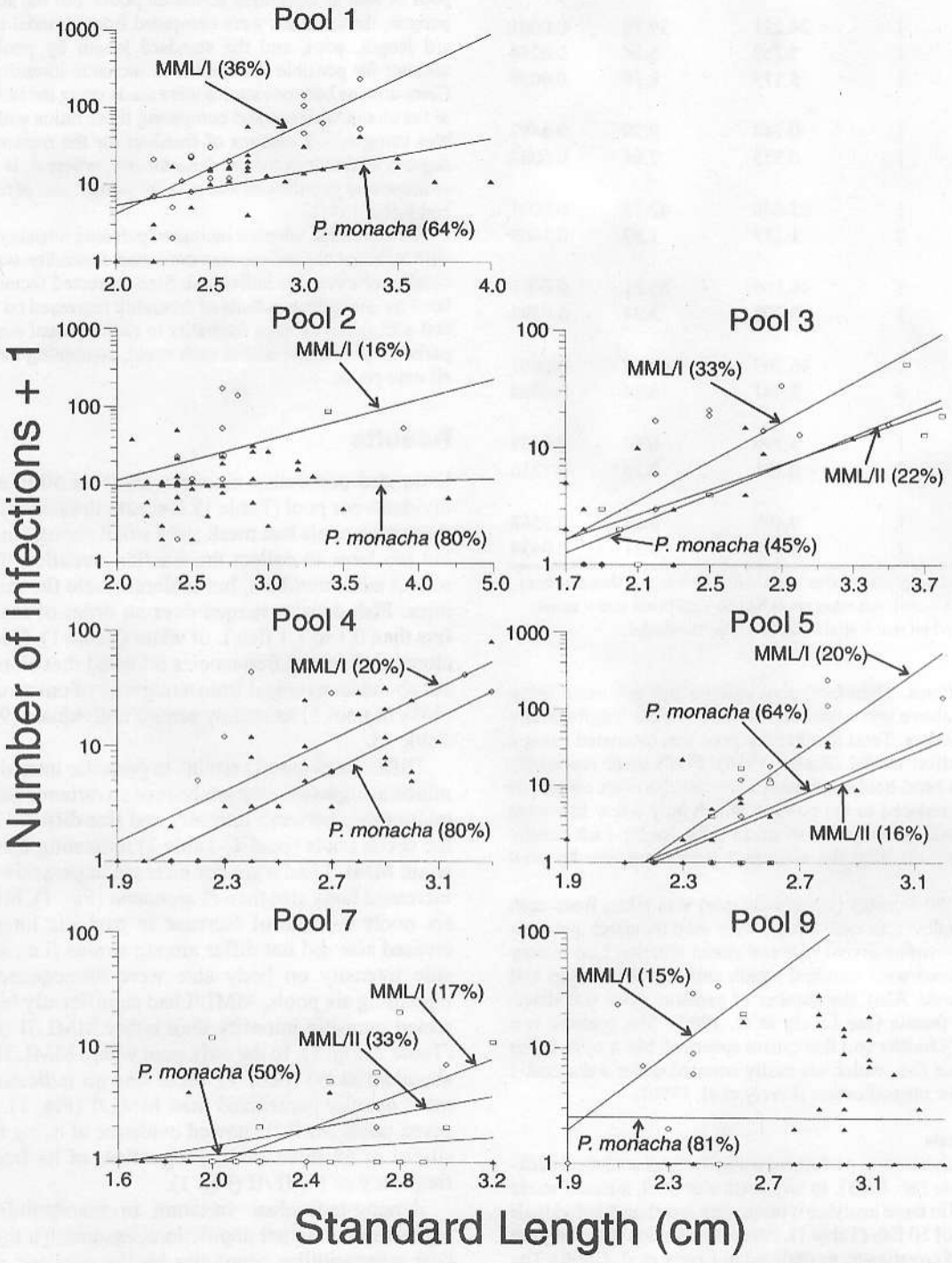
Results

Estimated population sizes ranged from 50 to almost 400 individuals per pool (Table 1). Because the seines used to sample these nine pools had mesh sizes small enough to sample males but too large to collect the smaller juveniles, these estimates reflect adult numbers, but underestimate the number of juveniles. Fish density ranged over an order of magnitude, from less than 0.1 to 1.1 fish/L of water (Table 1). Quantification of clonal and sexual frequencies revealed that clonal versus sexual abundance ranged from a majority of unisexual individuals (56% in pool 3) to mainly sexual individuals (94% in pool 6; Table 1).

Differences among strains in parasitic intensity were determined using a two-step analysis of covariance (see above). The relationship between intensity and size differed in only one of the seven pools (pool 1; Table 2) indicating that, in this pool, strain MML/I had a greater increase in parasitic intensity with increased body size than *P. monacha* (Fig. 1). In the remaining six pools the rate of increase in parasitic intensity with increased size did not differ among strains (i.e., slopes of parasitic intensity on body size were homogeneous). In these remaining six pools, MML/I had significantly higher size-corrected parasitic intensity than either MML/II or *P. monacha* (Table 2, Fig. 1). In the only pool where MML/II was the most abundant clone (pool 7), there was no indication that it was more heavily parasitized than MML/I (Fig. 1). In fact, in all seven pools MML/I showed evidence of being the most parasitized of all three strains, regardless of its frequency or the frequency of MML/II (Fig. 1).

Among-individual variation in susceptibility should be highest in the sexual populations, assuming a tight tracking of host susceptibility genotypes by the parasitic population. In only 3 of 10 comparisons was the variance in parasitic infection greater in the sexual than the clonal population, and none of these differences were significant (Table 3). Since within-pool sample sizes were only moderate, no single within-pool comparison of variance had high statistical power. However, sample sizes were large enough to detect a trend of a higher variance in infection rate for sexuals if one existed. Also, when results were combined across pools, increasing the statistical power to detect differences, there was no evidence of in-

Fig. 1. Plots of parasitic intensity versus standard length for seven of the nine pools with coexisting clones and sexual individuals. Regression lines and strain frequencies (in parentheses) are shown for strains having more than five individuals per pool. Note that the axes differ among pools. Symbols are as follows: open diamonds, MML/I; open squares, MML/II; solid triangles, *P. monacha*.



creased variability of parasitic intensity for the sexual individuals relative to the clones.

Size-corrected fecundity was regressed on number of parasitic cysts per individual within strains and across pools to examine whether level of parasitism was associated with reduced fecundity (Table 4). All three strains had a negative association of size-corrected fecundity with parasitic intensity, but in no case was this association significant (Table 4). In fact, in all three strains the number of cysts per individual only accounted for between 0.4 and 3% of the variation in size-corrected fecundity (Table 4, Fig. 2).

Discussion

The RQ model assumes that biological antagonists (e.g.,

Table 3. Comparisons of variance in parasitic intensity among sexual and clonal strains.

Comparison		Numerator	Denominator	F
Pool 1	Pm/C1	0.6465 (30)	0.4719 (16)	1.370
Pool 2	Pm/C1	0.5859 (38)	1.8315 (6)	0.320
Pool 3	Pm/C1	0.5301 (14)	0.7380 (10)	0.718
	Pm/C2	0.5301 (14)	0.3360 (6)	1.578
Pool 4	Pm/C1	0.7489 (36)	1.2868 (8)	0.582
Pool 5	Pm/C1	0.7935 (30)	0.9073 (8)	0.875
	Pm/C2	0.7935 (30)	1.1720 (6)	0.677
Pool 7	Pm/C1	0.1373 (23)	0.2067 (7)	0.664
	Pm/C2	0.1373 (23)	1.0066 (16)	0.136
Pool 9	Pm/C1	1.3347 (40)	1.0808 (6)	1.235
Overall	Pm/C1	0.720 (211)	0.783 (61)	0.920
	Pm/C2	0.720 (211)	0.838 (28)	0.859

Note: The sexual strain (numerator) is always compared with one or both of the clonal strains (denominator) as shown in the Comparison column. Strain designations are as follows: Pm, *P. monacha*; C1, MML/I; and C2, MML/II. Degrees of freedom for the F tests are shown in parentheses.

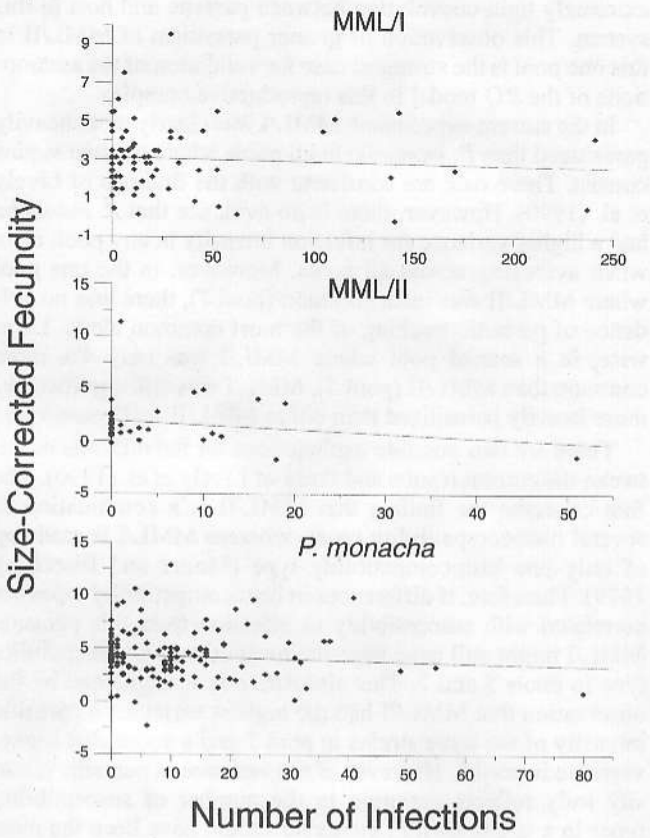
Table 4. Regression of size-corrected fecundity on level of parasitism, combining individuals across pools.

Strain	N	Slope	R^2	F	P
MML/I	75	−0.002	0.0036	0.266	0.6077
MML/II	34	−0.041	0.0314	1.038	0.3158
P. monacha	310	−0.021	0.0075	2.332	0.1278

predators and parasites) selectively target the most common genotype in a population, thereby disproportionately reducing that genotype's fitness relative to rarer genotypes (Hamilton 1980, 1990; Hutson and Law 1981). Such an interaction would select for the ability of prey and hosts to rearrange their genotypes to produce some number of rare genotypes that enjoy higher fitness (Hamilton 1980, 1990; Anderson and May 1982; Bell 1982). This rearrangement also selects for concomitant genotypic rearrangement in the predators and parasites, setting up the conditions for coevolution (Hamilton 1980, 1990). Three testable assumptions are important to the RQ model. First, the assumption that parasites track the most common genotype can be easily tested in clonal–sexual systems. Assuming the sexual population is sufficiently outcrossed and thus has a large number of susceptibility types, the most common clone should provide the most common susceptibility type in a population and therefore should be the most heavily parasitized. Second, a correlated assumption is that the sexual population should include a number of susceptibility types, whereas clones should be either largely susceptible or resistant. Therefore, among-individual variation in parasitism should always be highest in outcrossed sexual populations. The third assumption is that the heavily parasitized individuals must have reduced fitness relative to less parasitized counterparts. In multiclonal populations, these three processes should combine to produce fluctuating clonal dominance as rarer clones enjoy a fitness advantage, increase in frequency, and then are attacked as the new most common genotype (Lively 1993). The singling out of these individuals decreases their prevalence because they have reduced fitness concomitant with the increased parasitism.

The first two of these three assumptions were addressed by

Fig. 2. Plots of size-corrected fecundity versus number of parasitic infections, combining fish across pools. Note that the axes differ among strains.



Lively et al. (1990) using fish from the same reproductive complex and from the same stream as in the current study. In their study, three pools were sampled, two being sampled in 2 different years. Two of the three pools were monoclonal, having only MML/I and *P. monacha*. In these pools, MML/I was more heavily parasitized per unit body length than *P. monacha*, and the variance in infection intensity was highest among *P. monacha* individuals, when the *P. monacha* populations were outcrossed. Both of these observations are consistent with the RQ model. In 1 of the 2 years, the *P. monacha* population was highly inbred. During this year, *P. monacha* was the most heavily parasitized of the two strains and had a lower variance in parasitic intensity than MML/I (Lively et al. 1990). Because the sexual individuals constituted only 43% of the population during this year and thus could not have been the most common susceptibility type even if the sexuals were monomorphic at all susceptibility loci, these latter results could only be consistent with the RQ model if one assumes that the sexual individuals' overall resistance to infection was reduced by inbreeding.

The strongest evidence that the parasite tracks the most common genotype in this reproductive complex was revealed in the third pool measured by Lively et al. (1990). In this pool, MML/II was the most common clone, and it had significantly more parasites per unit length than either MML/I or *P. monacha*. There was no difference in the variance of parasitism intensity between any of the three strains. The observed rever-

sal of the pattern of parasitism such that the most common clone was always the most heavily parasitized across a distance of no more than several hundred metres suggests an extremely tight coevolution between parasite and host in this system. This observation of greater parasitism of MML/II in this one pool is the strongest case for validation of the assumptions of the RQ model in this reproductive complex.

In the current experiment, MML/I was clearly more heavily parasitized than *P. monacha* in all pools where the two strains coexist. These data are consistent with the findings of Lively et al. (1990). However, there is no evidence that *P. monacha* had a higher variance for infection intensity in any pool, even when averaging across all pools. Moreover, in the one pool where MML/II was most common (pool 7), there was no evidence of parasitic tracking of the most common clone. Likewise, in a second pool where MML/I was only 4% more common than MML/II (pool 5), MML/I was still significantly more heavily parasitized than either MML/II or *P. monacha*.

There are two possible explanations for the differences between the current results and those of Lively et al. (1990). The first concerns the finding that MML/II is a combination of several histocompatibility types, whereas MML/I is made up of only one histocompatibility type (Moore and Eisenbrey 1979). Therefore, if differences in histocompatibility types are correlated with susceptibility to infection from this parasite, MML/I might still have been the most common susceptibility type in pools 5 and 7. This alternative is strengthened by the observation that MML/II had the highest variance in parasitic intensity of the three strains in pool 7 and a somewhat higher variance in pool 5. However, if the variance in parasitic intensity truly reflects variation in the number of susceptibility types in a strain, then *P. monacha* should have been the most variable of all three strains. Since this was not the case, it is unlikely that the variation in parasitic intensity is an indicator of the number of susceptible types within MML/II. Also, if a high number of susceptibility types within MML/II explains the lack of evidence for parasitic tracking of common genotypes in this study, then one must assume that the MML/II in Lively et al.'s (1990) sample was primarily composed of one of these susceptibility types, which would account for the observed reversal of parasitism recorded in their study. Unfortunately, without genetic markers to distinguish these susceptibility types, any experiments designed to quantify the number of these types within MML/II would require that large numbers of these fish be transported live into a laboratory setting for tissue grafting experiments (see Moore and Eisenbrey 1979).

A second possible explanation is that the reversal of parasitism reported by Lively et al. (1990) was not caused by the coevolutionary processes outlined in the RQ model but was probably due to one or more unrelated processes. A major problem of applying the RQ model to the *Poeciliopsis* system concerns the ability of the parasite to track their hosts over the distances reported by Lively et al. (1990). The parasites probably do not track their hosts so tightly as to follow individuals in a single pool but more likely track an average population over space and time. *Uvulifer* spp. are strigeoid trematodes with complex life cycles (Schmidt and Roberts 1985). The adult stage of this trematode infects fish-eating birds (e.g., kingfishers). Eggs produced by these adults hatch into miracidia that infect snails in the genus *Helisoma*. Cercariae develop in the snail hosts and are released into the water column where they then infect passing fish by encysting in their dermis. The life cycle is completed when the fish are eaten by a foraging bird. This life cycle creates two potential problems for operation of the RQ model over the distances separating the pools sampled in this stream. First, because the relative clonal composition of each isolated pool is unlikely to be maintained during the rainy season, the parasites essentially have a single dry season in which to adapt to the most common clone within any one pool. Because completion of the life cycle requires several steps, it is unlikely that enough generations are possible for the parasites to adapt to the most common genotypes in these local pools. Second, the tight host tracking outlined by Lively et al. (1990) requires genetic isolation of the parasite over distances of no more than several hundred metres. Because the activities of the parasites' primary host, piscivorous birds, undoubtedly spread the parasites over much greater distances than this, such local genetic isolation in the parasite populations is unlikely. Both of these qualifications indicate that the operation of the processes outlined in the RQ model in this reproductive complex on such a localized scale is doubtful.

One of two alternative hypotheses is more likely to explain the observation that MML/I is the most heavily parasitized fish strain in this complex. It is probable that selective pressures on the parasites are averaged across a series of evolutionarily interconnected pools. It is possible that the parasite is selectively infecting MML/I because it is the most common genotype across this series of pools, which would be consistent with the RQ model. In this scenario, the parasite would be tracking the most common genotype across a larger geographical range, which would explain the higher frequency of parasitism for MML/I across all of the Platanos. However, this alternative hypothesis remains to be confirmed.

A second explanation is that MML/I is inherently more susceptible to parasitism than the other two strains, regardless of its frequency. Such a situation has been described in a related clonal–sexual complex. Leberg and Vrijenhoek (1994) found that one of two hybridogenetic *P. monacha-lucida* clones was susceptible to infection by a different trematode parasite (*Gyrodactylus turnbulli*), while another coexisting clone was completely resistant. This trematode parasite does not cooccur with the fish in this hybridogenetic system, and thus there is no opportunity for coevolution among host and parasite in this particular example. Nevertheless, this example is interesting in that it underscores the possibility that different susceptibility genotypes can be frozen from a sexual population (Leberg and Vrijenhoek 1994). Such a difference in clonal susceptibility would cause a permanent parasitic bias for one clone over the other rather than the cyclic parasitic intensity outlined by the RQ model.

Distinguishing between these two possibilities requires either using a more intensive sampling regime that includes streams that can be considered as containing truly distinct parasite populations or, more conclusively, devising an artificial selective regime to demonstrate the tracking of hosts by these parasites under controlled conditions. Clearly, there is no conclusive evidence for tight parasitic tracking by these trematodes on the most common genotype in this reproductive complex.

Even if tracking of the most common genotype by the tre-

matodes in this system is verified, there is no evidence that this parasite has a negative effect on fitness. In MML/I, the most heavily parasitized clone, there were two cases in which females had more than 225 parasitic cysts. These fish were almost completely black owing to the number of cysts in the epidermis. If parasitism negatively affects fitness (as assumed by the RQ model), then these fish should have either reduced fecundity or increased mortality. Nevertheless, one of these two had a size-corrected fecundity that was greater than average, while the other was smaller than average. In general, the relationship of size-corrected fecundity to parasite load was very weak. Thus, there is no indication that parasitism can create the reduced fecundity in the most common genotype that would be necessary to cause the type of coevolution envisioned in the RQ model. There is still a possibility that parasitism increases mortality, such as by making heavily parasitized fish more obvious to predators (Krause and Godin 1994) or by decreasing overwintering survival (Lemly and Esch 1984). These possibilities remain to be tested.

Nevertheless, clonal composition has remained reasonably stable for at least 90 generations in this reproductive complex (Schenck and Vrijenhoek 1989), with little evidence of the cycling in clonal dominance one would expect from the RQ model (Lively 1993). Although extensive sampling on a monthly basis over an extended period has not been undertaken, dozens of collections have been made over two decades at these sites (Vrijenhoek 1994). Overall, MML/I has remained the dominant clone in this stream over this time period, with MML/II always being in low abundance. This further suggests that the coevolutionary relationships between host and parasite predicted by the RQ model are either not present in this system or are so weak as to be ineffective at modifying clonal performance.

The genetic and ecological evidence so far collected from fish in this reproductive complex (see Vrijenhoek 1994) suggests that the long-term clonal and sexual coexistence of these lineages (Quattro et al. 1991) is best explained by resource or niche partitioning among strains, as predicted by the FNV model. At this point, the evidence for coevolutionary relationships between parasites and hosts remains equivocal, and the likelihood that the RQ model explains the coexistence of sexuals and clones in this system is doubtful.

Acknowledgments

I thank A. Varela and R. Schenck for help with the field collections; C. Craddock, F. Hensley, C. Lively, G. Meffe, and P. Niewiarowski for critically reviewing the manuscript; P. Dixon and K. Garrett for statistical advice; and R.C. Vrijenhoek for constructive criticism of the design of the study. This study was funded in part by a National Science Foundation doctoral dissertation support grant (BSR–8815423).

References

Anderson, R.M., and May, R.M. 1982. Coevolution of hosts and parasites. Parasitology, **85**: 411–426.

Bell, G. 1982. The masterpiece of nature, the evolution and genetics of sexuality. University of California Press, Berkeley, Calif.

Bell, G. 1985. Two theories of sex and variation. Experientia, **41**: 1235–1245.

Bernstein, H., Byers, G.S., and Michod, R.E. 1981. Evolution of sexual reproduction. Importance of DNA repair, complementation and variation. Am. Nat. **117**: 537–549.

Bernstein, H.H., Byerly, C.F., Hopf, A., and Michod, R.E. 1984. Origin of sex. J. Theor. Biol. **110**: 323–351.

Bernstein, H.H., Byerly, C.F., Hopf, A., and Michod, R.E. 1985. Genetic damage, mutation and the evolution of sex. Science (Washington, D.C.), **229**: 1277–1281.

Bremermann, H.J. 1980. Sex and polymorphism as strategies in host–pathogen interactions. J. Theor. Biol. **87**: 671–702.

Bremermann, H.J. 1985. The adaptive significance of sexuality. Experientia, **41**: 1245–1254.

Case, T.J., and Taper, M.L. 1986. On the coexistence and coevolution of asexual and sexual competitors. Evolution, **40**: 366–387.

Cimino, M.C. 1972. Meiosis in a triploid all-female fish (*Poeciliopsis*, Poeciliidae). Science (Washington, D.C.), **175**: 1484–1486.

Ghiselin, M.T. 1974. The economy of nature and the evolution of sex. University of California Press, Berkeley, Calif.

Glesener, R.R., and Tilman, D. 1978. Sexuality and the components of environmental uncertainty. Clues from geographic parthenogenesis in terrestrial animals. Am. Nat. **112**: 659–673.

Hamilton, W.D. 1975. Gamblers since life began. Barnacles, aphids, elms. Q. Rev. Biol. **50**: 175–180.

Hamilton, W.D. 1980. Sex versus non-sex versus parasite. Oikos, **35**: 282–290.

Hamilton, W.D. 1990. Memes of Haldane and Jayakar in a theory of sex. J. Genet. **69**: 17–32.

Hutson, V., and Law, R. 1981. Evolution of recombination in populations experiencing frequency-dependent selection with time delay. Proc. R. Soc. Lond. B Biol. Sci. **213**: 345–359.

Jaenike, J. 1978. An hypothesis to account for the maintenance of sex within populations. Evol. Theory, **3**: 191–194.

Kirkpatrick, M., and Jenkins, C.D. 1989. Genetic segregation and the maintenance of sexual reproduction. Nature (London), **339**: 300–301.

Kondrashov, A.S. 1982. Selection against harmful mutations in large sexual and asexual populations. Genet. Res. **40**: 325–332.

Kondrashov, A.S. 1984. Deleterious mutations as an evolutionary factor. 1. The advantage of recombination. Genet. Res. **44**: 199–217.

Krause, J., and Godin, J.J. 1994. Influence of parasitism on the shoaling behavior of banded killifish, *Fundulus diaphanus*. Can. J. Zool. **72**: 1775–1779.

Leberg, P.L., and Vrijenhoek, R.C. 1994. Variation among desert topminnows in their susceptibility to attack by exotic parasites. Conserv. Biol. **8**: 419–424.

Lemly, A.D., and Esch, G.W. 1984. Effects of the trematode *Uvulifer ambloplitis* on juvenile bluegill sunfish, *Lepomis macrochirus*: ecological implications. J. Parasitol. **70**: 475–492.

Levin, D.A. 1975. Pest pressure and recombination systems in plants. Am. Nat. **109**: 437–451.

Levins, D. 1942. The evolution of sex in flowering plants. Biol. Rev. Cambridge Philos. Soc. **17**: 46–67.

Lively, C.M. 1993. Rapid evolution by biological enemies. Trends Ecol. Evol. **8**: 345–346.

Lively, C.M., Craddock, C., and Vrijenhoek, R.C. 1990. Red Queen hypothesis supported by parasitism in sexual and clonal fish. Nature (London), **344**: 864–866.

Maynard Smith, J. 1971*a*. The origin and maintenance of sex. *In* Group selection. *Edited by* G.C. Williams. Aldine-Atherton, Chicago, Ill. pp. 163–175.

Maynard Smith, J. 1971*b*. What use is sex? J. Theor. Biol. **30**: 319–335.

Moore, W.S., and Eisenbrey, B. 1979. The population structure of an asexual vertebrate, *Poeciliopsis 2 monacha-lucida* (Pisces: Poeciliidae). Evolution, **33**: 563–578.

Quattro, J.M., Avise, J.C., and Vrijenhoek, R.C. 1991. Molecular

evidence for multiple origins of hybridogenetic fish clones (Poeciliidae: *Poeciliopsis*). Genetics, **127**: 391–398.

Ricker, W.E. 1958. Handbook of computations for biological statistics of freshwater fishes. Bull. Fish. Res. Board Can. No. 119.

SAS Institute Inc. 1985. SAS user's guide, statistics, version 5 edition. SAS Institute Inc., Cary, N.C.

Schenck, R.A., and Vrijenhoek, R.C. 1986. Spatial and temporal factors affecting coexistence among sexual and clonal forms of *Poeciliopsis*. Evolution, **40**: 1060–1070.

Schenck, R.A., and Vrijenhoek, R.C. 1989. Coexistence among sexual and asexual *Poeciliopsis*, foraging behavior and microhabitats. *In* Evolution and ecology of unisexual vertebrates. *Edited by* R.M. Dawley and J.P. Bogart. Bulletin No. 466. New York State Museum, New York. pp. 39–48.

Schmidt, G.D., and Roberts, L.S. 1985. Foundations of parasitology. Times Mirror/Mosby College Publishing, St. Louis, Mo.

Schultz, R.J. 1967. Hybridization experiments with an all-female fish of the genus *Poeciliopsis*. Biol. Bull. (Woods Hole), **130**: 415–429.

Schultz, R.J. 1969. Hybridization, unisexuality, and polyploidy in the teleost *Poeciliopsis* (Poeciliidae) and other vertebrates. Am. Nat. **103**: 605–619.

Sokal, R.R., and Rohlf, F.J. 1981. Biometry. W.H. Freeman & Co., New York.

Vrijenhoek, R.C. 1972. Genetic relationships of unisexual-hybrid fishes to their sexual progenitors using lactate dehydrogenase isozymes as gene markers (*Poeciliopsis*, Poeciliidae). Am. Nat. **106**: 754–766.

Vrijenhoek, R.C. 1979. Factors affecting clonal diversity and coexistence. Am. Zool. **19**: 787–797.

Vrijenhoek, R.C. 1984. Ecological differentiation among clones. The frozen niche variation model. *In* Population biology and evolution. *Edited by* K. Wohrmann and V. Loeschcke. Springer-Verlag, Berlin. pp. 217–231.

Vrijenhoek, R.C. 1994. Unisexual fish: model systems for studying ecology and evolution. Annu. Rev. Ecol. Syst. **25**: 71–96.

Vrijenhoek, R.C., Angus, R.A., and Schultz, R.J. 1977. Variation and heterozygosity in sexually vs. clonally reproducing populations of *Poeciliopsis*. Evolution, **31**: 767–781.

Vrijenhoek, R.C., Angus, R.A., and Schultz, R.J. 1978. Variation and clonal structure in a unisexual fish. Am. Nat. **112**: 41–55.

Weeks, S.C. 1993. The effects of recurrent clonal formation on clonal invasion patterns and sexual persistence: a Monte Carlo simulation of the frozen niche-variation model. Am. Nat. **141**: 409–427.

Weeks, S.C., Gaggiotti, O.E., Schenck, R.A., Spindler, K.P., and Vrijenhoek, R.C. 1992. Feeding behavior in sexual and clonal strains of *Poeciliopsis*. Behav. Ecol. Sociobiol. **30**: 1–6.

Williams, G.C. 1975. Sex and evolution. Princeton University Press, Princeton, N.J.

Williams, G.C., and Mitton, J.B. 1973. Why reproduce sexually? J. Theor. Biol. **39**: 545–554.