ELSEVIER

International Journal for Parasitology 30 (2000) 697–703

INTERNATIONAL Journal for PARASITOLOGY

www.elsevier.nl/locate/ijpara

Metazoan parasite species richness and genetic variation among freshwater fish species: cause or consequence?

Robert Poulin*, Leigh J. Marshall, Hamish G. Spencer

Department of Zoology, University of Otago, P.O. Box 56, Dunedin, New Zealand

Received 7 February 2000; received in revised form 21 March 2000; accepted 21 March 2000

Abstract

The factors responsible for the maintenance of genetic variation among natural populations remain a mystery. Recent models of host–parasite co-evolution assume that parasites exert frequency-dependent selection on their hosts by favouring rare alleles that may confer resistance against infection. We tested this prediction in a comparative analysis that sought relationships between levels of genetic variation and the number of metazoan parasite species exploiting each host species. We used data on 40 species of North American freshwater fishes. After controlling for sampling effort and phylogenetic influences, we found no relationship between genetic polymorphism and parasite species richness among fish species. However, we found a marginal negative correlation between parasite species richness and heterozygosity. This result goes against the prediction that increased selective pressure by parasites should be associated with higher levels of genetic variation. Instead, it suggests that parasites may be colonising host species showing low levels of genetic variation with greater success than genetically more variable host species.

Keywords: Comparative analysis; Frequency-dependent selection; Heterozygosity; Metazoan parasites; Phylogeny

1. Introduction

Most natural populations of plants and animals exhibit high levels of genetic variability [1,2]. The forces maintaining this variation, however, are still poorly understood and are the subject of much debate [3–7]. Comparative analyses can shed some light on the mechanisms maintaining genetic variation in natural populations, since they can help identify traits of organisms that are frequently associated with high levels of variation. For instance, Mitton and Lewis [8] found that heterozygosity, or the average proportion of heterozygous loci per individual, correlates with various life history features of fish. Their analysis revealed that the most heterozygous fish species tend to be the ones with smaller body sizes and lower age at maturity. Mitton and Lewis [8] interpreted these results as supporting a role for environmental variability in the maintenance of genetic variation, since unstable environments should select for high intrinsic rates of population increase and thus the kinds of life history traits found to be linked with high levels of genetic variation.

A comparative approach can be used to test other hypotheses regarding the maintenance of genetic variation. A possible mechanism widely discussed in recent years is that parasites and pathogens exert frequency-dependent selection on their hosts, favouring rare host alleles that may confer greater resistance against widespread parasites [9]. Indeed, genetic resistance against parasites quickly becomes obsolete as parasites evolve at higher rates than their hosts. Sexual reproduction may even be an adaptation against parasites that allow gene recombination and promote genetic variation [10–12]. Evidence of the selective pressure placed by parasites on host genetic variability has come from a

* Corresponding author. Tel.: +64-3-479-7983; fax: +64-3-479-7584.

E-mail address: robert.poulin@stonebow.otago.ac.nz (R. Poulin).

0020-7519/00/$20.00
PII: S0020-7519(00)00047-3

few studies on given host species [13–17]. However, only a comparative study among related species can determine whether parasitism explains differences between species in levels of genetic variation.

We examined the relationship between the number of metazoan parasite species exploiting North American freshwater fish species and both their heterozygosity (average proportion of heterozygous loci per individual) and polymorphism (proportion of polymorphic loci among the loci surveyed). Metazoan parasites of fish belong to several taxa, including trematodes, cestodes, nematodes, acanthocephalans, crustaceans and molluscs. They inhabit a wide range of tissues and organs in their hosts, such as the digestive tract, viscera, swim bladder, nervous system, muscles, skin and gills. They invade the host in many different ways and use a variety of mechanisms to evade the host's defenses. Therefore, the strength of selection maintaining genetic variability in hosts should be proportional to the diversity of parasites exploiting a host species if indeed parasites exert frequency-dependent selection on hosts. In other words, if parasites are important in maintaining overall genetic variation, rather than just variation at specific loci known to be involved in parasite resistance, then a simple prediction is that host species showing higher levels of genetic variation do so in response to infections by a wider range of parasite species. We tested this prediction in a comparative analysis among fish species that controlled for important confounding variables such as sampling effort and host phylogeny.

2. Methods

Data on heterozygosity, *H*, and polymorphism, *P*, were obtained from published electrophoretic studies of allozyme variation and used as indices of overall genetic variation. We used two estimates of polymorphism, based on two different criteria for polymorphism: *P99*, for which the frequency of the most common allele had to be $\leqslant 0.99$, and *P95*, for which the frequency of the most common allele had to be $\leqslant 0.95$. Because of the high variance in single-locus polymorphism among loci, estimates of genetic variation are far more severely influenced by the number of loci sampled than by the number of individuals examined [18,19]. Thus, following Nei's [18] suggestions, estimates were only used if at least 20 loci had been investigated from at least five individual fish, or if at least two individuals had been used and scored for at least 25 loci. Nevertheless, because investigators may decide to increase their sample size when little variation is detected, artefactual correlations can appear between numbers of loci or individuals examined and the estimated genetic variation, and we therefore corrected for both variables in the analysis (see below). When two or more studies per fish species were available, we averaged estimates of heterozygosity and polymorphism, weighting by the number of loci, to obtain single values of *H*, *P95* and *P99* per species.

Parasite data were obtained from the checklists of Margolis and Arthur [20] and McDonald and Margolis [21], which include fish species from Canada and the northern USA. For each fish species, we recorded the total number of known metazoan parasite species, whether they infected fish as adults or larval stages and whether they were internal or external parasites. We excluded protozoan parasites, because they are only recorded from fish species that have been the subject of detailed surveys; their occurrence is therefore poorly known for most fish species. The species richness of the metazoan parasite fauna of a given fish species is to some extent dependent on how much effort has gone into studying that fish: on average, more parasites are known from intensely-studied host species than from poorly-studied ones [22,23]. To control for variability in study effort among host species, we used the average number of articles published per year on each fish species over a 10 year period (1984–1993), obtained from Cvancara's compilations [24], as an independent index of our relative knowledge of each fish species. Finally, we obtained data on body size (total length in mm, from Scott and Crossman [25]) for each fish species since this variable may be associated with parasite species richness and mask any relationship with genetic variation [22].

We used Felsenstein's [26] phylogenetically independent contrasts method for the comparative analysis. Contrasts were computed on log-transformed data using the CAIC 2.0 statistical package [27]. The method consists in deriving statistically independent sets of contrasts between sister taxa from a phylogeny, and using these contrasts to test for relationships between variables. We used Nelson's [28] proposed phylogeny of higher taxa of fish, and various sources of information on within-family relationships [29–34]. Contrasts need to be standardised for the time since divergence of sister taxa, or branch lengths in the phylogeny [35]. Since this information is unavailable for the fish phylogeny, we used the estimation method of Grafen [36]. Relationships among contrasts were assessed using correlations forced through the origin (see [35] for justification). To control for the confounding effect of study effort (i.e. number of individuals or loci examined, number of articles per year per fish species), residuals of a regression of contrasts in the variable of interest against contrasts in the confounding variable were used instead of uncorrected contrasts [35].

3. Results

Our data set comprised 40 fish species from nine different families (Table 1). The fully-resolved phylogeny (Fig. 1) allowed a maximum of 39 sets of contrasts to be used; the actual number varies among analyses since data on the three measures of genetic variability were not always available for all fish species.

The three measures of genetic variation correlated

Table 1
Data on parasite species richness and genetic variation for the 40 freshwater fish species included in the comparative analysis

FAMILY species	Parasite richness (study effort)[a]	No. loci[b] (no. fish sampled)[c]	*H*	*P95*	*P99*	Ref.
CATOSTOMIDAE						
Ictiobus cyprinellus	1 (0.8)	32 (15)	0.102	0.281	0.371	[43]
Carpiodes cyprinus	38 (1.8)	31 (15)	0.083	0.323	0.409	[43]
Catostomus columbianus	5 (0.2)	29 (15)	0.048	0.138	0.190	[43]
Catostomus catostomus	67 (4.0)	29 (15)	0.038	0.172	0.236	[43]
Catostomus commersoni	86 (18.2)	30 (85)	0.029	0.094	0.114	[43,44]
Minytrema melanops	0 (0.2)	27 (15)	0.059	0.222	0.293	[43]
Erimyzon sucetta	0 (0.3)	29 (15)	0.058	0.207	0.273	[43]
Moxostoma macrolepidotum	26 (2.2)	27 (15)	0.075	0.222	0.293	[43]
Moxostoma duquesnei	0 (0.2)	27 (15)	0.015	0.111	0.153	[43]
Moxostoma erythrurum	10 (2.3)	27 (15)	0.034	0.148	0.203	[43]
Hypentelium nigricans	4 (0.5)	38 (72)	0.016	0.056	0.066	[43,45]
CYPRINIDAE						
Notemigonus crysoleucas	29 (3.8)	24 (15)	0.068	0.210	0.277	[46]
Notropis emiliae	4 (0.2)	21 (38)	0.036	0.119	0.119	[47]
Pimephales promelas	26 (21.0)	22 (5)	0.197	0.182	0.318	[48]
Hybognathus nuchalis	0 (0.1)	22 (20)	0.064	0.227	0.318	[48]
Hybognathus hankinsoni	11 (0.8)	22 (21)	0.026	0.058	0.221	[48]
Campostoma anomalum	3 (2.0)	22 (367)	0.048	0.141	0.208	[48,49]
Nocomis micropogon	7 (1.0)	43 (15)	0.032	0.047	0.047	[50]
Rhinichthys cataractae	24 (3.2)	43 (346)	0.029	0.047	0.102	[49,50]
ICTALURIDAE						
Ictalurus punctatus	39 (76.3)	23 (13)	0.022	0.040	0.170	[51]
ESOCIDAE						
Esox lucius	65 (26.3)	26 (40)	0.001	0.040	0.040	[52]
SALMONIDAE						
Thymallus arcticus	25 (2.3)	36 (60)	0.033	–	0.110	[49]
Salmo salar	64 (134.0)	43 (1434)	0.023	–	0.101	[49]
Salvelinus fontinalis	90 (42.7)	39 (284)	0.081	0.191	0.249	[53]
Salvelinus namaycush	65 (23.8)	50 (484)	0.015	–	0.142	[49]
Salvelinus alpinus	45 (24.0)	37 (474)	0.006	–	0.016	[49]
Oncorhynchus kisutch	56 (103.7)	27 (1971)	0.016	–	0.129	[49]
Oncorhynchus tshawytscha	29 (83.3)	28 (1516)	0.063	–	0.196	[49,54]
Oncorhynchus gorbuscha	45 (60.7)	29 (910)	0.032	–	0.151	[49]
Oncorhynchus keta	23 (75.3)	28 (706)	0.034	–	0.079	[49]
Oncorhynchus nerka	73 (69.5)	27 (2067)	0.017	–	0.100	[49]
Oncorhynchus mykiss	73 (364.3)	24 (1321)	0.107	0.243	0.356	[55]
Oncorhynchus clarki	24 (18.2)	59 (293)	0.016	0.040	0.051	[56]
CYPRINODONTIDAE						
Fundulus heteroclitus	11 (21.2)	24 (5)	0.170	0.330	0.330	[57]
COTTIDAE						
Cottus bairdi	23 (7.7)	32 (257)	0.020	0.061	0.133	[58]
PERCICHTHYIDAE						
Morone americana	23 (6.5)	33 (29)	–	0.061	0.061	[59]
Morone chrysops	30 (6.5)	33 (142)	–	0.030	0.061	[59]
PERCIDAE						
Stizostedion vitreum	70 (24.3)	39 (80)	0.049	0.128	0.231	[60]
Stizostedion canadense	29 (2.3)	39 (5)	0.009	0.026	0.026	[60]
Etheostoma microperca	1 (9.8)	23 (60)	0.010	–	0.055	[49]

[a] Mean number of articles published per year per fish species.
[b] When data come from more than one source, the number of loci used is from the study that looked at the most loci.
[c] When data come from more than one source, the number of fish is summed up across studies.

positively with one another (H vs $P95$: $r = 0.655$, $n = 27$ sets of contrasts, $P < 0.001$; H vs $P99$: $r = 0.750$, $n = 37$, $P < 0.001$; $P95$ vs $P99$: $r = 0.839$, $n = 29$, $P < 0.001$). Heterozygosity correlated significantly with the number of individual fish examined ($r = -0.352$, $n = 37$ sets of contrasts, $P = 0.03$), whereas both measures of polymorphism correlated with the number of loci investigated ($P95$: $r = -0.432$, $n = 29$, $P = 0.019$; $P99$: $r = -0.303$, $n = 39$, $P = 0.056$). In subsequent analyses, we used corrections for either number of fish or number of loci to control for the influence of these confounding variables. Similarly, we corrected parasite species richness for study effort since these two variables were strongly positively correlated and study effort explains more than 50% of the variance in parasite species richness ($r = 0.724$, $n = 39$ sets of contrasts, $P < 0.001$).

Fish body size tended to covary with parasite species richness ($r = 0.277$, $n = 39$ sets of contrasts, $P = 0.088$), though it explains only about 7% of the variance in richness. However, fish size did not correlate with any measure of genetic variation (H: $r = 0.086$, $n = 37$, $P > 0.50$; $P95$: $r = 0.163$, $n = 29$, $P = 0.39$; $P99$: $r = 0.226$, $n = 39$, $P = 0.16$).

Parasite species richness was weakly associated with heterozygosity (Fig. 2), with the more heterozygous fish taxa tending to harbour fewer parasite species than their less heterozygous relatives ($r = -0.319$, $n = 37$ sets of contrasts, $P = 0.056$). Polymorphism, on the other hand, showed no relationship with parasite species richness ($P95$: $r = -0.146$, $n = 29$, $P = 0.45$; $P99$: $r = 0.012$, $n = 39$, $P > 0.50$; Fig. 3).

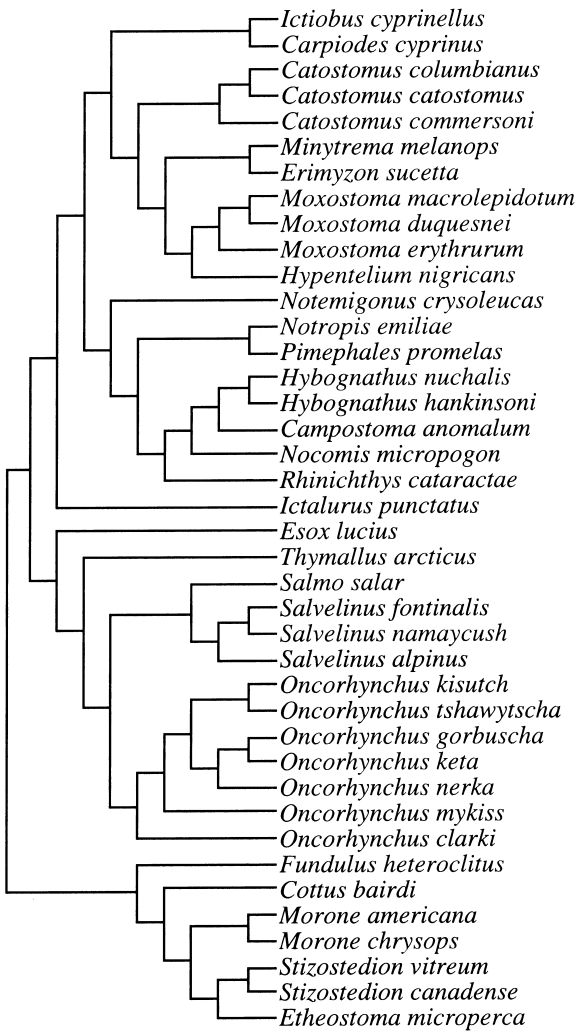

Fig. 1. Phylogeny of the 40 North American freshwater fish species included in the comparative analysis. True branch lengths are not available in this composite tree, so the scale used in the figure is arbitrary.

4. Discussion

Based on previous theoretical work [9], we expected that parasite-mediated frequency-dependent selection should play a role in the maintenance of genetic diversity among fish species. In other words, we expected fish species exposed to a wide range of parasite species to display higher levels of heterozygosity and/or polymorphism because such species are subject to a greater variety of selection pressures. Not only do our results fail to support this prediction, they suggest the opposite, i.e. a negative relationship between parasite species richness and heterozygosity. This result was obtained after controlling for phylogenetic influences and correcting for sampling effort. We also controlled for fish body size; other ecological variables sometimes, but not always, correlate with parasite species

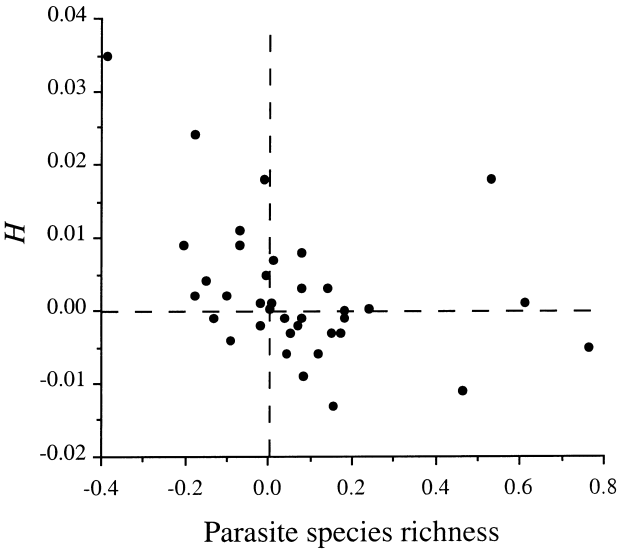

Fig. 2. Relationship between parasite species richness (corrected for study effort) and heterozygosity (H, corrected for the number of individual fish examined) among North American freshwater fish. Data points represent phylogenetically independent contrasts.

richness in fish and other vertebrates, but fish body size is the most pervasive covariate among these [22,37]. We eliminated several fish species from our data set because they had been investigated at fewer than 20 loci and estimates of their genetic variability may have been imprecise [18,19]. In a further analysis in which we lowered our standards to include all fish species that had been investigated at more than 15 loci (this procedure allowed inclusion of species from three additional fish families: Centrarchidae, Gasterosteidae, and Clupeidae), the negative relationship between parasite species richness and heterozygosity remained approximately the same ($r = -0.311$, $n = 42$ sets of contrasts, $P = 0.046$). Therefore, although they may select for variation at some specific loci, parasites do not appear to be involved in the maintenance of variation throughout the genome in general.

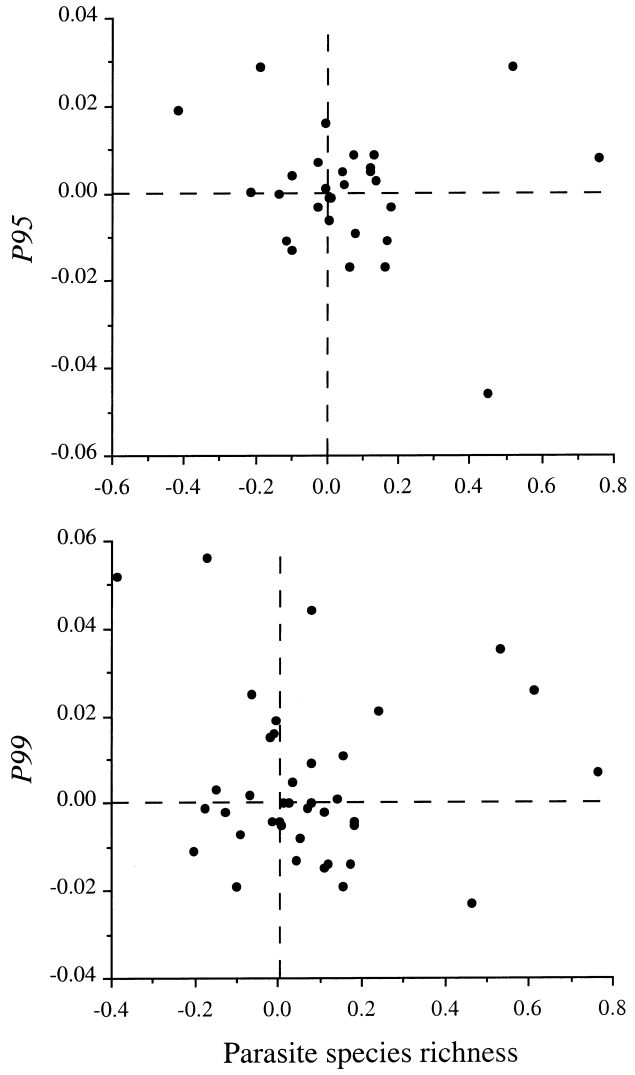

Fig. 3. Relationship between parasite species richness (corrected for study effort) and genetic polymorphism (*P95* and *P99*, both corrected for the number of loci investigated) among North American freshwater fish. Data points represent phylogenetically independent contrasts.

In an earlier study [38], we found no relationship between genetic variation and the prevalence of infection by protozoan blood parasites among bird species. These findings, combined with the results of the present study, provide no support for the idea that parasites maintain genetic variation among host species via frequency-dependent selection. Some other neutral or selection mechanism must be at work. Alternatively, one could argue that the data we used contain too much noise for existing patterns to emerge. For instance, parasite species richness is only one of many ways of estimating the selective pressure exerted by parasites on their hosts. The level of infection by a particularly virulent parasite species may give a better idea of the strength of selection; this approach, however, does not allow comparisons among host species exploited by different parasites. Also, data for parasite species richness and genetic variation should ideally have been obtained from the same fish populations, perhaps even from the same individual animals, rather than compiled from independent studies. While the data have shortcomings, these are not fatal to our analysis. Other comparative studies, using the same type of genetic data, have found correlations between genetic variation and other biological traits among vertebrate species [8,39] despite the potential weakness of the data.

It is interesting to note that our analysis, although not designed to test the suggestion of Mitton and Lewis [8] that environmental variability promotes genetic variation, does not support their conclusions either. We did not find a significant relationship between fish body size and genetic variation, as did Mitton and Lewis [8]. The discrepancy between their results and ours may result from the fact that we eliminated species sampled at fewer than 20 loci from our analysis (they included fish species sampled at 10 or more loci) and that we controlled for phylogenetic influences whereas they did not. Thus it may be necessary to re-evaluate the importance of environmental stability, and other factors such as aquatic habitat type (see [40]), in the maintenance of genetic variation among fishes using a more rigorous comparative approach.

Whatever force (or forces) is actually maintaining genetic variation among natural populations, we propose that parasite species may be more successful at colonising host species or host populations that show low levels of genetic variation. A generally low diversity of possible allelic combinations in hosts may favour parasites in the evolutionary arms race by limiting the range of possible host defenses. This would lead to host species that are genetically homogeneous accumulating parasite species at a higher rate than

related but genetically more variable species. A recent study of mouse populations provides support for this hypothesis. Meagher [41] found that populations of deer mice (*Peromyscus maniculatus*) with low levels of allozyme heterozygosity had higher prevalences of nematode infections than genetically more diverse populations. This result and ours suggest that populations or species of hosts with low levels of genetic variation will be more susceptible to parasite infections. Recently, Guégan and Morand [42] showed that, among African freshwater cyprinid fish, polyploid species harbour more species of ectoparasitic worms than their diploid relatives. This result is the opposite of what we might have expected, since polyploid species have more opportunities to fight parasites via genetic resistance because they have twice or more the number of genes to do so. The evolutionary significance of this result is unclear, but could be related to our suggestion that parasites take advantage of other forces acting on the genome of their hosts. According to this scenario, a high number of parasite species exploiting a host species would be a consequence, and not a cause, of genetic variation in the host.

References

[1] Nevo E. Genetic variation in natural populations: patterns and theory. Theor Popul Biol 1978;13:121–77.

[2] Nei M, Graur D. Extent of protein polymorphism and the neutral mutation theory. Evol Biol 1984;17:73–118.

[3] Lewontin RC. The genetic basis of evolutionary change. New York: Columbia University Press, 1974.

[4] Kimura M. The neutral theory of molecular evolution. Cambridge: Cambridge University Press, 1983.

[5] Nei M. Molecular evolutionary genetics. New York: Columbia University Press, 1987.

[6] Spencer HG, Marks RW. The evolutionary construction of molecular polymorphisms. NZ J Bot 1993;31:249–56.

[7] Kreitman M, Akashi H. Molecular evidence for natural selection. Annu Rev Ecol Syst 1995;26:403–22.

[8] Mitton JB, Lewis Jr. WM. Relationships between genetic variability and life-history features of bony fishes. Evolution 1989;43:1712–23.

[9] Hamilton WD. Pathogens as causes of genetic diversity in their host populations. In: Anderson RM, May RM, editors. Population Biology of Infectious Diseases. New York: Springer, 1982. p. 269–96.

[10] Hamilton WD. Sex versus non-sex versus parasites. Oikos 1980;35:282–90.

[11] Lively CM. Evidence from a New Zealand snail for the maintenance of sex by parasitism. Nature 1987;328:519–21.

[12] Hamilton WD, Axelrod R, Tanese R. Sexual reproduction as an adaptation to resist parasites (a review). Proc Nat Acad Sci USA 1990;87:3566–73.

[13] Lively CM, Craddock C, Vrijenhoek RC. Red Queen hypothesis supported by parasitism in sexual and clonal fish. Nature 1990;344:864–6.

[14] Dybdahl MF, Lively CM. Host–parasite coevolution: evidence for rare advantage and time-lagged selection in a natural population. Evolution 1998;52:1057–66.

[15] Liersch S, Schmid-Hempel P. Genetic variation within social insect colonies reduces parasite load. Proc R Soc London B 1998;265:221–5.

[16] Paterson S, Wilson K, Pemberton JM. Major histocompatibility complex variation associated with juvenile survival and parasite resistance in a large unmanaged ungulate population (*Ovis aries* L.). Proc Nat Acad Sci USA 1998;95:3714–9.

[17] Coltman DW, Pilkington JG, Smith JA, Pemberton JM. Parasite-mediated selection against inbred Soay sheep in a free-living, island population. Evolution 1999;53:1259–67.

[18] Nei M. Estimation of average heterozygosity and genetic distance from a small number of individuals. Genetics 1978;89:583–90.

[19] Gorman GC, Renzi J. Genetic distance and heterozygosity estimates in electrophoretic studies: effects of sample size. Copeia 1979;1979:242–9.

[20] Margolis L, Arthur JR. Synopsis of the Parasites of Fishes of Canada. Ottawa: Fisheries Research Board of Canada, Bulletin 199, 1979.

[21] McDonald TE, Margolis L. Synopsis of the Parasites of Fishes of Canada. Ottawa: Canadian Special Publication of Fisheries and Aquatic Sciences, 122 (Suppl.1978–1993). National Research Council of Canada, 1995.

[22] Poulin R. Phylogeny, ecology, and the richness of parasite communities in vertebrates. Ecol Monogr 1995;65:283–302.

[23] Walther BA, Cotgreave P, Price RD, Gregory RD, Clayton DH. Sampling effort and parasite species richness. Parasitol Today 1995;11:306–10.

[24] Cvancara V. Current References in Fish Research, 9–18. Eau Claire, WI: University of Wisconsin, 1984–1993.

[25] Scott WB, Crossman EJ. Freshwater Fishes of Canada. Ottawa: Fisheries Research Board of Canada, Bulletin 184, 1973.

[26] Felsenstein J. Phylogenies and the comparative method. Am Nat 1985;125:1–15.

[27] Purvis A, Rambaut A. Comparative Analysis by Independent Contrasts (CAIC), version 2.0. Oxford: Oxford University, 1994.

[28] Nelson JS. Fishes of the World. 3rd edition. New York: John Wiley & Sons, 1994.

[29] McVeigh HP, Davidson WS. A salmonid phylogeny inferred from mitochondrial cytochrome b gene sequences. J Fish Biol 1991;39(Suppl):277–82.

[30] Coburn MM, Cavender TM. Interrelationships of North American cyprinid fishes. In: Mayden RL, editor. Systematics, Historical Ecology, and North American Freshwater Fishes. Stanford, CA: Stanford University Press, 1992. p. 328–73.

[31] Smith GR. Phylogeny and biogeography of the Catostomidae, freshwater fishes of North America and Asia. In: Mayden RL, editor. Systematics, Historical Ecology, and North American Freshwater Fishes. Stanford, CA: Stanford University Press, 1992. p. 778–826.

[32] Stearley RF. Historical ecology of Salmoninae, with special reference to *Oncorhynchus*. In: Mayden RL, editor. Systematics, Historical Ecology, and North American Freshwater Fishes. Stanford, CA: Stanford University Press, 1992. p. 622–58.

[33] Wainwright PC, Lauder GV. The evolution of feeding biology in sunfishes (Centrarchidae). In: Mayden RL, editor. Systematics, Historical Ecology, and North American Freshwater Fishes. Stanford, CA: Stanford University Press, 1992. p. 472–91.

[34] Domanico MJ, Phillips RB, Oakley TH. Phylogenetic analysis of Pacific salmon (genus *Oncorhynchus*) using nuclear and mitochondrial DNA sequences. Can J Fish Aquat Sci 1997;54:1865–72.

[35] Garland Jr. T, Harvey PH, Ives AR. Procedures for the analysis of comparative data using phylogenetically independent contrasts. Syst Biol 1992;41:18–32.

[36] Grafen A. The phylogenetic regression. Phil Trans R Soc London B 1989;326:119–57.
[37] Poulin R. Species richness of parasite assemblages: evolution and patterns. Annu Rev Ecol Syst 1997;28:341–58.
[38] Poulin R, Marshall LJ, Spencer HG. Genetic variation and prevalence of blood parasites do not correlate among bird species. J Zool, in press.
[39] Petrie M, Doums C, Møller AP. The degree of extra-pair paternity increases with genetic variability. Proc Nat Acad Sci USA 1998;95:9390–5.
[40] Ward RD, Woodwark M, Skibinski DOF. A comparison of genetic diversity levels in marine, freshwater, and anadromous fishes. J Fish Biol 1994;44:213–32.
[41] Meagher S. Genetic diversity and *Capillaria hepatica* (Nematoda) prevalence in Michigan deer mouse populations. Evolution 1999;53:1318–24.
[42] Guégan J-F, Morand S. Polyploid hosts: strange attractors for parasites? Oikos 1996;77:366–70.
[43] Ferris SD, Whitt GS. Genetic variability in species with extensive gene duplication: the tetraploid catostomid fishes. Am Nat 1980;115:650–66.
[44] Ferris SD, Buth DG, Whitt GS. Substantial genetic differentiation among populations of *Catostomus plebeius*. Copeia 1982;1982:444–9.
[45] Buth DG. Evolutionary genetics and systematic relationships in the Catostomid genus *Hypentelium*. Copeia 1980;1980:280–90.
[46] Avise JC. Genic heterozygosity and rate of speciation. Paleobiology 1977;3:422–32.
[47] Dimmick WW. Phylogenetic relationships of *Notropis hubbsi*, *N. welaka* and *N. emiliae* (Cypriniformes: Cyprinidae). Copeia 1987;1987:316–25.
[48] Cook JA, Besthen KR, Propst DL, Yates TL. Allozymic divergence and systematics of the Rio Grande silvery minnow, *Hybognathus amarus* (Teleostei: Cyprinidae). Copeia 1992;1992:36–44.
[49] Mani GS. Evolutionary Dynamics of Genetic Diversity. Berlin: Springer Verlag, 1983.
[50] Goodfellow WLJ, Hocutt CH, Morgan II RP, Stauffer Jr JR. Biochemical assessment of the taxonomic status of *Rhinichthys bowersi* (Pisces: Cyprinidae). Copeia 1984;1984:652–9.
[51] Kelsch SW, Hendricks FS. An electrophoretic and multivariate morphometric comparison of the American catfishes *Ictalurus lupus* and *I. punctatus*. Copeia 1986;1986:646–52.
[52] Healy JA, Mulcany MF. A biochemical genetic analysis of populations of the northern pike, *Esox lucius* L., from Europe and North America. J Fish Biol 1980;17:317–24.
[53] Stoneking M, Wagner DJ, Hildebrand AC. Genetic evidence suggesting subspecies differences between northern and southern populations of brook trout (*Salvelinus fontinalis*). Copeia 1981;1981:810–9.
[54] Utter F, Milner G, Stahl G, Teel D. Genetic population structure of chinook salmon, *Oncorhynchus tshawytscha*, in the Pacific Northwest. US Fish Bull 1989;87:239–64.
[55] Currens KP, Schreck CB, Li HW. Allozyme and morphological divergence of rainbow trout (*Oncorhynchus mykiss*) above and below waterfalls in the Deschutes River, Oregon. Copeia 1990;1990:730–46.
[56] Bartley DM, Gall GAE. Genetic identification of native cutthroat trout (*Oncorhynchus clarkii*) and introgressive hybridization with introduced rainbow trout (*O. mykiss*) in streams associated with the Alvord Basin, Oregon and Nevada. Copeia 1991;1991:854–9.
[57] Rogers JS, Cashner RC. Genetic variation, divergence, and relationships in the subgenus *Xenisma* of the genus *Fundulus*. In: Matthews WJ, Heins DC, editors. Community and evolutionary ecology of North American stream fishes. Norman, OK: University of Oklahoma Press, 1987. p. 251–64.
[58] Howard JH. Allozyme variation in the mottled sculpin (*Cottus bairdi*): a test of stream capture hypotheses. Copeia 1993;1993:870–5.
[59] Todd TN. Occurrence of white bass–white perch hybrids in Lake Erie. Copeia 1986;1986:196–9.
[60] Billington N, Hebert PDN, Ward RD. Allozyme and mitochondrial DNA variation among three species of *Stizostedion* (Percidae): phylogenetic and zoogeographical implications. Can J Fish Aquat Sci 1990;47:1093–102.