

Parasites and the Evolutionary Diversification of Primate Clades

Charles L. Nunn,^{1,*} Sonia Altizer,^{2,†} Wes Sechrest,^{3,‡} Kate E. Jones,^{3,§} Robert A. Barton,^{4,||} and John L. Gittleman^{3,#}

1. Section of Evolution and Ecology, University of California, Davis, California 95616;

2. Department of Environmental Studies, Emory University, Atlanta, Georgia 30322;

3. Department of Biology, University of Virginia, Charlottesville, Virginia 22904;

4. Evolutionary Anthropology Research Group, Department of Anthropology, University of Durham, Durham DH1 3HN, United Kingdom

ABSTRACT: Coevolutionary interactions such as those between hosts and parasites have been regarded as an underlying cause of evolutionary diversification, but evidence from natural populations is limited. Among primates and other mammalian groups, measures of host diversification rates vary widely among lineages, but comparative studies have not yet identified a reliable explanation for this variation. In this study, we used a comprehensive data set of disease-causing organisms from free-living primates to illustrate how phylogenetic comparative methods can be used to examine mammalian lineage diversity in relation to parasite species richness. Our results provide evidence that the phylogenetic diversity of primate clades is correlated positively with the number of parasite species harbored by each host and that this pattern is largely independent of other host traits that have been shown to influence diversification rates and parasite species richness in primates. We investigated two possible mechanisms that could explain this association, namely that parasites themselves drive host evolutionary diversification through processes linked with sexual selection and that host shifts or host sharing increases parasite species richness among diverse primate

clades. Neither parasite species richness nor host diversification is related to measures of sexual selection in primates. Further, we found only partial evidence that more rapidly diversifying host lineages produced increased opportunities for host sharing or host shifting by parasites through mechanisms involving species' geographic range overlap. Thus, our analyses provide evidence for an important link between the evolutionary diversification of primates and the richness of their parasite communities, but other mechanisms, particularly those related to reciprocal selection or coextinction of hosts and parasites, require further investigation.

Keywords: comparative study, host-parasite interactions, primates, sexual selection, geographic range, species richness.

Interspecific interactions, including those between hosts and parasites, are thought to play an important role in the diversification of natural populations. Several convincing examples have shown that host-parasite coevolution can lead to surprisingly high levels of genetic diversity within and among interacting populations (Dybdahl and Lively 1998; Burdon and Thrall 1999; Altizer 2001), and geographic variation in traits associated with parasite resistance suggests a role for infectious agents in host allopatric divergence (Berenbaum and Zangerl 1998; Kaltz and Shykoff 1998; Thompson 1999). Moreover, major historical diversification events have been linked to species interactions. For example, phylogenetic analyses have been used to examine the hypothesis that coevolution among plants and insect herbivores has increased rates of diversification among selected lineages, driven in part by host plant specialization and novel defensive or counterdefensive strategies (e.g., Mitter et al. 1991; Farrel 1998; Percy et al. 2004). Despite their inferred importance, however, direct evidence for the role of coevolutionary interactions in the phylogenetic diversification of host and natural enemy lineages is remarkably limited, and existing studies of host-parasite evolutionary diversification have focused on relatively narrow taxonomic groups (e.g., Johnson and Clayton 2002; Poulin 2002; Zietara and Lumme 2002).

Clades of the same age vary markedly in the number of species they contain (Purvis and Hector 2000; Magellon and Sanderson 2001). Variables that influence the speci-

* Corresponding author. Present address: Department of Integrative Biology, University of California, Berkeley, California 94720-3140; e-mail: cnunn@socrates.berkeley.edu.

† E-mail: saltize@emory.edu.

‡ Present address: World Conservation Union Species Survival Commission and Conservation International's Center for Applied Biodiversity Science, Washington, D.C. 20036; e-mail: wes.sechrest@iucn.org.

§ Present address: Center for Environmental Research and Conservation, Columbia University, New York, New York 20027; e-mail: kj2107@columbia.edu.

|| E-mail: r.a.barton@durham.ac.uk.

E-mail: jlgittleman@virginia.edu.

ation and extinction of lineages should determine overall patterns of diversification (Hey 1992; Nee et al. 1994*a*, 1994*b*; Purvis 1996; Paradis 1997; Nee 2001). Phylogenetic trees and recently developed comparative methods allow researchers to quantify diversification rates and compare these rates across lineages, in part to investigate whether particular intrinsic traits or environmental pressures are associated with species-rich groups (reviewed in Purvis 1996; Sanderson and Donoghue 1996; Barraclough and Nee 2001). Additional methods are available to estimate the key components of diversification rate, specifically rates of speciation and extinction (Nee et al. 1994*a*, 1994*b*; Purvis 1996), and to compare evolutionary rates and branching patterns of lineages of interacting species (e.g., Hafner and Page 1995; Zietara and Lumme 2002).

In this article, we used primates and their infectious diseases as a model system to explore comparative patterns and mechanisms underlying the relative diversity of host lineages. Recent comparative studies have examined body size, life history, resource specialization, morphological innovations, and sexual selection as factors that might explain why some groups are more diverse than others (Farrell 1998; Owens et al. 1999; Desdevises et al. 2001; Panhuis et al. 2001; Gage et al. 2002; Mayhew 2002; Morrow et al. 2003). Body size has received the most attention in diversification analyses of birds and mammals, with lower diversification expected among larger-bodied animals because these species should be more susceptible to extinction and should show lower speciation rates relative to smaller-bodied animals (Peters 1983; Brown 1995; Gardezi and da Silva 1999). Counter to these predictions, however, body mass has failed to reliably account for differences in species diversity among sister clades in comparative studies spanning a broad range of animal groups (Nee et al. 1992; Gittleman and Purvis 1998; Gardezi and da Silva 1999; Katzourakis et al. 2001; Gage et al. 2002; Orme et al. 2002*a*, 2002*b*). In fact, virtually no consistent explanation has emerged in broader comparisons of the evolutionary diversification across mammalian clades (e.g., Gittleman and Purvis 1998; Gage et al. 2002; Morrow et al. 2003).

Interactions between species have also received much attention as potential drivers of evolutionary diversification among coevolving lineages. For example, pollination and seed dispersal by animals have been proposed to account for major shifts in the diversification of angiosperms (e.g., Pellmyr 1992; Bawa 1995). Theoretical and empirical studies have focused on the Red Queen process of host-parasite coevolution in maintaining genetic diversity within and among populations (Anderson and May 1991; Lively and Apanius 1995; Abrams 2000). From a macroevolutionary perspective, theoretical work has shown that frequency-dependent selection between prey and natural enemies can lead to evolutionary branching in both the

host and enemy populations (Doebeli and Dieckmann 2000). Phylogenetic analyses (combined with related fossil evidence) have been used to examine the hypothesis that coevolution among plants and insect herbivores has increased rates of diversification within certain lineages, driven in part by host plant specialization and novel defensive or counterdefensive strategies (e.g., Ehrlich and Raven 1964; Mitter et al. 1991; Farrel 1998). Moreover, a recent empirical study of coevolution between bacteria and virulent phage in spatially structured environments demonstrated that parasites can drive allopatric divergence among host populations, increasing host diversification by selecting for antiparasite defenses linked to different traits in different populations (Buckling and Rainey 2002).

Virtually no comparative studies have applied phylogenetic tools to test for a positive association between host and parasite diversity, with the exception of studies examining cospeciation among particular interacting lineages (Hafner and Page 1995; Hugot 1999; Johnson and Clayton 2002; Poulin 2002; Zietara and Lumme 2002). Here, we investigate parasite species richness as a correlate of primate evolutionary diversity, asking whether host diversity and parasite species richness are positively correlated. It is also important to consider the possibility that parasites could reduce host diversity by increasing extinction rates, thus leading to a negative relationship between measures of host and parasite diversity. Primate clades show variation in species diversity (Purvis et al. 1995; Paradis 1998). Because they are one of the best-studied groups of mammals with species-level information available for a wide range of traits and because they harbor a diverse array of parasites and infectious diseases (Nunn et al. 2003), primates provide an opportunity to investigate the links between parasite richness and host diversity. As with recent studies of other organisms (Gage et al. 2002; Morrow et al. 2003), previous attempts to account for variation in primate diversity have produced few compelling results, with little or no support for an effect of body mass, life history, or group size (Gittleman and Purvis 1998; Isaac et al., in press).

To investigate the association between host and parasite diversity, we applied phylogenetic comparative methods to a comprehensive data set encompassing a wide range of micro- and macroparasites from wild primate populations, and we used multivariate statistics to control for potentially confounding variables. These are two major strengths of our approach because many previous studies of the correlates of evolutionary diversification have focused on single host traits and most previous studies of host-parasite coevolution have focused on relatively narrow taxonomic groups. In addition to sampling effort (Gregory 1990), we included five socioecological variables that have been shown to correlate with parasite species richness in pri-

mates: population density, body mass, geographic range size, host longevity, and day journey length (Nunn et al. 2003).

Finally, we examined two mechanisms that might underlie a significant positive association between host diversity and parasite species richness, namely that (1) parasites increase the speciation rates of their hosts and (2) increased host lineage diversity leads to greater parasite species richness. To examine mechanism 1, we focused on sexual selection as a process linking both parasitism and host speciation, testing whether measures of sexual dimorphism were correlated with parasite species richness and primate diversification rates. We focused on sexual selection because mate preferences have been shown to be important in reproductive isolation and speciation in other taxonomic groups (Lande 1981; Barraclough et al. 1995; Gavrilets 2000; Panhuis et al. 2001; Turelli et al. 2001). Moreover, past work has highlighted sexually selected traits as indicators of parasite resistance (Hamilton and Zuk 1982; Read 1988), providing a mechanism by which hosts with high parasite pressure might demonstrate greater potential for speciation. To examine mechanism 2, we focused on links between host geographic range overlap (sympatry), parasite species richness, and host diversification. In particular, sympatric host populations might provide greater opportunities for host sharing by generalist parasites and host shifting by specialist parasites, leading to greater parasite richness on a per host basis (Raibaut et al. 1998; Coombes 2001; Dobson and Foufopoulos 2001; Roy 2001; Taylor et al. 2001). Thus, we tested whether geographic range overlap correlates with both parasite species richness and primate evolutionary diversity.

Methods

Host-Parasite Records

The primary analyses make use of two sources of data: records of parasites in primates and the evolutionary diversity of primate clades. A comprehensive data set on parasites that infect free-living primates was compiled from the published literature. We located records of micro- and macroparasites using primate Latin binomials as keywords in searches of major online reference databases (Biological Abstracts, AGRICOLA, Medline, Web of Science, and PrimateLit). We also searched by primate genus name and common taxonomic variants (based on Rowe 1996; Groves 2001). In addition to electronic databases, we examined edited volumes and reviews (e.g., Fiennes 1972; Brack 1987) and studies that were cited by publications that we located in our first round of searches. The data set used here is an updated version of the data set on anthropoid primates (monkeys and apes) used by Nunn

et al. (2003) and also includes 15 species of prosimians (lemurs and lorises). Although not reported here, similar results were obtained with previous versions of the database and when analyses were restricted to anthropoids.

For each parasite reported from a wild primate population, we recorded the type of parasite (virus, protozoan, fungi, arthropod, helminth, bacteria), parasite genus and species names, host genus and species names (later revised according to Corbet and Hill 1991), and information on the location and method of sampling. Data collectively represented 934 unique host-parasite combinations, which included 119 primate species and 386 parasite species in 160 genera. Relative to previous comparative studies of parasite species richness in mammals that used independent contrasts (e.g., Poulin 1995; Morand and Harvey 2000), this is a relatively large data set, and the number of primate species included in this study is comparable to other recent analyses of evolutionary diversification in mammals (e.g., Gittleman and Purvis 1998; Isaac et al., in press). Host species were included only if they had been sampled for parasites, and the main predictor variable was the number of parasite species reported per host species. In addition to examining all parasites combined, we repeated analyses separately for protozoa, helminths, and viruses. Collectively, these three parasite groups are the best studied from wild primates (Nunn et al. 2003), representing 88% of all host-parasite combinations in the data set and spanning a diverse array of parasite life cycles and transmission strategies. Further details on this database are provided by Nunn et al. (2003).

Controlling for Sampling Effort

A host might have few parasite records because it naturally harbors few parasite species or because it has not been sampled adequately in the wild (Gregory 1990). We controlled for uneven sampling effort by using least squares residuals from the regression of the log-transformed number of parasite species documented for each host species on a measure of sampling effort for that species. As the primary measure of sampling effort, we used the number of citations for each host species (and common taxonomic variants) from an online database, PrimateLit (<http://primatelit.library.wisc.edu/>), without restricting to studies of parasites or infectious disease. PrimateLit provides the most complete reference information for journal articles and books on primates, and it most closely matches the sampling period (1940 to present) during which most of the studies in our database were published. We found a highly significant association between log-transformed citation counts and log-transformed parasite diversity, with all P values $<.001$ (e.g., regression of all parasites combined on Primate Lit, independent contrasts: $F = 32.4$, $df =$

1,107, $P < .0001$; species values: $F = 60.9$, $df = 1,117$, $P < .0001$). The association between log-transformed citation counts and parasite numbers was linear in all phylogenetic and most nonphylogenetic analyses, with the quadratic term nonsignificant in nonlinear regression (e.g., all parasites, independent contrasts using lineages in diversification analysis: $t = 0.03$, $df = 70$; species values, using all hosts: $t = 1.40$, $df = 116$; the one exception to this general pattern involved virus diversity but only in the nonphylogenetic tests used to investigate hypothesized mechanisms). Use of alternative citation indices gave congruent results, including analyses based on the Web of Science (<http://isi0.isiknowledge.com/>) and Research Libraries Group anthropology citations (<http://eureka.rlg.org/>). Consistent results also were obtained when controlling for sampling effort by including citation counts as an independent variable in multiple regression rather than using residuals.

Phylogenetic Diversity of Primates

We used information on primate phylogeny (Purvis 1995) to quantify variation in primate host diversity. We did not incorporate information on the phylogeny of parasites, however, because we lacked comprehensive information spanning the diverse clades of parasitic organisms in the database, even in analyses restricted to helminths, viruses, or protozoa. Moreover, a strict phylogenetic approach based on cospeciation (Hafner and Page 1995; Hugot 1999; Morand et al. 2000) is impossible to implement when generalist parasites infect a wide range of hosts. Indeed, 56% of the parasites in our database were classified as generalists, meaning that they were reported to infect a range of hosts at the taxonomic level of family or above, in many cases also infecting nonprimate hosts. Thus, we followed previous researchers (Gregory 1990; Poulin 1995; Gregory et al. 1996; Morand 2000) by measuring the number of parasite species reported to infect a given host species after controlling for sampling effort, as described above.

We used the computer program MacroCAIC 1.1.1 (Agapow and Isaac 2002) to investigate the correlates of host phylogenetic diversity. This program generates phylogenetically independent contrasts in a continuous predictor variable at each node with three or more descendents and compares this to the number of species within the clade represented at each node. On the basis of the simulation results of Isaac et al. (2003), we used two measures of host phylogenetic diversity. One measure of diversity, the relative rate difference (RRD), is calculated as the natural logarithm of the ratio of the number of species in sister clades. A second measure, the proportional dominance index (PDI), is the ratio of the number of species in one

of two clades against the total number of species in both clades combined and standardizes this ratio so that it ranges from -0.5 to 0.5 (see Agapow and Isaac 2002; Isaac et al. 2003). As expected, these two measures were strongly positively associated in our data set ($t = 46.5$, $df = 72$, $P < .0001$; see Isaac et al. 2003 for details on how RRD and PDI relate to each other).

Evaluating the Importance of Other Host Traits

Any association between parasite species richness and host lineage diversity could exist because a "third" factor drives variation in both of these variables. To investigate potentially confounding effects of host traits that could influence patterns of host diversification and parasite species richness (Gittleman and Purvis 1998; Nunn et al. 2003; Isaac et al., in press), we compiled data on five host traits shown to be important predictors of parasite species richness (Nunn et al. 2003), including primate body mass (Smith and Jungers 1997), geographic range size (Sechrest et al. 2002; Global Mammal Assessment, unpublished data), longevity (Ross and Jones 1999), day journey length, and population density (unpublished comparative databases; see Nunn and van Schaik 2001). We used stepwise regression to examine the effects of these variables, along with measures of parasite species richness, on primate evolutionary diversity (mixed model run with all variables entered and then rerun with all variables removed; criterion for entry or removal of variables: $P = .10$).

Mechanisms Affecting Host and Parasite Diversity

As noted in the introduction to this article, we investigated two mechanisms that might account for an association between host and parasite diversity. First, we investigated whether parasites influence rates of host diversification by examining associations between sexual selection, host evolutionary diversity, and parasite species richness. We predicted that if parasites drive host cladogenesis through a mechanism involving sexual selection, then both parasite species richness and host evolutionary diversification should be correlated with measures of sexual selection. For these analyses, we compiled information on probable measures of sexual selection, including body mass dimorphism (male mass divided by female mass; Smith and Jungers 1997), canine size dimorphism (based on principal components analysis of six canine dimensions; Plavcan and van Schaik 1992), the presence of exaggerated sexual swellings in females (among catarrhine primates only; Nunn 1999), and patterns of sexual dichromatism and male genital coloration (R. Barton, unpublished comparative data set compiled from Rowe 1996, Dixson 1998, and other sources). In the dichromatism analyses, we recoded six

intermediate species as dichromatic (only one contrast was available when these were coded as nondichromatic). For male genital coloration, we compared species in which males exhibit red, pink, or blue on genitals, face, or chest to those without such coloration.

Second, we tested for associations between host geographic range overlap, parasite species richness, and host diversity. We predicted that if diverse host clades support more parasite species through host shifts or host sharing, then both host diversity and parasite richness should correlate positively with measures of primate geographic range overlap. We collected data on the geographic range for each host species in our data set using geographically rectified range maps for each of the world's primate species as compiled in a geographic information system (Sechrest et al. 2002; Global Mammal Assessment, unpublished data). Host species synonyms were obtained from Groves (2001) for conversion to Corbet and Hill's (1991) taxonomy, which was used to organize host-parasite combinations and to conduct phylogenetic tests using Purvis's (1995) phylogeny. From these maps, we calculated the amount of geographic range overlap in two ways. First, we calculated the number of primate species with ranges that overlapped to any degree with the focal species (number overlapping). Our second measure of overlap accounted for the total overlapping area relative to the focal species geographic range size (total overlapping area_(species i) = geographic range size_(species i) × \sum (percent overlap_(% species i area within n's area))/100). Although primate species geographic ranges have undoubtedly shifted over evolutionary time, the hypothesized mechanism that we tested is explicitly ecological; thus, present ranges were suitable for investigating patterns with geographic range overlap. To explore whether host shifting or host sharing generated parasite diversity, we repeated analyses by dividing parasites into "specialists" (those that were host or genus specific) and "generalists" (those that were family, order, or class specific). The proportion of generalist parasites per primate host ranged from 0% to 100%, with the mean percentage of generalists per host species at 41% (\pm 33% SD; S. M. Altizer, unpublished data).

Assumptions of Comparative Methods and Statistical Analyses

MacroCAIC makes a number of assumptions that should be tested (Agapow and Isaac 2002; Isaac et al. 2003), many of which are similar to the assumption checks required when using independent contrasts (Harvey and Pagel 1991; Garland et al. 1992; Purvis and Rambaut 1995). Before running analyses, we therefore tested whether nodal trait values and standard deviations were correlated with absolute values of contrasts (Orme et al. 2002b; Isaac et

al. 2003). We found violations in analyses of some variables, and no single transformation of data and branch lengths was found to correct all violations of the assumptions, particularly standardization of contrasts with respect to time, which affects the homoscedasticity of the contrasts. Thus, we conducted the major analyses of parasite species richness and host lineage diversity using two different sets of branch lengths (log-transformed branches proportional to time and equal branches), we tested the generality of parametric tests under different methods, and we used one nonparametric test that makes fewer assumptions. For parametric tests, we present standard statistical tests with regression lines forced through the origin (Isaac et al. 2003), as generally required for analysis of independent contrasts (Harvey and Pagel 1991) and as recommended on the basis of computer simulation tests of MacroCAIC (Isaac et al. 2003). Results were generally similar when the intercept was free to vary. We also retested patterns after removing outliers (using Mahalanobis distance measures, calculated in JMP 5.0.1; see Nunn and Barton 2000, 2001). Use of equal branch lengths and time-based (gradual) branch lengths had little effect on the results, and so we present results using the latter set of branches. The nonparametric test (Spearman rank order correlation coefficient, r_s) helps to deal with errors in branch lengths and the standardization of contrasts.

For tests that investigated the mechanism accounting for the association between host diversification and parasite species richness, we examined correlated trait changes using species values (nonphylogenetic tests) and independent contrasts (Felsenstein 1985) calculated using the program CAIC (Purvis and Rambaut 1995). Data and branch lengths were log transformed in analyses of the possible mechanisms, and we examined output with and without outlying contrasts (Nunn and Barton 2000, 2001). Most analyses involved two or more continuous traits, but a subset of analyses involving sexual selection required analysis of discrete and continuous traits in the same test. The computer program CAIC provides a procedure called BRUNCH that examines evolutionary change in continuous traits over transitions in a discretely coded variable. To investigate how discrete traits influence patterns of host diversity, we first calculated RRD and PDI contrasts in MacroCAIC with the discrete trait identified as the independent variable. We then identified transitions in the discrete trait using the BRUNCH algorithm in CAIC, excluded the other RRD and PDI values from the MacroCAIC output, and tested whether the mean of the remaining RRD and PDI values was significantly different from 0.

In tests of the hypothesis that parasite and host diversity are correlated, we had no a priori expectation for the direction of the relationship because higher parasite diversity

could reduce host diversity through the effects of increased extinction. Hence, we tested the association between parasite species richness and host diversity using two-tailed tests, with the level of significance set at .05. For analyses of mechanisms involving sexual selection and host sympatry, however, we used directed tests because these enable detection of patterns that are opposite to predictions while retaining much of the statistical power of one-tailed tests (Rice and Gaines 1994). Directed tests allocate a disproportionate probability under the null hypothesis to the tail of the distribution in the predicted direction (γ) while retaining a smaller probability in the other tail to detect unexpected deviations in the opposite direction ($\delta < \gamma$). Directed tests are subject to the constraint that $\delta + \gamma = \alpha$. We followed the guidelines of Rice and Gaines (1994) by setting γ/α to 0.8, giving values of $\gamma = 0.04$ and $\delta = 0.01$.

Results

Associations between Parasite Species Richness and Host Diversification

Total parasite species richness was significantly positively correlated with the evolutionary diversity of primate clades (fig. 1). Thus, parasite diversity on a per host species basis was higher among primates from relatively more diverse clades. The direction and strength of results for overall parasite species richness were similar for both measures of host diversification (RRD vs. PDI) and when using non-parametric tests (table 1). Results involving tests of all parasites combined were insensitive to exclusion of out-

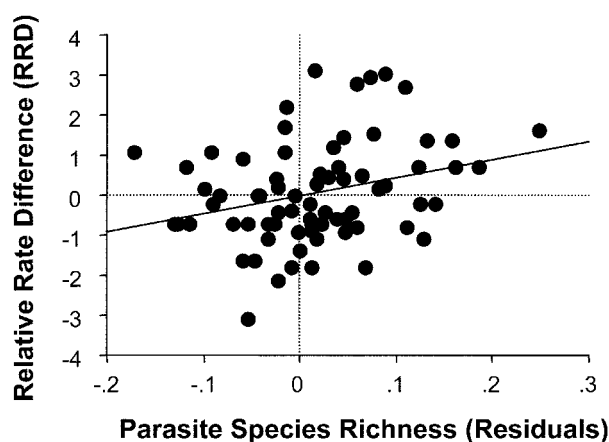


Figure 1: Overall parasite species richness and host phylogenetic diversity. Plots show host diversity contrasts (relative rate difference) versus evolutionary changes in parasite species richness after controlling for sampling effort.

Table 1: Associations between parasite species richness and host phylogenetic diversity

	RRD	PDI	r_s	Ratio of positive to negative diversity contrasts
All parasites	2.54*	2.45*	.27*	40+/29-
Helminths	1.62	1.41	.09	28+/41-
Protozoa	2.08*	1.84	.18	38+/31-
Virus	2.13*	2.25*	.26*	38+/31-

Note: Table shows t statistics, with sign of the t statistic corresponding to the slope of the relationship in bivariate regression analyses; a nonparametric correlation coefficient (Spearman's r_s); and the proportion of diversity measures that were positive (after excluding four contrasts involving no difference in diversity). Two-tailed, $n = 73$ contrasts. RRD = relative rate difference; PDI = proportional dominance index.

* $P < .05$.

liers, although the total amount of variation accounted for was low in these tests ($r^2 < 0.10$).

Separate analyses based on viral, protozoan, and helminth parasite species richness showed that the strength of the relationship between host diversification and parasite diversity varied among parasite groups. Viruses were largely responsible for the positive association between overall parasite species richness and host diversity (table 1), with some analyses of protozoa also producing statistically significant results. For helminths, the direction of the overall relationship was always positive, but most of the contrasts were negative, and no results were statistically significant. Results were generally similar after excluding outliers for all tests involving PDI, but protozoa and virus diversity were no longer significant predictors of RRD when outliers were removed ($t_s = 1.80$ and 1.78 , respectively). Because significance levels in tests involving protozoa depended on the measure of diversification (table 1), we also repeated tests using the two major phyla of protozoa reported from primates. Significant results were obtained in analyses of Apicomplexa (RRD: $t = 2.27$, $df = 72$, $P = .026$) but not Euglenozoa (RRD: $t = 0.52$, $df = 72$).

The Importance of Other Host Traits

For analyses of the association between total parasite species richness, host diversification, and other primate traits, no host socioecological variables were significant predictors of host diversity in any of the stepwise analyses (e.g., for RRD, P values ranged from .08 for longevity to .83 for female mass when all variables were entered into the model). By comparison, total parasite species richness remained a significant predictor of host diversity in all tests (table 2). In tests that examined specific parasite groups separately, parasite species richness was a statistically sig-

Table 2: Associations between parasites and host diversification, controlling for potentially confounding primate socioecological traits

	RRD	PDI	Other socioecological variables entered
All parasites	3.75***	3.38***	Longevity ($t = -1.99^\dagger$)
Helminths	2.27*	1.96 [†]	None
Protozoa	2.67*	2.33*	None
Virus	2.37*	2.24*	None

Note: Table shows t statistics, with sign of the t statistic corresponding to the slope of the relationship in multiple regression analyses. Two-tailed, $n = 43$ contrasts. Variables used in the stepwise model: parasite species richness, body mass, longevity, population density, day range length, and geographic range size. Results differ from table 1 for helminths, protozoa, and viruses because a different number of contrasts were used (i.e., only those species with information on all variables were included in these tests). RRD = relative rate difference; PDI = proportional dominance index.

[†] $P < .10$.

* $P < .05$.

*** $P < .001$.

nificant predictor of primate diversity in all tests for protozoa and viruses, with helminths significant in one test and nearly significant in the other test (table 2), but again, no socioecological variables were significant as covariates. In tests with three outliers removed, population density was the only host trait that was statistically significant, and this reduced the relative importance of total parasite species richness and the richness of helminths, protozoa, and viruses tested separately (e.g., for all parasites and RRD, population density: $t_s = 2.32$, $P = .03$; residual parasite species richness: $t_s = 1.81$, $P = .08$, two-tailed test; see also Isaac et al., in press). Thus, the association between host evolutionary diversity and parasite species richness (fig. 1) was largely independent of other host traits that have been shown to influence host diversity and parasite richness, but this depended on the method of analysis.

Mechanism 1: Parasites as Drivers of Host Diversity

To investigate mechanisms that could account for the pattern shown in figure 1, we first examined the association between parasite species richness and five measures of sexual selection in primates involving female choice or male-male competition (table 3). Body mass dimorphism was significantly associated with parasite diversity in only one phylogenetic test (involving viruses) and in some nonphylogenetic tests (viruses and body dimorphism: $t = 2.99$, $df = 113$, $P = .002$). Sexual dichromatism also accounted for variation in parasite species richness in some nonphylogenetic tests (all parasites: $t = 2.15$, $P = .034$; helminths and protozoa also were significant), but these results became nonsignificant after controlling for primate

phylogeny (table 3). Moreover, one-half of the comparative tests produced results consistent with a negative association, suggesting that the lack of significant results is not due to low statistical power.

Second, we examined the association between measures of sexual selection and primate diversification using the program MacroCAIC. We found no evidence for a link between sexual selection and host diversity, with all tests producing nonsignificant patterns in the direction opposite to predictions (table 3). Thus, we found no evidence for the hypothesis that parasites drive greater host speciation as mediated by their potential links with traits under sexual selection.

Mechanism 2: Hosts as Drivers of Parasite Diversity

To test whether parasite species richness is higher in diverse lineages as a result of greater opportunities for host sharing or host shifting among sympatric primates, we examined whether parasite species richness increased with two measures of host geographic range overlap. Associations between parasite species richness and host geographic overlap were significant for both total parasite diversity and viruses tested separately (table 4). Among specialist parasites, measures of host geographic range overlap were correlated positively with total parasite diversity and with the diversity of protozoa and viruses tested separately. For generalist parasites, however, measures of primate geographic range overlap were not significantly correlated with parasite richness (viruses were the only exception; table 4). Results remained similar when restricted to anthropoid primates, but nonphylogenetic tests produced few significant results.

Despite positive associations between host geographic range overlap and parasite diversity, we found no support for the prediction that host evolutionary diversity was associated with greater host sympatry (table 5). Moreover, host sympatry measures were correlated with total geographic range size. Although larger geographic ranges could favor host sharing or host shifting among primate taxa, larger ranges might also increase opportunities for parasite exposure because of greater habitat diversity or greater contact with nonprimate taxa. Indeed, when geographic range was taken into account, overlap measures were less commonly significant in table 4, except in the case of viruses (e.g., number overlapping and specialist parasites: $t = 2.22$, $df = 105$, $P = .028$; generalist parasites: $t = 2.04$, $df = 105$, $P = .044$, directed tests).

Discussion

An increasing number of studies have demonstrated differences in the potential for evolutionary diversification

Table 3: Associations between measures of primate sexual selection and parasite diversity and host phylogenetic diversity

	Contrasts analysis (CAIC)					MacroCAIC analysis		
	<i>N</i>	All parasites	Helminths	Protozoa	Viruses	<i>N</i>	PDI	RRD
Body dimorphism	105	.97	1.08	.85	1.79*	72	-1.67	-1.60
Canine dimorphism	54	-1.34	-.46	-.79	-1.58	41	-1.13	-1.11
Sexual swellings	4	-.17	.62	-.59	.44	5	-.32	-.53
Sexual dichromatism	7	.33	1.34	1.36	-1.10	5	-2.03	-2.06
Bright genitalia	12	-.62	.06	-1.71	-.15	7	-1.64	-1.61

Note: Table shows *t* statistics, with sign of the *t* statistic corresponding to the slope of the relationship. Analyses were conducted with directed tests, as described in "Methods." Parasite measures were residuals after controlling for sampling effort. In analyses of body mass dimorphism and canine dimorphism, we controlled for body mass, but results were similar when body mass was not included. Analyses using MacroCAIC provide fewer contrasts because comparisons involving sister species are uninformative (i.e., no difference in diversity is possible with only two species). RRD = relative rate difference; PDI = proportional dominance index.

* $P < .05$.

among taxonomic lineages and have explored hypotheses for underlying mechanisms (Purvis and Hector 2000), including factors affecting rates of speciation or extinction across different clades (Barraclough and Nee 2001). Antagonistic coevolution can also operate through intersexual conflict and has been proposed as a major factor that promotes diversification in lineages of hosts and their natural enemies, specifically by increasing speciation rates and evolutionary divergence among populations (Rice and Holland 1997; Parker and Partridge 1998; Arnqvist et al. 2000; Doebeli and Dieckmann 2000; Gavrillets 2000). Although previous studies have focused on parasites as maintaining host genetic diversity within single host-parasite systems (e.g., Dybdahl and Lively 1998; Burdon and Thrall 1999) and patterns of cospeciation among narrowly defined host and parasite lineages (e.g., Hafner and Page 1995; Johnson and Clayton 2002), our article represents the first comparative analysis of broad patterns of host diversity in relation to parasite diversity across a wide array of parasite taxa.

Our results indicate a robust positive relationship between parasite species richness and host phylogenetic diversity. In other words, primates from more diverse lineages harbor a greater number of parasite species on a per host species basis. Results are most consistent in analyses of all parasites combined and for viruses and protozoa examined separately. This pattern could emerge if more diverse clades of primates are better sampled for parasites, but our use of citation counts specifically controlled for sampling effort biases among host taxa. Our article illustrates a comparative approach that could easily be repeated for other host and parasite taxa. As more information on broadly sampled parasite phylogenies becomes available, more powerful methods for host-parasite evolution can be implemented, as well as approaches based on host and

parasite characteristics that may influence diversification rates.

The relative diversity of evolutionary lineages increases when extinction rates decline or speciation rates increase. In subsequent analyses, we found no support for a mechanism in which parasites increase host diversity through effects on speciation rates as mediated by sexual selection. A commonly cited hypothesis is that parasites play an important role in sexual selection and that species with greater levels of parasitism experience more intense sexual selection on traits that signal resistance to or tolerance of high parasite burdens (Hamilton and Zuk 1982; Andersson 1994; Moore and Wilson 2002). The link between sexual selection and host diversity arises because sexual selection has been proposed as an "engine" of speciation (Lande 1981; Owens et al. 1999; Katzourakis et al. 2001; Turelli et al. 2001). However, our analyses did not support a link between parasite species richness and measures of sexual selection in primates. Moreover, primate lineages with evidence of more intense sexual selection were not associated with increased evolutionary diversity. Thus, if parasites drive host diversification by providing pressure for evolutionary branching, this probably occurs via mechanisms other than sexual selection.

We also investigated a mechanism that reversed the direction of causality, specifically by proposing that more rapidly expanding primate lineages provide greater opportunities for parasite accumulation and diversification, as compared with lower diversity host clades. Hosts in rapidly diversifying lineages might be more likely to overlap geographically with close relatives, with these sympatric populations providing an effectively larger total population of hosts for generalist parasites that utilize a wide range of host species. Increased overlap also could increase opportunities for specialist parasites to host shift, followed

Table 4: Association between host geographic range overlap and parasite species richness

	All parasites	Helminths	Protozoa	Viruses
All parasites:				
Number overlapping	1.81*	.36	1.70	2.57**
Total overlapping area	1.79*	-.09	1.25	3.31***
Specialists:				
Number overlapping	2.98**	1.47 [†]	3.00**	3.57***
Total overlapping area	3.12**	1.14	3.19**	3.20**
Generalists:				
Number overlapping	1.13	-.27	.87	2.09*
Total overlapping area	.93	-1.00	-.02	3.21**

Note: Table shows *t* statistics, with sign of the *t* statistic corresponding to the slope of the relationship. Results are from analyses of independent contrasts using residual parasite species richness. All *P* values from directed tests using 108 contrasts.

[†] *P* < .10.

* *P* < .05.

** *P* < .01.

*** *P* < .001.

by rapid evolution in novel hosts (e.g., Ricklefs and Fallon 2001; Zietara and Lumme 2002). In support of this hypothesized mechanism, we found that primate host sympatry correlated positively with parasite species richness, particularly for viruses, protozoa, and specialist parasites (table 4). However, when geographic range size was included as an additional independent variable, measures of host sympatry were significant predictors of parasite species richness only for viruses. Moreover, measures of host evolutionary diversity showed no association with measures of host overlap (table 5). These tests examined overlap among primate hosts on the basis of the assumption that more closely related hosts provide the most likely reservoirs for parasites (e.g., Pfennig 2000; Perlman and Jaenike 2003), but by doing so, we might have missed nonprimate sources of infection. For example, some primate pinworms share a most common recent ancestor with species of pinworms that infect rodents (Hugot 1999). Geographic range overlap with other mammalian hosts may therefore be informative in future tests of the hypothesis that host diversification and parasite richness are correlated.

In context of host-pathogen coevolution, the Red Queen hypothesis (Van Valen 1973) is important because it predicts that species interactions will influence evolutionary dynamics in the absence of any changes in the abiotic environment. Evidence for primate diversity increasing with parasite richness is consistent with the Red Queen hypothesis and suggests that antagonistic coevolution might play a role in increasing both host and parasite diversity. The question is whether there is a tight causal mechanism, or “interconnectedness” (Eldredge 1989), between primate speciation rates and parasite numbers ir-

respective of other factors. The tests we conducted point to no other significant correlates of host diversification rates in primates, with the exception of population density in some tests (see also Isaac et al., in press). However, we cannot rule out other biogeographic factors (e.g., sympatry with other mammalian hosts) as increasing both primate diversity and parasitism within primate clades. It is also possible that a more general arms race occurs between hosts and parasites. For example, Buckling and Rainey (2002) showed experimentally that phage-imposed selection for resistance led to rapid increases in allopatric divergence among bacterial populations, in part because resistance-conferring traits were linked to different bacterial characteristics in different environments. A similar

Table 5: Tests of association between host geographic range overlap and host diversity

Host sympatry measure and host diversity measure	<i>t_s</i>
Number overlapping:	
RRD	.43
PDI	.22
Total overlap:	
RRD	.52
PDI	.26

Note: Table shows *t* statistics, with sign of the *t* statistic corresponding to the slope of the relationship in bivariate regression analyses. Removal of four to five outliers produced similar results. No results were statistically significant (*n* = 73 contrasts). *r_s* = -0.03 for number overlapping and -0.01 for total overlap, both nonsignificant. Including geographic range had no effect on the outcome of the statistical tests. RRD = relative rate difference; PDI = proportional dominance index.

mechanism in primates would be difficult to detect without comparative estimates of host resistance phenotypes or genotypes in parasite-rich versus parasite-poor taxa.

Our results provided no support for the idea that a high diversity of infectious diseases increases primate extinction risk (and reduces host phylogenetic diversity). This is important because parasites are commonly viewed as causing declines in primates and other host taxa, which may then lead to extinction (MacPhee and Marx 1997). For example, pathogens have been implicated in dramatic declines of black-footed ferrets, lions, wild dogs, Hawaiian forest birds, a range of amphibians, and harbor seals (reviewed in Dobson and Grenfell 1995; Daszak et al. 2000; Lafferty and Gerber 2002). Nearly all recent outbreaks of parasites and infectious diseases leading to wildlife declines in recent years have been directly or indirectly linked with human activities, including host crowding leading to stress and increased contact rates among wildlife species, interactions between wildlife and domesticated animals, pollution, climate change, and the direct introduction of novel pathogens (Harvell et al. 1999; Altizer et al. 2000; Daszak et al. 2000; Dobson and Foufopoulos 2001; Lafferty and Gerber 2002). Emerging diseases or pathogens introduced or exacerbated by human activity have been shown to decimate primate populations, as evidenced by recent examples of Ebola virus outbreaks in gorillas and chimpanzees (Walsh et al. 2003). The frequency of such epidemics (and resulting host population declines) probably has increased in recent years because of human activities that cause novel pathogen introductions or host shifts (Dobson and Foufopoulos 2001). Therefore, although our results did not show a generally negative effect of parasites on host diversity, it is possible that future increases in parasite-mediated host extinctions could lead to such a negative effect.

One final explanation for lower parasite diversity among species-poor primate lineages is that higher extinction rates in declining primate lineages are associated with reductions in parasite diversity; in other words, parasites are lost as host populations decline, causing parasites to go extinct before their hosts. Thus, while we have focused on the factors involving increased speciation that may increase parasite species richness in more diverse hosts, increased host extinction rates could reduce parasite richness in less diverse hosts when parasites, particularly specialists, become extinct before their hosts. Testing this hypothesized mechanism requires estimates of host extinction rates and should provide further insights into causal mechanisms underlying patterns of host and parasite diversity in natural systems.

Increasingly, successful conservation programs are recognized as those that maintain populations with intact evolutionary processes (Crandall et al. 2000; Stockwell et

al. 2003). The importance of species interactions and geographically structured populations has been highlighted as a major factor promoting both genetic and species diversity in natural communities (Thompson 1999). Although the risks they pose to threatened taxa are apparent, parasites and infectious diseases can play an important role in maintaining host genetic and evolutionary diversity (reviewed in Altizer et al. 2003). Our study also highlights the possibility that diverse host lineages provide new evolutionary opportunities for diverse assemblages of parasites. Given that many parasites themselves have become extinct along with their specific hosts (Gompper and Williams 1998), it becomes important to consider the impacts of mammalian extinctions on the collective biodiversity represented by their parasites.

Acknowledgments

We thank N. Isaac and M. Sanderson for helpful discussion and A. Cunningham, A. Pedersen, M. Poss, and N. Vitone for assistance with coding host specificity for the parasites in our data set. D. Harvel, H. McCallum, and an anonymous reviewer provided helpful comments on the manuscript. D. Chivers provided assistance with the dichromatism data set, and C. Leone assisted in managing references and preparing the manuscript. This research was supported by funding from the National Science Foundation (NSF; grants DEB-0212096 to C.L.N. and S.A. and DEB-0129009 to J.L.G.) and the Center for Applied Biodiversity Science at Conservation International. This work was also conducted as part of the Infectious Disease and Host Behavior Working Group supported by the National Center for Ecological Analysis and Synthesis, funded by the NSF, the University of California, and the Santa Barbara campus.

Literature Cited

- Abrams, P. A. 2000. Character shifts of prey species that share predators. *American Naturalist* 156(suppl.):S46–S61.
- Agapow, P. M., and N. J. B. Isaac. 2002. MacroCAIC: revealing correlates of species richness by comparative analysis. *Diversity and Distributions* 8:41–43.
- Altizer, S., J. Foufopoulos, and A. Gager. 2000. Conservation and disease. Pages 109–126 *in* S. Levin, ed. *Encyclopedia of biodiversity*. Academic Press, San Diego, Calif.
- Altizer, S., D. Harvell, and E. Friedle. 2003. Rapid evolutionary dynamics and disease threats to biodiversity. *Trends in Ecology & Evolution* 18:589–596.
- Altizer, S. M. 2001. Migratory behaviour and host-parasite co-evolution in natural populations of monarch but-

- terflies infected with a protozoan parasite. *Evolutionary Ecology Research* 3:1–22.
- Anderson, R. M., and R. M. May. 1991. *Infectious diseases of humans: dynamics and control*. Oxford University Press, Oxford.
- Andersson, M. 1994. *Sexual selection*. Princeton University Press, Princeton, N.J.
- Arnqvist, G., M. Edvardsson, U. Friberg, and T. Nilsson. 2000. Sexual conflict promotes speciation in insects. *Proceedings of the National Academy of Sciences of the USA* 97:10460–10464.
- Barracough, T. G., and S. Nee. 2001. Phylogenetics and speciation. *Trends in Ecology & Evolution* 16:391–399.
- Barracough, T. G., P. Harvey, and S. Nee. 1995. Sexual selection and taxonomic diversity in passerine birds. *Proceedings of the Royal Society of London B* 259:211–215.
- Bawa, K. S. 1995. Pollination, seed dispersal and diversification of angiosperms. *Trends in Ecology & Evolution* 8:311–312.
- Berenbaum, M. R., and A. R. Zangerl. 1998. Chemical phenotype matching between a plant and its insect herbivore. *Proceedings of the National Academy of Sciences of the USA* 95:13743–13748.
- Brack, M. 1987. *Agents transmissible from simians to man*. Springer, Berlin.
- Brown, J. H. 1995. *Macroecology*. University of Chicago Press, Chicago.
- Buckling, A., and P. B. Rainey. 2002. The role of parasites in sympatric and allopatric host diversification. *Nature* 420:496–499.
- Burdon, J. J., and P. H. Thrall. 1999. Spatial and temporal patterns in coevolving plant and pathogen associations. *American Naturalist* 153(suppl.):S15–S33.
- Coombes, C. 2001. *Parasitism: the ecology and evolution of intimate interactions*. University of Chicago Press, Chicago.
- Corbet, G. B., and J. E. Hill. 1991. *A world list of mammalian species*. Oxford University Press, Oxford.
- Crandall, K. A., O. R. P. Bininda-Emonds, G. M. Mace, and R. K. Wayne. 2000. Considering evolutionary processes in conservation biology. *Trends in Ecology & Evolution* 15:290–295.
- Daszak, P., A. A. Cunningham, and A. D. Hyatt. 2000. Emerging infectious diseases of wildlife: threats to biodiversity and human health. *Science* 287:443–449.
- Desdevises, Y., S. Morand, and G. Oliver. 2001. Linking specialisation to diversification in the Diplectanidae Bychowsky, 1957 (Monogenea, Platyhelminthes). *Parasitology Research* 87:223–230.
- Dixon, A. F. 1998. *Primate sexuality*. Oxford University Press, Oxford.
- Dobson, A., and J. Foufopoulos. 2001. Emerging infectious pathogens of wildlife. *Philosophical Transactions of the Royal Society of London B* 356:1001–1012.
- Dobson, A., and B. Grenfell. 1995. *Ecology of infectious disease in natural populations*. Cambridge University Press, Cambridge.
- Doebeli, M., and U. Dieckmann. 2000. Evolutionary branching and sympatric speciation caused by different types of ecological interactions. *American Naturalist* 156(suppl.):S77–S101.
- Dybdahl, M. F., and C. M. Lively. 1998. Host parasite coevolution: evidence for rare advantage and time-lagged selection in a natural population. *Evolution* 52:1057–1066.
- Ehrlich, P. R., and P. H. Raven. 1964. Butterflies and plants: a study in coevolution. *Evolution* 18:586–608.
- Eldredge, N. 1989. *Macroevolutionary dynamics: species, niches, and adaptive peaks*. McGraw-Hill, New York.
- Farrell, B. 1998. Inordinate fondness explained: why are there so many beetles? *Science* 281:555–559.
- Felsenstein, J. 1985. Phylogenies and the comparative method. *American Naturalist* 125:1–15.
- Fiennes, R. 1972. Pathology of simian primates. II. Infectious and parasitic diseases. Karger, London.
- Gage, M. J. G., G. A. Parker, S. Nylin, and C. Wiklund. 2002. Sexual selection and speciation in mammals, butterflies and spiders. *Proceedings of the Royal Society of London B* 269:2309–2316.
- Gardezi, T., and J. da Silva. 1999. Diversity in relation to body size in mammals: a comparative study. *American Naturalist* 153:110–123.
- Garland, T. J., P. H. Harvey, and A. R. Ives. 1992. Procedures for the analysis of comparative data using phylogenetically independent contrasts. *Systematic Biology* 4:18–32.
- Gavrilets, S. 2000. Rapid evolution of reproductive barriers driven by sexual conflict. *Nature* 403:886–889.
- Gittleman, J. L., and A. Purvis. 1998. Body size and species richness in carnivores and primates. *Proceedings of the Royal Society of London B* 265:113–119.
- Gompper, M. E., and E. S. Williams. 1998. Parasite conservation and the black-footed ferret recovery program. *Conservation Biology* 12:730–732.
- Gregory, R. D. 1990. Parasites and host geographic range as illustrated by waterfowl. *Functional Ecology* 4:645–654.
- Gregory, R. D., A. E. Keymer, and P. H. Harvey. 1996. Helminth parasite richness among vertebrates. *Biodiversity and Conservation* 5:985–997.
- Groves, C. P. 2001. *Primate taxonomy*. Smithsonian Institution, Washington, D.C.
- Hafner, M. S., and R. D. M. Page. 1995. Molecular phylogenies and host-parasite cospeciation: gophers and lice

- as a model system. *Philosophical Transactions of the Royal Society of London B* 349:77–83.
- Hamilton, W. D., and M. Zuk. 1982. Heritable true fitness and bright birds: a role for parasites? *Science* 218:384–387.
- Harvell, C. D., K. Kim, J. M. Burkholder, R. R. Colwell, P. R. Epstein, D. J. Grimes, E. E. Hofmann, et al. 1999. Emerging marine diseases: climate links and anthropogenic factors. *Science* 285:1505–1510.
- Harvey, P. H., and M. D. Pagel. 1991. *The comparative method in evolutionary biology*. Oxford University Press, Oxford.
- Hey, J. 1992. Using phylogenetic trees to study speciation and extinction. *Evolution* 46:627–640.
- Hugot, J. P. 1999. Primates and their pinworm parasites: the Cameron hypothesis revisited. *Systematic Biology* 48:523–546.
- Isaac, N. J. B., P.-M. Agapow, P. H. Harvey, and A. Purvis. 2003. Phylogenetically nested comparisons for testing correlates of species richness: a simulation study of continuous variables. *Evolution* 57:18–26.
- Isaac, N. J. B., K. E. Jones, J. L. Gittleman, and A. Purvis. In press. Correlates of species richness in mammals: body size, life history, energetics and ecology. *American Naturalist*.
- Johnson, K. P., and D. H. Clayton. 2002. Coevolutionary history of ecological replicates: comparing phylogenies of wing and body lice to Columbiform hosts. Pages 262–286 in R. Page, ed. *Tangled trees: phylogenies, cospeciation and coevolution*. University of Chicago Press, Chicago.
- Kaltz, O., and J. A. Shykoff. 1998. Local adaptation in host-parasite systems. *Heredity* 81:361–370.
- Katzourakis, A., A. Purvis, S. Azmeh, G. Rotheray, and F. Gilbert. 2001. Macroevolution of hoverflies (Diptera: Syrphidae): the effect of using higher-level taxa in studies of biodiversity, and correlates of species richness. *Journal of Evolutionary Biology* 14:219–227.
- Lafferty, K. D., and L. R. Gerber. 2002. Good medicine for conservation biology: the intersection of epidemiology and conservation theory. *Conservation Biology* 16:593–604.
- Lande, R. 1981. Models of speciation by sexual selection on polygenic traits. *Proceedings of the National Academy of Sciences of the USA* 78:3721–3725.
- Lively, C. M., and V. Apanius. 1995. Genetic diversity in host-parasite interactions. Pages 421–449 in B. T. Grenfell and A. P. Dobson, eds. *Ecology of infectious diseases in natural populations*. Cambridge University Press, Cambridge.
- MacPhee, R. D. E., and P. A. Marx. 1997. The 40,000-year plague. Pages 169–217 in S. M. Goodman and B. D. Patterson, eds. *Natural change and human impact in Madagascar*. Smithsonian Institution, Washington, D.C.
- Magallon, S., and M. Sanderson. 2001. Absolute diversification rates in angiosperm clades. *Evolution* 55:1762–1780.
- Mayhew, P. J. 2002. Shifts in hexapod diversification and what Haldane could have said. *Proceedings of the Royal Society of London B* 269:969–974.
- Mitter, C., B. Farrell, and D. J. Futuyma. 1991. Phylogenetic studies of insect-plant interactions: insights into the genesis of diversity. *Trends in Ecology & Evolution* 6:290–293.
- Moore, S. L., and K. Wilson. 2002. Parasites as a viability cost of sexual selection in natural populations of mammals. *Science* 297:2015–2018.
- Morand, S. 2000. Wormy world: comparative tests of theoretical hypotheses on parasite species richness. Pages 63–79 in R. Poulin, S. Morand, and A. Skorping, eds. *Evolutionary biology of host-parasite relationships*. Elsevier, Amsterdam.
- Morand, S., and P. H. Harvey. 2000. Mammalian metabolism, longevity and parasite species richness. *Proceedings of the Royal Society of London B* 267:1999–2003.
- Morand, S., M. S. Hafner, R. D. M. Page, and D. L. Reed. 2000. Comparative body size relationships in pocket gophers and their chewing lice. *Biological Journal of the Linnean Society* 70:239–249.
- Morrow, E. H., T. E. Pitcher, and G. Arnqvist. 2003. No evidence that sexual selection is an “engine of speciation” in birds. *Ecology Letters* 6:228–234.
- Nee, S. 2001. Inferring speciation rates from phylogenies. *Evolution* 55:661–668.
- Nee, S., A. O. Mooers, and P. H. Harvey. 1992. Tempo and mode of evolution revealed from molecular phylogenies. *Proceedings of the National Academy of Sciences of the USA* 89:8322–8326.
- Nee, S., E. C. Holmes, R. M. May, and P. H. Harvey. 1994a. Extinction rates can be estimated from molecular phylogenies. *Philosophical Transactions of the Royal Society of London B* 344:77–82.
- Nee, S., R. M. May, and P. H. Harvey. 1994b. The reconstructed evolutionary process. *Philosophical Transactions of the Royal Society of London B* 344:305–311.
- Nunn, C. L. 1999. The evolution of exaggerated sexual swellings in primates and the graded signal hypothesis. *Animal Behaviour* 58:229–246.
- Nunn, C. L., and R. A. Barton. 2000. Allometric slopes and independent contrasts: a comparative test of Kleiber’s law in primate ranging patterns. *American Naturalist* 156:519–533.
- . 2001. Comparative methods for studying primate adaptation and allometry. *Evolutionary Anthropology* 10:81–98.

- Nunn, C. L., and C. P. van Schaik. 2001. Reconstructing the behavioral ecology of extinct primates. Pages 159–216 in J. M. Plavcan, R. F. Kay, W. L. Jungers, and C. P. van Schaik, eds. *Reconstructing behavior in the fossil record*. Kluwer Academic/Plenum, New York.
- Nunn, C. L., S. Altizer, K. E. Jones, and W. Sechrest. 2003. Comparative tests of parasite species richness in primates. *American Naturalist* 162:597–614.
- Orme, C. D. L., N. J. B. Isaac, and A. Purvis. 2002a. Are most species small? not within species-level phylogenies. *Proceedings of the Royal Society of London B* 269:1279–1287.
- Orme, C. D. L., D. L. J. Quicke, J. M. Cook, and A. Purvis. 2002b. Body size does not predict species richness among the metazoan phyla. *Journal of Evolutionary Biology* 15:235–247.
- Owens, I. P. F., P. M. Bennett, and P. H. Harvey. 1999. Species richness among birds: body size, life history, sexual selection or ecology? *Proceedings of the Royal Society of London B* 266:933–939.
- Panhuis, T. M., R. Butlin, M. Zuk, and T. Tregenza. 2001. Sexual selection and speciation. *Trends in Ecology & Evolution* 16:364–371.
- Paradis, E. 1997. Assessing temporal variations in diversification rates from phylogenies: estimation and hypothesis testing. *Proceedings of the Royal Society of London B* 264:1141–1147.
- . 1998. Detecting shifts in diversification rates without fossils. *American Naturalist* 152:176–187.
- Parker, G. A., and L. Partridge. 1998. Sexual conflict and speciation. *Philosophical Transactions of the Royal Society of London B* 353:261–274.
- Pellmyr, O. 1992. Evolution of insect pollination and angiosperm diversification. *Trends in Ecology & Evolution* 7:46–49.
- Percy, D., R. Page, and Q. Cronk. 2004. Plant-insect interactions: double-dating associated insect and plant lineages reveals asynchronous radiations. *Systematic Biology* 53:120–127.
- Perlman, S. J., and J. Jaenike. 2003. Infection success in novel hosts: an experimental and phylogenetic study of *Drosophila*-parasitic nematodes. *Evolution* 57:544–557.
- Peters, R. H. 1983. *The ecological implications of body size*. Cambridge University Press, Cambridge.
- Pfennig, D. W. 2000. Effect of predator-prey phylogenetic similarity on the fitness consequences of predation: a trade-off between nutrition and disease? *American Naturalist* 155:335–345.
- Plavcan, J. M., and C. P. van Schaik. 1992. Intrasexual competition and canine dimorphism in anthropoid primates. *American Journal of Physical Anthropology* 87:461–477.
- Poulin, R. 1995. Phylogeny, ecology, and the richness of parasite communities in vertebrates. *Ecological Monographs* 65:283–302.
- . 2002. The evolution of monogenean diversity. *International Journal for Parasitology* 32:245–254.
- Purvis, A. 1995. A composite estimate of primate phylogeny. *Philosophical Transactions of the Royal Society of London B* 348:405–421.
- . 1996. Using interspecies phylogenies to test macroevolutionary hypotheses. Pages 153–168 in P. H. Harvey, A. J. Leigh Brown, J. Maynard Smith, and S. Nee, eds. *New uses for new phylogenies*. Oxford University Press, Oxford.
- Purvis, A., and A. Hector. 2000. Getting the measure of biodiversity. *Nature* 405:212–219.
- Purvis, A., and A. Rambaut. 1995. Comparative analysis by independent contrasts (CAIC): an Apple Macintosh application for analysing comparative data. *Computer Applications in the Biosciences* 11:247–251.
- Purvis, A., S. Nee, and P. H. Harvey. 1995. Macroevolutionary inferences from primate phylogeny. *Proceedings of the Royal Society of London B* 260:329–333.
- Raibaut, A., C. Combes, and F. Benoit. 1998. Analysis of the parasitic copepod species richness among Mediterranean fish. *Journal of Marine Systems* 15:185–206.
- Read, A. 1988. Sexual selection and the role of parasites. *Trends in Ecology & Evolution* 3:97–102.
- Rice, W. R., and S. D. Gaines. 1994. Heads I win, tails you lose: testing directional alternative hypotheses in ecological and evolutionary research. *Trends in Ecology & Evolution* 9:235–237.
- Rice, W. R., and B. Holland. 1997. The enemies within: intergenomic conflict, interlocus contest evolution (ICE), and the intraspecific red queen. *Behavioral Ecology and Sociobiology* 41:1–10.
- Ricklefs, R., and S. Fallon. 2001. Diversification and host switching in avian malaria parasites. *Proceedings of the Royal Society of London B* 269:885–892.
- Ross, C., and K. E. Jones. 1999. Socioecology and the evolution of primate reproductive rates. Pages 73–110 in P. C. Lee, ed. *Comparative primate socioecology*. Cambridge University Press, Cambridge.
- Rowe, N. 1996. *The pictorial guide to the living primates*. Pogonias, East Hampton, N.Y.
- Roy, B. A. 2001. Patterns of association between crucifers and their flower mimic pathogens: host jumps are more common than coevolution or cospeciation. *Evolution* 55:41–53.
- Sanderson, M. J., and M. J. Donoghue. 1996. Reconstructing shifts in diversification rates on phylogenetic trees. *Trends in Ecology & Evolution* 11:15–20.
- Sechrest, W., T. M. Brooks, G. A. B. da Fonseca, W. R. Konstant, R. A. Mittermeier, A. Purvis, A. B. Rylands,

- et al. 2002. Hotspots and the conservation of evolutionary history. *Proceedings of the National Academy of Sciences of the USA* 99:2067–2071.
- Smith, R. J., and W. L. Jungers. 1997. Body mass in comparative primatology. *Journal of Human Evolution* 32: 523–559.
- Stockwell, C. A., A. P. Hendry, and M. T. Kinnison. 2003. Contemporary evolution meets conservation biology. *Trends in Ecology & Evolution* 18:94–101.
- Taylor, L. H., S. M. Latham, and M. E. J. Woolhouse. 2001. Risk factors for human disease emergence. *Philosophical Transactions of the Royal Society of London B* 356:983–989.
- Thompson, J. N. 1999. Specific hypotheses on the geographic mosaic of coevolution. *American Naturalist* 153(suppl.):S1–S14.
- Turelli, M., N. H. Barton, and J. A. Coyne. 2001. Theory and speciation. *Trends in Ecology & Evolution* 16:330–343.
- Van Valen, L. 1973. A new evolutionary law. *Evolutionary Theory* 1:1–30.
- Walsh, P. D., K. A. Abernethy, M. Bermejo, R. Beyersk, P. De Wachter, M. E. Akou, B. Huljbreghis, et al. 2003. Catastrophic ape decline in western equatorial Africa. *Nature* 422:611–614.
- Zietara, M., and J. Lumme. 2002. Speciation by host switch and adaptive radiation in a fish parasite genus *Gyrodactylus* (Monogenea, Gyrodactylidae). *Evolution* 56: 2445–2458.