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RESEARCH ARTICLE

The changing ecology of primate parasites: Insights from wild-captive comparisons

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Abstract

Host movements, including migrations or range expansions, are known to influence parasite communities. Transitions to captivity—a rarely studied yet widespread human-driven host movement—can also change parasite communities, in some cases leading to pathogen spillover among wildlife species, or between wildlife and human hosts. We compared parasite species richness between wild and captive populations of 22 primate species, including macro- (helminths and arthropods) and micro-parasites (viruses, protozoa, bacteria, and fungi). We predicted that captive primates would have only a subset of their native parasite community, and would possess fewer parasites with complex life cycles requiring intermediate hosts or vectors. We further predicted that captive primates would have parasites transmitted by close contact and environmentally—including those shared with humans and other animals, such as commensals and pests. We found that the composition of primate parasite communities shifted in captive populations, especially because of turnover (parasites detected in captivity but not reported in the wild), but with some evidence of nestedness (holdovers from the wild). Because of the high degree of turnover, we found no significant difference in overall parasite richness between captive and wild primates. Vector-borne parasites were less likely to be found in captivity, whereas parasites transmitted through either close or non-close contact, including through fecal-oral transmission, were more likely to be newly detected in captivity. These findings identify parasites that require monitoring in captivity and raise concerns about the introduction of novel parasites to potentially susceptible wildlife populations during reintroduction programs.

KEYWORDS

host-parasite interactions, nestedness, parasite species richness, turnover, zoonosis

1 | INTRODUCTION

When moving into a new habitat, hosts can lose some parasite species, retain others, and acquire new ones from novel environments or hosts. Transitions to new environments can occur through multiple mechanisms, including dispersal and migration (e.g., Altizer, Bartel, & Han, 2011), the unintentional anthropogenic introduction of plants and animals, and by intentional translocation of wildlife by humans (Chomel, Belotto, & Meslin, 2007; Snyder et al., 1996; Wolfe et al., 1998). Capturing wild animals and moving them into captivity is a form of translocation that occurs for a variety of reasons, including pet and wildlife trade, to acquire animals for captive research, and for conservation purposes (Mittermeier, Konstant, & Mast, 1994; Smith et al., 2009).

An ecological understanding of how parasites of captive populations differ from their wild counterparts is important for investigating fundamental questions in wildlife disease ecology, and also for evaluating health outcomes of captivity to inform captive breeding programs and efforts to reintroduce captive individuals into the wild (Cunningham, 1996; Hudson, Dobson, & Lafferty, 2006; Lyles & Dobson, 1993). Here, we use the ecological definition of a parasite as any infectious agent that lives in or on a host, at some cost to that host, including micro-parasites (viruses, bacteria, fungi, and protozoa) and macro-parasites (helminths and arthropods). The transition of hosts from the wild to captivity has important parallels with parasite dynamics observed in migratory animals and in exotic species introductions, both of which can reduce infection risk (Altizer et al., 2011; Torchin & Mitchell, 2004; Torchin, Lafferty, Dobson, McKenzie, & Kuris, 2003). Specifically, migratory animals can escape from parasites in their breeding range as they move to their winter range, and heavily infected individuals may die during strenuous migrations, lowering parasite prevalence (Altizer et al., 2011; Altizer, Hobson, Davis, De Roode, & Wassenaar, 2015). Similarly, when hosts are introduced into new environments, they often lose parasite species present in their native range and experience lower parasite burdens, which facilitates their invasion (Mitchell & Power, 2003; Torchin et al., 2003).

Wildlife might lose parasites during three stages of transition from natural habitats to captivity: (a) collection from the wild, because captured individuals likely harbor only a subset of parasites from the original wild population (Torchin et al., 2003), (b) transport to captivity, because the stress of transport and acute infections might cause some infected animals to die (e.g., Kock, Mihok, Wambua, Mwanzia, & Saigawa, 1999; Lafferty & Holt, 2003; Scope, Filip, Gabler, & Resch, 2002), and (c) establishment in captivity, where parasites from the native range might be lost because of housing conditions that are not conducive to pathogen transmission. Specialized parasites capable of infecting only one or a few host species might be more likely to be lost in captivity (Lyles & Dobson, 1993), whereas generalist parasites that can infect a broad range of host species might tend to persist in captive environments that house multiple species. Similarly, captive animals might disproportionately lose parasites with complex life cycles if vectors or intermediate hosts necessary for transmission are rare or absent from captive settings (Torchin et al., 2003). Finally, captive animals often receive medical treatment to reduce parasite loads, such as with anti-helminthic drugs, antibiotics, or vaccines (Munene et al., 1998), potentially resulting in further declines in parasite diversity.

Alongside the loss of parasites from the native wild environment, captivity could facilitate the acquisition of novel parasites. Stressful conditions in captive settings might suppress host immunity, leaving captive hosts susceptible to new infections (Fowler, 1986; Lyles & Dobson, 1993; Mason, 2010). Captive animals might also gain parasites when their housing facilitates close proximity to other host species not encountered in the wild, including domesticated species and humans (Lyles & Dobson, 1993). This is particularly important if two or more host species are phylogenetically similar, which has been shown to predict parasite sharing in wild populations (Cooper et al., 2012; Gilbert & Webb, 2007). Captive animals might also acquire parasites through exposure to new intermediate hosts or vectors, especially when housed outdoors (Pung, Spratt, Clark, Norton, & Carter, 1998; Ratterree et al., 2003). If captive animals are re-introduced, they have the potential to transmit novel pathogens acquired in captivity to wild individuals (Hatcher, Dick, & Dunn, 2012; Lyles & Dobson, 1993), posing risks to wild populations.

Primates are an especially important host group in which to consider parasite differences between wild and captive environments. The risk of parasite spillover from captive nonhuman primates to humans is substantial in zoos, laboratories, and rescue centers. In this context, captive primates harbor many different parasites (Brack, 2012; Johnson-Delaney, 2009; Lyles & Dobson, 1993; McPherson, 2013), some of which can infect humans and other animals (Ballou, 1993; Gyuranecz et al., 2009; Jones-Engel et al., 2004; McPherson, 2013; Weigler, 1992). For example, research indicates that captive primates might be responsible for *Leptospira* and simian foamy virus infections among zookeepers (Romero, Astudillo, Sánchez, González, & Varela, 2011; Sandstrom et al., 2000). Similarly, monkeys and an employee tested seropositive for Reston Ebola virus at a quarantine facility in Virginia, and a young lab worker died tragically after acquiring herpes B virus from a macaque at a primate research center (CDC, 1998; Miranda et al., 1999). Thus, understanding the ecology of captive primate parasites is important to both human and nonhuman animal health.

We compared parasite diversity between wild and captive populations using a new database of 22 captive primate species that have been sampled well for parasites in wild populations. To investigate population-level differences in exposure and susceptibility to parasites, we compared parasite species richness (PSR), or the total number of parasite species per host (Bordes & Morand, 2009; Bordes & Morand, 2011). On the basis of findings for invasive species (Mitchell & Power, 2003; Torchin & Mitchell, 2004; Torchin et al., 2003), we predicted that captive primates would have lower PSR than their wild counterparts. We investigated changes in the composition of parasite communities in wild and captive primates using beta diversity (Koleff, Gaston, & Lennon, 2003). We quantified both nestedness and turnover of parasite communities (Baselga, 2010), where nestedness captures the degree to which parasites in the new environment are a subset of the original parasite community (Figure 1, Patterson, 1987) and turnover measures the addition of new parasite species (Figure 1). We predicted that parasite communities in captive primates would be a nested subset of the wild parasite community, with notable absences including native range parasites that require intermediate hosts or vectors. We also predicted that primates would acquire new parasites in captivity, especially parasites transmitted by close or non-close contact, for which transmission opportunities might exist in captive settings, as well as parasites known to infect humans.

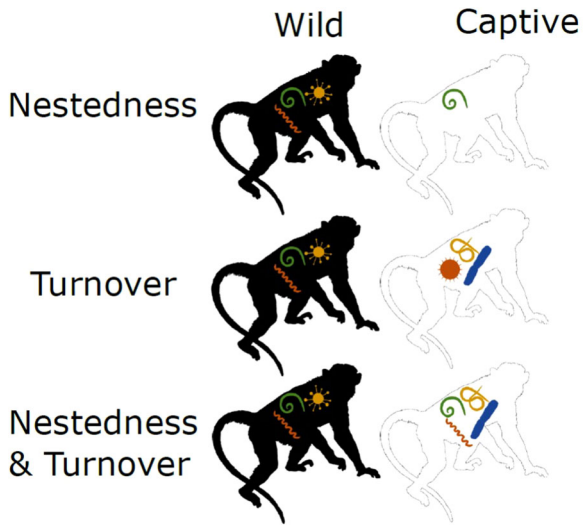

FIGURE 1 Schematic demonstrating the differences between nestedness and turnover components of beta diversity. Nestedness results when parasites in wild hosts are not present in captivity. Turnover results when parasite species are different between wild and captive hosts. Both nestedness and turnover can occur in a host to varying degrees, and make up the beta diversity between the wild and captive environments

2 | MATERIALS AND METHODS

2.1 | Data collection

We collected captive nonhuman primate parasite occurrence data from the primary literature using studies published between 1920 and 2012. We focused on 22 primate species representing the four major primate lineages on the basis of an initial list of primate species that were sampled well for parasites in the wild, and that were known to be housed in captive settings (Table S1). Primate species' scientific names, including synonyms, were on the basis of well-accepted mammal taxonomy (Wilson & Reeder, 2005). The data on parasites from captive primates were collected by systematically searching the Web of Science (https://webofknowledge.com/), National Agricultural Library (AGRICOLA, http://agricola.nal.usda.gov/), and PubMed (http://www.ncbi.nlm.nih.gov/pubmed) databases. The search strategy involved the use of general parasite search terms with host scientific name (i.e., "species name" AND (parasit* OR pathogen* OR disease OR infect* OR arthropod OR bacteria OR helminth OR fungi OR protozoa OR virus OR vector)). Captive settings included zoological parks, wildlife rehabilitation centers, animals kept as pets, and captive colonies used for behavioral or biomedical research. We removed all cases of experimental infections and challenges, retaining only reports of naturally-occurring infections in captive settings, resulting in data from 241 sources. Comparable data on parasite infection from wild populations of the same set of primate species were obtained from the Global Mammal Parasite Database (GMPD; Nunn & Altizer, 2005; Stephens et al., 2017). The data from the GMPD included 359 sources, are publicly available (https://parasites.nunn-lab.org/), and have been used in numerous analyses of parasitism in wild primates (e.g., Altizer, Nunn, & Lindenfors, 2007; Altizer et al., 2003; Cooper et al., 2012; Dallas, Huang, Nunn, Park, & Drake, 2017; Davies & Pedersen, 2008; Nunn et al., 2004; Park et al., 2018). For each host, parasites were only included if they were identified to the genus level at least. To avoid potential double counting, parasites that were not identified to the species level were omitted if a congener with a species epithet was present.

We recorded the transmission strategy of each parasite into five non-mutually exclusive categories (Pedersen, Altizer, Poss, Cunningham, & Nunn, 2005): Close contact, non-close contact, vector-borne, sexually transmitted, and intermediate hosts. Parasites categorized as spread by close contact were communicable by close proximity or direct contact, such as biting, scratching, mating contact, or other touching. Sexually transmitted parasites were a subset of close-contact transmitted parasites that are spread during copulation. Non-close contact involved transmission via fomites or contact with contaminated soil or water (which could include fecal-oral transfer). Vector-borne parasites were those spread via biting arthropods (ticks, mites, fleas, flies, and other invertebrates). Parasites transmitted by intermediate hosts have complex life cycles typically characterized by trophic transmission, and primates could serve as either intermediate or final hosts, or dead-end hosts. Parasites could exhibit more than one transmission mode (e.g., sexually transmitted parasites may also be transmitted by close contact, and many parasites transmitted by close contact can also be transmitted by non-close contact). We also recorded whether the parasite species were known to infect humans and/or were zoonotic on the basis of the known human parasites (Center for Disease Control, www.cdc.gov, Taylor, Latham, & Mark, 2001).

2.2 | Statistical analyses

PSR estimates can be influenced by sampling effort, defined as the degree to which each host species or population has been studied for parasites (Altizer et al., 2003; Poulin, 1998). We accounted for variation in sampling effort between wild and captive hosts using three approaches. First, we compared the number of parasite studies available for each primate host species in the wild and under captive conditions. We used the smaller number of studies to randomly subsample the condition with the larger number of studies, and used rarefaction to calculate the PSR expected if sampling efforts were equal between wild and captive conditions (e.g., Colwell et al., 2012). To obtain standard errors on estimates of PSR and quantify intraspecific variation because of variation among studies, we bootstrapped the studies 1,000 times. Second, we also rarefied both conditions to one less study than the total number of studies using 1,000 bootstrap replicates to obtain PSR and the standard deviation in PSR. We used the *specaccum* function in the package *vegan* (Oksanen et al., 2013) in the R statistical environment (R Core Team, 2014) to conduct the rarefaction. In these analyses, *Trachypithecus cristata* was omitted because this primate species had only one study of parasitism in captivity. As an alternative, the third approach to

correct for differences in sampling effort between conditions was to divide the observed PSR by the number of studies in our data set reporting on parasitism.

We used a paired-sample *t* test to investigate the hypothesis that PSR is higher in wild versus captive hosts. To account for the statistical nonindependence of species in comparative studies (Griffin & Nunn, 2011; Harvey & Pagel, 1991), including in paired differences, such as those used here (Lindenfors, Revell, & Nunn, 2010), we used a phylogenetic paired *t* test with the function *phyl.pairedttest* in the R package *phytools* (Lindenfors et al., 2010; Revell, 2015). In addition to performing the paired *t* test, this function provides an estimate of phylogenetic signal, λ, which can range from 0 to 1. When λ = 0, this indicates that captive versus wild differences are unrelated to phylogeny, whereas λ = 1 indicates that the difference covaries with phylogeny as expected under a Brownian motion model of evolution (Freckleton, Harvey, & Pagel, 2002; Nunn, 2011). We included the standard deviation or standard error of the rarefied PSR in the *t* tests. We downloaded a 50% majority rules consensus tree from the posterior distribution of trees inferred using a supermatrix approach and a Bayesian inference framework, available via the 10k trees project (Arnold, Matthews, & Nunn, 2010). We pruned this tree to include only the 22 species in our study. We ran these *t* tests using the three corrected estimates of PSR mentioned above.

To visualize parasite community similarity between captive and wild primates, we used principal components analysis (PCA) to summarize the matrix of parasite presence/absence in captive and wild hosts. We used the *prcomp* function in R to conduct a singular value decomposition of the original matrix, with each host having one row for captive and one row for wild presence/absence of each parasite species. Axes represent the maximum shared variance in parasite presence among hosts (Legendre & Gallagher, 2001). We retained the first two principal components on the basis of the observation of decreasing variance explained by subsequent components in a scree plot. To determine if parasite transmission mode predicted separation in the two-dimensional space, we averaged the factor loadings for parasite species in each transmission mode. This approach is a way of visualizing the axes of variation in the species composition of communities (McGarigal, Cushman, & Stafford, 2000), and have been used to investigate diversity in microbial ecology (Dollhopf, Hashsham, & Tiedje, 2001) and in the analyses of the microbiome (Clayton et al., 2016).

Beta diversity was measured as the dissimilarity between wild and captive parasite communities, in which a value of 0 indicates that the communities shared exactly the same parasites and a value of 1 indicates that communities are completely different (i.e., sharing no species). The nestedness component of beta diversity reflects the loss of some species from the original wild community and the retention of others, whereas species turnover is because of new parasite species occurring in the captive population (Baselga, 2010). Values close to 1 for the turnover component reflect total change between parasite species found in the wild versus captivity, whereas values for the turnover component close to 0 indicate that all beta diversity is because of nestedness. We computed the Sorenson index of beta diversity partitioned into the turnover component (Simpson index) and nestedness component, calculated in the R package *betapart* (Baselga & Orme, 2012).

We tested the hypothesis that parasite species identity differed between wild and captivity (dependent variables) depending on transmission mode (independent variable) using binomial logistic regressions, with transmission mode coded as a binary (presence-absence) variable. Our first set of models tested the hypothesis that parasites transmitted through vectors or intermediate hosts (independent variables) were not detected in captive animals (dependent variable). For this, we divided the whole parasite data set into separate subsets of parasites found in wild versus captive primates. When a parasite present in a wild host species was absent from the corresponding captive sample, that parasite was recorded as not found in captivity, otherwise, it was present. Our second set of models tested the hypotheses that parasites are more likely to be reported in captive environments (dependent variable) if they (a) exhibit close-contact transmission, (b) exhibit non-close contact transmission, and (c) are zoonotic (independent variables). Specifically, if a parasite species in the captive data set was not present in the wild data set, we recorded that parasite to be newly detected in captivity. Logistic regressions were conducted using the *glm* function in R, specifying a logit probability link. The significance of the model was tested using the χ^2 statistic, implemented with the ANOVA function in R. These analyses were only run for the 13 primate species with sufficient numbers of parasite species with variation in transmission mode.

To characterize how parasite traits predicted the occurrence of a parasite in captivity (whether carried over from the wild or newly acquired), we also calculated the proportion of parasites detected or not, across all host species and within each of the five transmission categories (which as noted above are not mutually exclusive). We present these data per host species and as means across species. We tested whether the proportion of parasites detected in captivity differed by transmission mode using the phylogenetic paired-sample *t* tests described above.

3 | RESULTS

The PSR of captive hosts was similar to that of their wild counterparts (phylogenetic mean difference in PSR = 1.55, $n = 22$ host species, phylogenetic paired *t* test: $t = -.82$, $p = .42$, $\sigma^2 = 1.06$, Table S1). Phylogenetic signal was low (λ = 0). Species showed remarkable variation in whether the captive or wild host communities had higher PSR (Figure 2). In one such example, *Ateles paniscus* had 31 parasite species reported from 12 captive studies, and 10 parasite species from 14 wild studies (Table S1). In general, the species accumulation curves for all species show that PSR rarely reaches an asymptote, but continues to rise with each additional study, indicating that there are many more host-parasite relationships to be discovered (Figures S1–22). Results of *t* tests were qualitatively similar across three analyses that used

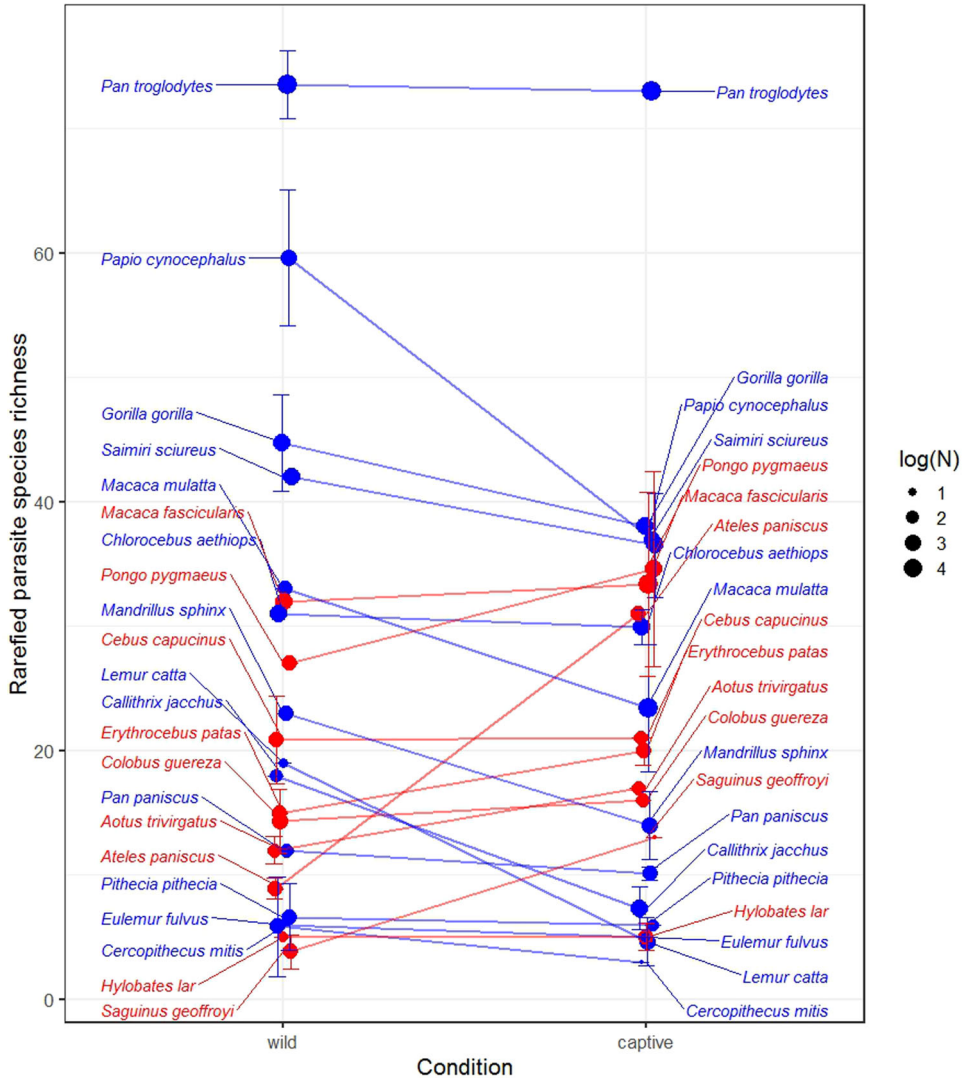

FIGURE 2 The plot of rarefied parasite species richness (PSR) in 21 paired wild and captive primate species. There was no significant difference in rarefied species richness between captive and wild conditions (phylogenetic paired-sample *t* test). PSR was rarefied by the minimum number of studies in either the wild or captive host. The size of the circle is proportional to the number of studies (log-transformed). Species in blue had lower PSR in captivity than in the wild, while species in red had higher PSR in captivity than the wild. The data were offset slightly to allow visualization of overlapping points

different corrections for differences in studies between conditions (Table S2).

The principal component analysis to examine dissimilarity in parasite community composition resulted in two primary axes (Figure 3, where each host species is represented by two points, one each for captive and wild settings, and points closer together have more similar parasite community composition than points that are farther apart). The first principal component axis (PC1) represented 22.55% of the variation in parasite community composition among hosts. The second principal component axis (PC2) represented 7.06% of the variation, and separated approximately half of the wild versus captive hosts: wild hosts largely exhibited positive values of PC2 and captive hosts tended to exhibit negative values (Figure 3). The factor loadings represent how strongly each parasite species was correlated with each axis. When averaging the mean factor loadings among parasites according to their transmission mode, parasites with intermediate hosts loaded more strongly on the positive end of PC2 (−.001) than parasites without intermediate hosts (mean = −.11).

Changes in parasite community composition between captive and wild host species pairs were predominantly because of the species turnover component of beta-diversity, with only a small contribution of the nestedness component for some host species (Figure 4, Table S3). These results indicate that the species composition of parasite communities in the wild was nearly completely replaced with a different set of parasites in captivity. Two host species were exceptions to this pattern. The captive parasite community of silvered leaf monkeys (*Trachypithecus cristata*) was a nested subset of the wild community, and for orangutans (*Pongo pygmaeus*), nestedness made up approximately 30% of the beta diversity.

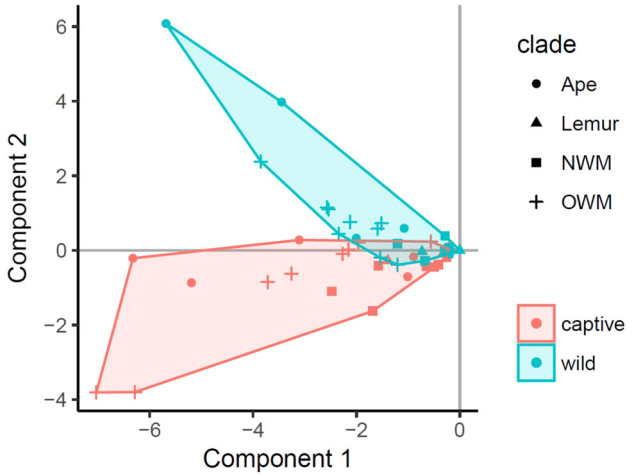

FIGURE 3 Principal components summarizing the host-parasite matrix in two dimensions. Every point in the plot is a captive or wild host and the distance among points illustrates their dissimilarity in parasite community composition. The second component, which discriminates captive and wild parasite communities, is characterized by parasites without intermediate hosts having negative factor loadings. NWM, new world monkeys, OWM, old world monkeys

Vector-borne parasites were significantly more likely to be found in the wild versus in captivity in six out of 13 primate hosts (Table 1). In only one host species were parasites with intermediate hosts significantly more likely to be reported from the wild than in captivity (Table 1). Parasites with close-contact transmission were significantly more likely to be detected in captivity in two out of 11 host species (Table 2). Non-close contact transmission was significantly more likely to be detected in captivity in one out of 11 hosts (Table 2). Parasites known to infect humans were not detected in captivity significantly more often than those that do not infect humans (Table 2). We note, however, that a large percentage of parasites detected in both wild and captive primates are also known to infect humans (mean = 88%, range = 43–100%).

Across primate species, the mean percentage of vector-borne parasites reported from wild populations but not detected in captivity was 37.5%, compared to 28% with close-contact transmission, 36% with non-close contact transmission, and 4.5% with the sexual transmission (Figure 5). Surprisingly, the proportion of parasites transmitted via intermediate hosts that were known from the wild but were not detected in captivity was relatively low (mean = 13.7%), although the proportion of parasites with intermediate hosts present in the wild sample was also low (16% in the primate GMPD). On average, 60% of parasites only detected in captivity had close-contact transmission, 55.5% of parasites only detected in captivity had non-close transmission, and 15.5% were sexually transmitted (Figure 5). Only 6.1% of parasites detected in captivity were vector-borne, and 14.0% of parasites detected in captivity had intermediate hosts. The proportion of parasites detected in captivity that had close-contact transmission was not significantly different from the proportion with non-close transmission, but was significantly higher for other transmission modes (Figure 5).

4 | DISCUSSION

Primates are held in captivity for many purposes, ranging from biological research colonies to zoological parks and wildlife rehabilitation centers. Primates also have been relatively well sampled for parasites and pathogens, in part owing to their close relationships to humans, making them well-suited for analyses comparing parasites in wild and captive populations. Building on findings from invasion biology in which many native parasite species are lost when host species are introduced into new habitats (e.g.,Torchin & Mitchell, 2004; Torchin et al., 2003), we investigated predictions involving changes in parasite composition and richness in wild compared to captive primates. Counter to our initial prediction that captive primates should harbor fewer parasites than their wild counterparts, we found no significant difference in PSR between captive and wild

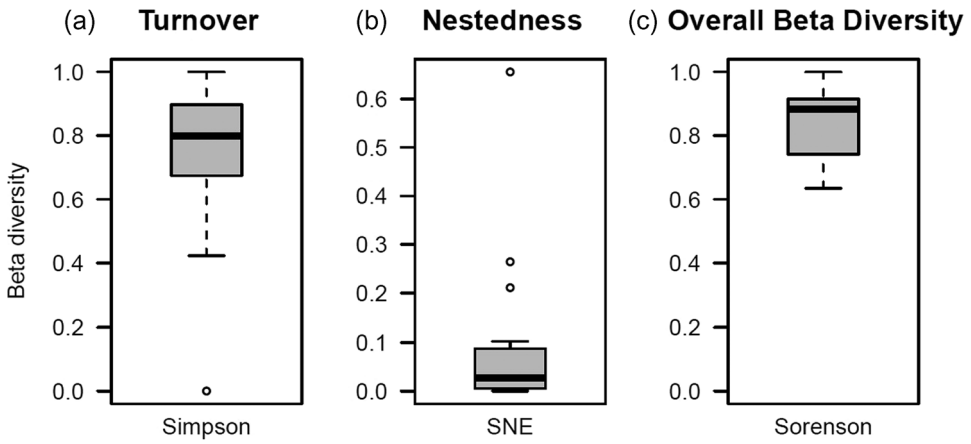

FIGURE 4 Boxplots representing the two components of beta diversity, nestedness, and turnover between parasite communities of wild and captive primates. (a) The turnover component of beta diversity (Simpson's index), (b) The nestedness component (SNE), (c) The overall beta diversity (Sorenson's index)

TABLE 1 Results of logistic regressions for each host species, predicting parasite species found in the wild and not reported in captivity, relative to parasite transmission mode. The number of parasite species lost in captivity is given out of the total wild parasite community. Rows in **bold** were significant at alpha = .05, tested against the χ^2 statistic

Host species binomial name	Proportion of parasites	Coefficient intermediate-host transmission	*p*	Coefficient vector transmission	*p*
Aotus trivirgatus	10/13	17.59	.28	−1.54	.26
Chlorocebus aethiops	**21/31**	.75	.51	**18.3**	**.006**
Colobus guereza	**15/23**	17.08	.18	17.08	.18
Erythrocebus patas	8/12	17.36	.33	17.36	.33
Gorilla gorilla	**17/34**	16.1	.16	**17.27**	**.03**
Macaca fascicularis	**25/32**	16.42	.21	1.55	.13
Macaca mulatta	27/33	−1.65	.29	17.31	.10
Mandrillus sphinx	**15/23**	1.25	.26	**18.34**	**.03**
Pan troglodytes	**55/80**	.27	.71	**2.43**	**.002**
Papio cynocephalus	53/64	**17.25**	**.02**	−.76	.43
Pongo pygmaeus	11/27	17.04	.17	.54	.53
Saimiri sciureus	**34/42**	−.55	.57	**19.16**	**.0004**
Trachypithecus cristata	**19/24**	17.4	.22	**19.1**	**.007**

groups. Instead, our findings indicated that the number of parasites detected only in captive settings generally offsets those known only from the wild. In other words, changes associated with captivity include the introduction of new parasites that replace the loss of others. Despite similar richness estimates, the community composition of parasites differed sharply between wild and captive primates. Rather than captive primate parasites being nested subsets of those from wild primate hosts, the parasite communities of many wild hosts were almost completely replaced by a novel parasite community in captivity.

Differences in parasite community composition between wild and captive populations can be partially explained by the dominant transmission mode of the parasite species. In particular, parasites found exclusively in the wild were commonly transmitted by vectors such as mosquitoes (*Aedes* sp.) and tsetse flies (*Glossina* sp.). We also predicted that parasite species detected in captivity should be transmitted by close-contact or non-close transmission (e.g., fecal-oral or contaminated substrates), but this prediction was only supported for two out of 13 host species. Across all 22 host species in this study, 60% of parasites not found in the wild but detected in captivity had close-contact transmission, whereas only 6% were vector-borne. It is interesting that parasites transmitted by close and non-close contact were common in captivity, despite regular medical care and hygiene practices. Collectively, these results illustrate that the mode of parasite transmission is an important mechanism of parasite community change when animals transition from wild to captive environments.

Table 3 highlights the examples of key parasites we observed to be common in the wild (but not in captivity) or common in captivity (but not in the wild) across parasite transmission modes. While

TABLE 2 Results of logistic regressions for each host species, predicting parasite species reported in captivity but not in the wild, on the basis of parasite transmission mode. Rows in **bold** were significant at alpha = .05, tested against the χ^2 statistic

Host species binomial name	Proportion of parasites	coefficient Zoonotic	*p*	coefficient Close contact	*p*	coefficient Environmental	*p*
Aotus trivirgatus	7/10	.41	.78	.41	.78	−18.82	.10
Chlorocebus aethiops	18/27	1.39	.18	−.8	.38	<0.001	1.00
Erythrocebus patas	14/16	NA	NA	−17.96	.26	−16.86	.34
Gorilla gorilla	18/28	1.45	.27	.69	.53	−0.62	.45
Macaca fascicularis	51/59	1.17	.16	−.23	.76	.23	.76
Macaca mulatta	**53/59**	.27	.82	**−19.18**	**.003**	**2.14**	**.03**
Mandrillus sphinx	7/16	NA	NA	−.41	.7	1.57	.18
Pan troglodytes	26/49	.59	.54	−.47	.42	−0.67	.26
Papio cynocephalus	19/30	1.16	.25	.59	.44	−0.18	.81
Pongo pygmaeus	28/44	.62	.56	.41	.54	1.1	.09
Saimiri sciureus	**18/26**	18.66	.99	**2.05**	**.02**	−1.2	.17

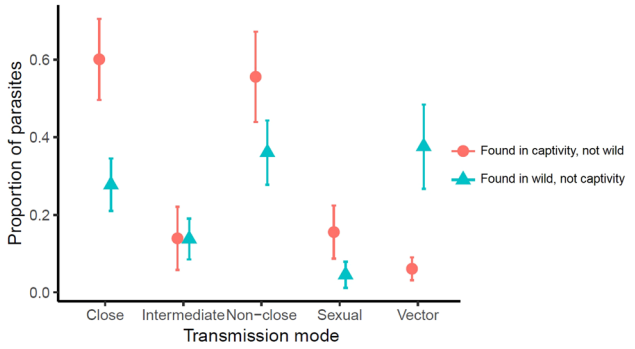

FIGURE 5 Comparison of the proportion of parasite species known from the wild but not detected in captivity (green triangles) or not reported in the wild but detected in captivity (orange circles) by parasite transmission mode. Points represent the mean proportion, and bars represent 95% confidence intervals. Note that transmission modes are not mutually exclusive (e.g., sexually transmitted parasites exhibit close contact transmission, and some parasites exhibit both close and non-close contact transmission). The proportion of parasites detected in captivity that had close-contact transmission was significantly higher than for parasites with vector-borne transmission ($t = -3.24$, $p = .005$, $\lambda = .52$), intermediate-host transmission ($t = -3.22$, $p = .005$, $\lambda = .22$), and sexual transmission ($t = -9.98$, $p < .001$, $\lambda = 0$), but was not significantly higher when compared with non-close transmission ($t = 0.43$, $p = .67$, $\lambda = .41$)

parasite species presented in Table 3 do not infect all primates, they were common across many species in our data set and are known to infect diverse hosts in captivity. The proportion of parasites that were identified exclusively in captivity and are known to infect humans ranged between 43% and 100% among primate species (Table 3, Taylor et al., 2001). Notable pathogenic and zoonotic parasites in our captive primate data set included protozoa, such as *Giardia duodenalis*, nematodes such as *Trichuris trichiura*, bacteria such as *Mycobacterium tuberculosis*, and viruses such as Herpes simplex virus and human parainfluenza viruses (Table 3). These parasites are a known concern in captive populations, given the potential fatality of captive primates, the generality, and host-breadth of the parasites, and their potential to spread to people (e.g., Stensvold et al., 2009). In future research, wild primates should be screened for parasites commonly found in captivity, because historically they have been under-appreciated in the wild (e.g., *Blastocystis*, Petrášová et al., 2011). Our results highlight important parasites to monitor in both captive and wild populations.

PSR in wild primate species depends on host life history traits and ecological factors, such as geographic range area, social group size, foraging area, and population density (Nunn, Altizer, Jones, & Sechrest, 2003; Nunn et al., 2004). In most cases, it is not possible to directly compare the effects of these variables between wild and captive animals, either because the data were not consistently provided by the authors of the original paper (e.g., group size and population density) or the variables are simply not comparable in wild versus captive settings (e.g., geographic range size). If group sizes or cumulative habitat sizes of captive and wild populations differ, this might contribute to differences in parasitism (e.g., Guégan,

TABLE 3 Common primate parasites reported in the wild but not reported in captivity, or reported in captivity but not in the wild, characterized by their transmission modes. Parasites with an asterisk are known to infect humans (Taylor et al., 2001). Note this is not an exhaustive list of all parasites in the data set

Transmission mode	Common in wild but not in captivity	Common in captivity but not in wild
Close or non-close contact	**Viruses**	**Bacteria**
	Ebolavirus sp.*	*Streptococcus pneumoniae**
	Simian immunodeficiency virus	*Pseudomonas aeruginosa**
	Bacteria	*Mycobacterium bovis**
	Treponema sp.*	*Salmonella* sp.*
		*Shigella flexerni**
		Viruses
		Deltaretrovirus STLV 2
		Simian foamy virus
		Human parainfluenza virus*
		Herpes simplex virus*
		Protozoa
		*Blastocystis hominis**
		Cryptosporidium sp.*
		*Entamoeba histolytica**
		Iodamoeba sp.*
Vector-borne	**Viruses**	**Bacteria**
	Yellow fever virus	*Francisella tularensis**
	Protozoa	**Viruses**
	Plasmodium sp.*	Chikungunya virus*
	Trypanosoma sp.*	
	Hepatocystis sp.	
	Helminth	
	*Loa loa**	

Morand, & Poulin, 2005; Poulin, 2014). If group sizes were artificially larger or smaller in captivity than in the wild, this could cause PSR in captivity to deviate from wild conditions. However, because some factors, such as group size, likely have stronger effects on infection prevalence than on PSR, we do not believe this would bias our results (Rifkin, Nunn, & Garamszegi, 2012). In addition, the captive setting itself could lead to variation in parasitism; relevant variables include whether housing was indoor versus outdoor, the number and types of other animal species in the facility, and changes in husbandry practices over time. Again, these data were not consistently reported in the papers on captive primates, and would most likely add random noise, not systematic bias.

In a comparative study such as ours, including over 550 parasites from 600 studies, differences in methodology among

studies could impact our estimates of PSR. For example, no single study quantified the total PSR of a particular host; instead, studies typically focus on a group of parasites, such as helminths, gut or blood-borne protozoa, or viruses. Detection methods used across studies have different sensitivities, and not all studies were able to identify parasites to the species level, or discern closely related species (e.g., *Entamoeba histolytica* vs. *E. dispar*). Infection statuses for viruses and bacteria are often inferred from serology or from molecular screening, whereas helminth and gastrointestinal protozoan infections are assessed from fecal examination following flotations or fecal smears. We do not expect that including results from serology and molecular techniques will adversely affect the results, because similar global analyses of parasite infection did not detect differences in results when omitting serology-based data (Olival et al., 2017; Pandit et al., 2018). Although these factors may limit the depth of interpretation of the results, we have no reason to expect that they will cause systematic biases across hosts and parasites that favor any particular hypotheses that we tested. Instead, these confounds should add random noise to the data, making patterns more difficult to detect, rather than mislead us to accept a false positive result. Looking forward, several new approaches offer opportunities for consistent sampling across species, which would revolutionize attempts to understand broad patterns of parasitism. In particular, DNA barcoding and metagenomics provide methods to consistently identify current infection (Besansky, Severson, & Ferdig, 2003; Pallen, 2014). Given rapid advances in molecular techniques, a standardized procedure for molecular identification of parasites may not be far off.

By providing a comparative context for understanding parasitism in wild and captive primates, our study reinforces the need for vigilance during reintroduction programs. Captive primates reintroduced to the wild could bring with them a number of parasites that are unique to the captive environment, with detrimental effects on the wild population (Viggers, Lindenmayer, & Spratt, 1993). Any animal intended for reintroduction should be quarantined and screened for disease agents before release, and individuals harboring pathogens should be cleared of infection or removed from reintroduction programs (e.g., callitrichid hepatitis in captive *Leontopithecus* populations, Viggers et al., 1993). Similarly, reintroduced individuals that had never encountered parasites from wild environments could be highly susceptible to naturally-occurring infections. Both of these patterns should be assessed when planning translocation programs (Petrášová et al., 2010). For example, moose and caribou reintroductions in North America suffered owing to the spread of a meningeal worm from sympatric white-tailed deer (Anderson, 1972), and reintroduced whooping cranes exhibited high mortality because of eastern equine encephalitis virus spread by mosquito vectors (Carpenter, Clark, & Watts, 1989). In an effort to restore wild populations of golden lion tamarins (*Leontopithecus rosalia*), individuals raised in captivity were released to the wild, and half died of an unknown disease (Kleiman et al., 1986). It is crucial to assess these factors in programs to reintroduce animals from captivity to the wild (Baker, 2002; Hartley & Sainsbury, 2017). Findings in this study highlight the specific parasites that are common for each species and should be monitored.

In conclusion, parasite communities varied considerably between wild and captive settings for 22 primate species, but without significant differences in the total number of parasite species harbored by each group. The dissimilarity between wild and captive parasite communities was driven more by parasite replacement rather than by net parasite loss. Replacement of vector-borne parasites from the wild, and the addition of new close-contact and non-close transmitted parasites in captivity, are potential threats to captive primates and present risk of spillover or spill-back to humans. Our results also contribute to the understanding the ecological drivers of parasite communities, with applications for captive and wild management of primate disease agents.

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DATA ACCESSIBILITY

Data are available as electronic supplementary material with this article.

ETHICS STATEMENT

We confirm that we adhered to the guidelines of the American Society of Primatology Principles for the Ethical Treatment of nonhuman Primates.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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