Seroprevalence of pathogens in domestic carnivores on the border of Madidi National Park, Bolivia

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Abstract

The importance of diseases of domestic animals in the conservation of wildlife is increasingly being recognised. Wild carnivores are susceptible to many of the pathogens carried by domestic dogs and cats and some of these pathogens have caused disease outbreaks and severe population declines in threatened species. The risk of disease spillover from domestic to wild carnivores in South America has not been extensively investigated. This study examined the disease exposure of domestic carnivores living near a protected area in Bolivia. Forty dogs and 14 cats living in three towns on the eastern border of Madidi National Park were sampled. High levels of exposure to canine distemper virus, canine parvovirus, *Sarcoptes scabiei* and *Toxoplasma gondii* were found among domestic dogs, with similarly high levels of exposure to feline parvovirus, feline calicivirus and *T. gondii* being found among domestic cats. If contact occurs between domestic and wild carnivores, disease spillover may represent an important risk for the persistence of wild carnivores in the region. Additional research is therefore necessary to determine if wild carnivores living in proximity to these domestic carnivore populations are being exposed to these pathogens.

INTRODUCTION

The conservation community is increasingly aware of the disease risks to threatened wildlife (Woodroffe, 1999; Daszak, Cunningham & Hyatt, 2000, 2001; Deem *et al.*, 2001; Funk *et al.*, 2001). Domestic animals, and dogs (*Canis familiaris*) in particular, have been the proven or suspected reservoir for infectious agents that have led to numerous epidemics in a variety of wild carnivore species (Sillero-Zubiri, King & MacDonald, 1996; Cleaveland *et al.*, 2000). African lions (*Panthera leo*: Roelke-Parker *et al.*, 1996), Ethiopian wolves (*Canis simensis*: Laurenson *et al.*, 1998), and African hunting dogs (*Lyacon pictus*: Gascoyne *et al.*, 1993), for instance, have suffered population declines as a result of disease spillover from domestic dogs.

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Populations of domestic carnivores, especially dogs, act as ideal disease reservoirs; they may travel large distances into wildlife habitats and, in some areas, high resident population densities of feral or unvaccinated animals allow even very virulent pathogens to persist in the broader carnivore population (Gottelli & Sillero-Zubiri, 1992; Cleaveland & Dye, 1995; Cleaveland, Laurenson & Taylor, 2001). As habitat fragmentation increases (Skole & Tucker, 1993), the frequency and intensity of interactions between domestic and wild carnivores will also probably increase (Laurance, Vasconcelos & Lovejoy, 2000). Even in the absence of direct contact between domestic and wild carnivores, the ability of some pathogens to remain viable in the environment for extended periods of time (Table 1) means that domestic and wild carnivore sympatry may be sufficient for disease transmission. There are many examples from Africa (Ginsberg, Mace & Albon, 1995; Laurenson *et al.*, 1998) and North America (Thorne & Williams, 1988; Valenzuela, Ceballos & Garcia, 2000; Miller *et al.*, 2002) in which diseases of domestic animals caused morbidity and mortality in endangered carnivore populations. Therefore, knowledge of the diseases present in the domestic carnivore populations in close proximity to wildlife is essential for conservation planning.

Table 1. Major modes of transmission, infectious materials and length of persistence in environment for carnivore pathogens

Disease agent	Method of transmission	Infectious material	Environmental persistence
Canine distemper virus	Oronasal exposure to aerosolised body fluids	Respiratory secretions, urine, tissues	Hours in warm temperatures; weeks in cold temperatures
Canine parvovirus	Oronasal exposure to faeces	Faeces	Months
Canine coronavirus	Ingestion	Faeces	Hours in warm temperatures
Canine herpesvirus	Oronasal exposure to body fluids; *in utero* exposure	Respiratory secretions, genital lesions	Probably not
Canine adenovirus-1	Oronasal exposure to body fluids; possibly also *via* ectoparasites	Saliva, urine, faeces, tissues	Days in warm temperatures; months in cold temperatures
Rabies virus	Bite wounds	Saliva, tissues	Probably not
Feline leukaemia virus	Oronasal exposure to saliva or other bodily fluids	Saliva, blood, tissues	Up to hours
Feline immunodeficiency virus	Bite wounds	Blood and saliva	No
Feline panleukopaenia virus	Oronasal exposure to faeces or body fluids	Urine, faeces, all other secretions	Months to a year
Feline herpesvirus	Inhalation of oronasal and ocular secretions	Respiratory and oral secretions	Probably not
Feline coronavirus	Oronasal exposure to faeces or body fluids; *in utero* exposure	Faeces, saliva, urine, respiratory secretions	Hours to weeks
Feline calicivirus	Inhalation of oronasal and ocular secretions	Respiratory and oral secretions; faeces	Up to a week
Toxoplasma gondii	Ingestion of infected tissues or oocysts from faeces; *in utero* exposure	Faeces of cats; tissues of intermediate hosts	Months
Dirofilaria immitis (heartworm disease)	Mosquito-borne	N/A	N/A
Sarcoptes scabiei (sarcoptic mange)	Direct and indirect contact	Mites (all life stages)	Hours to weeks
Leptospira interrogans	Bite wounds, contact with or ingestion of tissues, urine or urine-contaminated water	Urine, tissues	Days in standing water

Source: Samuel, Pybus & Kocan (2001) and Greene (1998*c*). N/A, not applicable.

While numerous reports of disease spillover exist in the literature, these events are not distributed evenly across the globe. Well-co-ordinated and long-standing domestic dog vaccination programmes in much of North America and Europe have made disease spillover from domestic carnivores to wildlife rare; in fact, in the case of rabies, it is spillover from wildlife into domestic populations that is more of a concern (Rupprecht *et al.*, 1995; Chang *et al.*, 2002). In the developing world, most of the well-documented occurrences of spillover have been in Africa (Gascoyne *et al.*, 1993; Sillero-Zubiri *et al.*, 1996; Nel *et al.*, 1997; Cleaveland *et al.*, 2000). This may be an artifact of the fact that many African carnivores inhabit open savannahs where they are easily monitored, or it may be simply an artifact of the lack of studies in other regions. In places where studies have been conducted, the potential for disease spillover has been documented. For instance, Mainka *et al.* (1994) identified antibodies to canine distemper virus (CDV) and canine parvovirus (CPV) in domestic dogs inhabiting the Wolong Reserve in China, indicating the potential for spillover from dogs to giant pandas (*Ailuropoda melanoleuca*) and other wild carnivores. Disease studies from Latin America are few, but as the human population there increases, it is likely that spillover events will, or already have, occurred. The present study aims to increase our knowledge of disease exposure in domestic carnivores living close to a large protected area, the Madidi National Park (MNP) and Integrated Management Area (IMA), in Bolivia.

Numerous species of canids and felids inhabit the park, including jaguars (*Panthera onca*), pumas (*Puma concolor*), ocelots (*Leopardus pardalis*), bush dogs (*Speothos venaticus*), margays (*Leopardus wiedi*), Geoffroy's cats (*Oncifelis geoffroyi*) and jaguarundis (*Herpailurus yaguarondi*) (WCS-Bolivia, 2002). Captive animals of many of these species have been infected with domestic carnivore disease agents, indicating their susceptibility to these pathogens (Table 2). All but the puma are considered to be threatened species by the World Conservation Union (IUCN) and are listed on Appendix I or II of the Convention on International Trade in Endangered Species (CITES).

Our primary objective was to determine the seroprevalence of antibodies to a variety of pathogens in domestic carnivores inhabiting a region of high priority for

Table 2. Selected reports of exposure to or infection with disease agents in Latin American non-domestic carnivores

Disease agent	Species	References
Canine distemper virus	Crab-eating fox[†‡], maned wolf[†], coati[†‡], jaguar[†‡]	Mann *et al.* (1980); Appel *et al.* (1994); Cubas (1996); Murray *et al.* (1999)
Canine parvovirus	Crab-eating fox[†‡], maned wolf[†], bush dog[†‡]	Cubas (1996)
Rabies virus	Puma[†‡]	Murray *et al.* (1999)
Feline leukaemia virus	Puma[†‡]	Jessup *et al.* (1993); Murray *et al.* (1999)
Feline immunodeficiency virus	Puma[‡], jaguar[‡], ocelot[‡], margay[‡], Geoffroy's cat, jaguarundi[‡], others	Olmsted *et al.* (1992); Brown, Miththapala & O'Brien (1993); Carpenter & O'Brien (1995)
Feline panleukopaenia virus	Jaguar[†], margay[†‡], ocelot[†‡], puma[†]	Paul-Murphy *et al.* (1994); Cubas (1996)
Feline herpesvirus	Puma[‡]	Paul-Murphy *et al.* (1994); Murray *et al.* (1999)
Feline coronavirus	Puma[‡]	Paul-Murphy *et al.* (1994)
Feline calicivirus	Puma[‡]	Murray *et al.* (1999)
Toxoplasma gondii	Puma[‡], jaguar[‡]	Paul-Murphy *et al.* (1994); Murray *et al.* (1999)
Dirofilaria immitis (heartworm disease)	Jaguar[‡], jaguarundi[‡]	Otto (1974)
Sarcoptes scabiei (mange)	Pampas fox[†]	Deem *et al.* (2002)

[†] Indicates that clinical signs were observed.
[‡] Indicates a species whose range includes the Madidi National Park.

carnivore conservation. We sampled owned, free-roaming domestic carnivores living in communities situated on the borders of the MNP/IMA and performed serological testing for numerous disease agents, including viral, bacterial, parasitic and protozoal pathogens. Serological assays are commonly used for epidemiological studies (Greiner & Gardner, 2000*b*), even though they cannot distinguish between active and previous infection, or infection and disease (Barr, 1996; Evermann & Eriks, 1999). While the limitations of serology can be problematical for determining the disease status of an individual animal, in this case we are using it to screen for exposure on a population level (Christensen & Gardner, 2000). For the purposes of this study, it is not important if a given dog was ill as a result of infection with canine distemper; instead, it is important to know if dogs in the population are commonly infected with this virus, regardless of its clinical effect on individuals.

STUDY SITE AND METHODS

We sampled in several locations to increase the number of animals represented and assess the heterogeneity in disease exposure among closely situated communities. Three towns on the eastern border of the MNP/IMA were chosen for sampling on the basis of ease of access for the field researchers and proximity to the park (Fig. 1). Madidi is a relatively new park of over $18\,000$ km^2 in north-western Bolivia. It consists of tropical rainforest and cloud forest habitat. San Buenaventura, which houses the main offices of the park, has a population of about 6200. It is situated on the Rio Beni, directly across the river from Rurrenabaque, a larger town (pop. 7000) with an active tourist industry. Therefore, dogs in San Buenaventura are probably exposed to dogs from Rurrenabaque and other

nearby towns and villages. Tumupasa, however, is an isolated village of just under 1000 people, situated about 60 km north-west of San Buenaventura. Ixiamas is about 30 km north-west of Tumupasa; it has a population of about 5600 and has grown rapidly over the last few years. The primary economic activities in the area are subsistence hunting and agriculture. Ixiamas has a commercial dairy farm; dogs from this town might therefore be more likely to be exposed to livestock pathogens. None of the towns (including Rurrenabaque) has a resident veterinarian, although rabies vaccination campaigns are periodically operated by the government. Ixiamas and Rurrenabaque both have small medical clinics and several pharmacies, but Tumupasa has neither.

We obtained permission from the municipal governments of each town prior to sampling and in both San Buenaventura and Ixiamas, a town resident was assigned to work with our team during sample collection. Although we tried to recruit dogs and cats from all of the neighborhoods in each village, no attempt was made to randomise the subjects. In fact, an effort was made to include hunting dogs, which are the most likely to have contact with wildlife. We went from house to house, discussing our project with the residents and asking for their consent to sample their pets. Dog owners were asked to provide information on age and vaccination status of their animal and to indicate whether the dog was purchased specifically for hunting. Unfortunately, conversations with residents revealed that although some dogs were designated as 'hunting dogs,' categorising dogs by hunting status was not realistic because most dogs not specifically identified as hunting dogs nevertheless participated in hunting activities. Only dogs 5 months of age and older were sampled, to ensure that the antibodies detected were endogenous and not of maternal origin (Greene, 1998*b*). Using a muzzle and manual restraint,
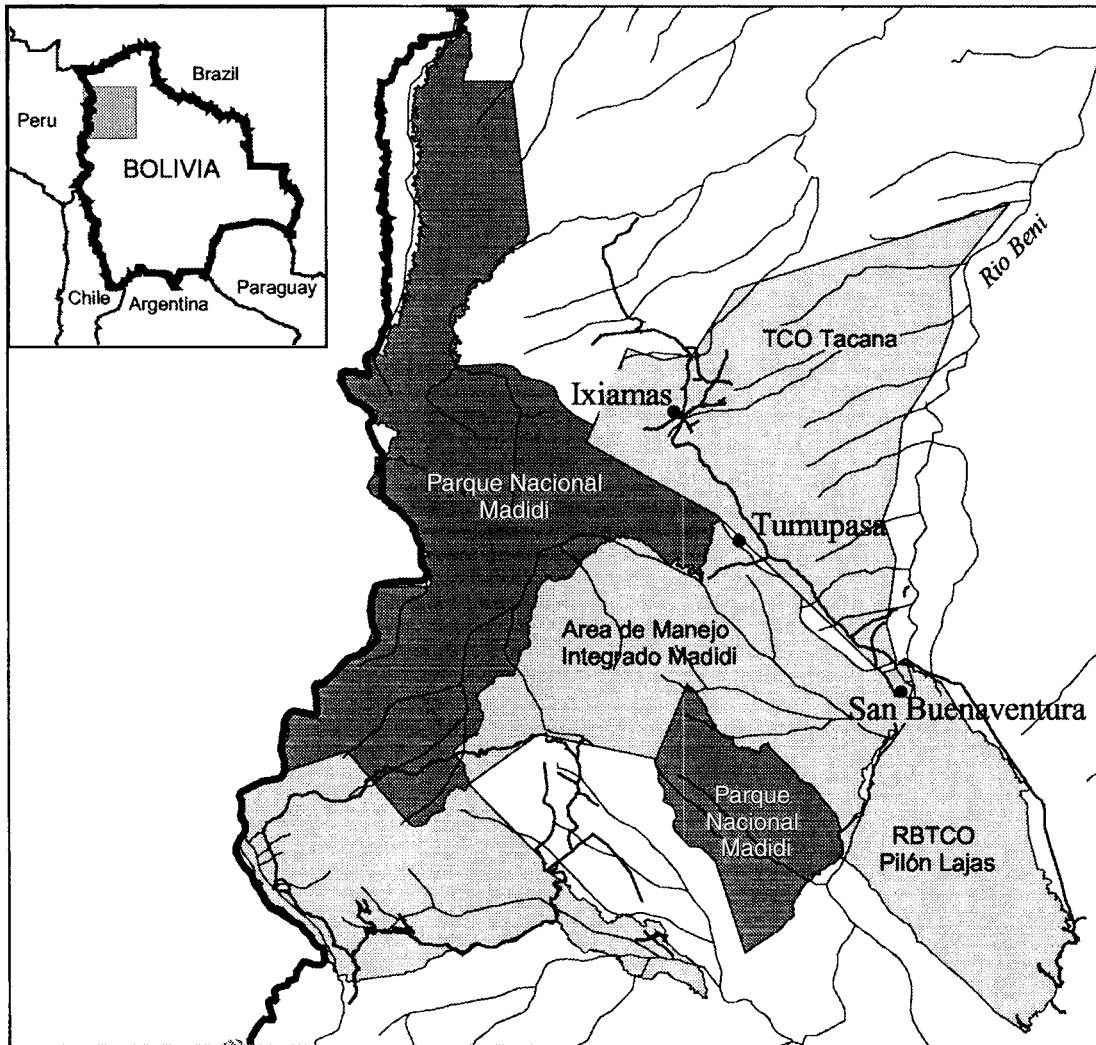
Fig. 1. Study sites in relation to Madidi National Park, Bolivia. Dark grey shading indicates the extent of the park, while light grey shading indicates park buffer zones – either indigenous territory (TCO) or Integrated Management Areas (IMA). The IMA was established at the same time as, and in association with, the park, but has less stringent restrictions on resource use and extraction.

blood was drawn from the jugular or cephalic vein. Three dogs required 1mg of acepromazine maleate (Boehringer-Ingelheim, Ingelheim, Germany) intramuscularly to facilitate sampling.

Cats (*Felis catus*) are infrequently kept as pets in this area, consequently our sample size was smaller than for dogs. Owners were often unsure of the age of their cats, but all the cats we sampled had adult dentition, indicating that they were at least 6 months of age. All cats required chemical restraint for sampling. Each cat was given an intramuscular injection of 10 mg of a premixed combination of equal parts tiletamine hydrochloride and zolazepam (Telazol®, Fort Dodge Laboratories, Fort Dodge, IA) either alone or with 0.1 mg of acepromazine. On a few occasions, additional ketamine hydrochloride (Ketaset®, Fort Dodge Laboratories, Fort Dodge, IA) was administered to effect sufficient immobilisation. Blood was collected from the medial femoral or jugular vein. Sterile ophthalmic ointment was applied to the eyes; temperature, heart rate, respiratory rate and pulse

quality were monitored every 3–5 min until the cats were ambulatory.

Serum was separated from the cellular components within a few hours of collection and stored on ice. Sera were transported to the USA on ice and then stored in a freezer at $-70\,^{\circ}$C prior to submission to diagnostic laboratories in the USA and Switzerland. The diagnostic methodology for each test is listed in Table 3. *Dirofilaria immitis* and the feline leukaemia virus (FeLV) assays detect antigen in blood and so generally indicate an active or very recently cleared infection (Barr, 1996; Goodwin, 1998).The remainder of the tests detect antibodies to the disease agent in question.

Chi-square tests were used to determine if there were statistically significant differences in the prevalences of each disease between the three towns; in some cases, small sample sizes required use of the more conservative G-test. Analyses of variance (ANOVA) were used to test for interactions between vaccination status, town and positive titres to different pathogens.

Table 3. Methodologies and positive cut-off values used by laboratories to detect exposure to disease agents

Pathogen	Methodology	Positive cut-off
Canine adenovirus	Antibody SN	1:4
Canine coronavirus	Antibody SN	1:8
Canine distemper virus	Antibody SN	1:8
Canine herpesvirus	Antibody SN	1:8
Canine parvovirus	Antibody HAI	1:10
Dirofilaria immitis	Antigen ELISA	P/N
Sarcoptes scabiei	Antibody ELISA	P/N
Feline calicivirus	Antibody SN	1:8
Feline coronavirus	Antibody KELA	1:8
Feline herpesvirus	Antibody SN	1:8
Feline immunodeficiency virus	Antibody ELISA	P/N
Feline immunodeficiency virus confirmatory	Western blot	P/N
Feline laeukaemia virus	Antigen ELISA	P/N
Feline panleukopaenia virus	Antibody HAI	1:10
Leptospira interrogans	Antibody MA	1:100
Toxoplasma gondii – canine	Antibody IHA	1:64
Toxoplasma gondii – feline	Antibody KELA	1:48

Analyses were performed on serum at the Cornell University Veterinary Diagnostic Lab, Ithaca, NY except the *S. scabiei* assay, which was performed at Labor Laupeneck, Switzerland.

SN, serum neutralisation; HAI, haemagglutination-inhibition; ELISA, enzyme-linked immunosorbent assay; KELA, kinetic ELISA; IHA, indirect haemagglutination; MA, micro-agglutination; P/N, test scored as positive or negative.

RESULTS

Sufficient quantities of serum for at least one test were collected from 14 dogs and three cats from San Buenaventura, 13 dogs and five cats from Ixiamas and 13 dogs and six cats from Tumupasa. Twenty-nine dogs and three cats had been previously vaccinated against rabies; the remainder were either never vaccinated or had an unknown vaccination status. Five dogs were identified as purpose-bred hunting dogs, although many other dogs participated in hunting activities. The majority of dogs were between 6 months and 3 years of age.

Seroprevalence for each disease agent in dogs is given in Table 4. Antibodies to canine distemper virus (CDV)

Table 5. Titres of the five dogs from Ixiamas testing positive for exposure to two *Leptospira interrogans* serovars

	Leptospira serovar	
Individual	*canicola*	*hardjo*
Dog 16	1:3200	Neg
Dog 18	1:400	Neg
Dog 19	1:1600	Neg
Dog 23	Neg	1:800
Dog 27	1:400	1:100

Titres ≥1:1600 are considered suggestive of recent infection with that serovar.

neg, negative.
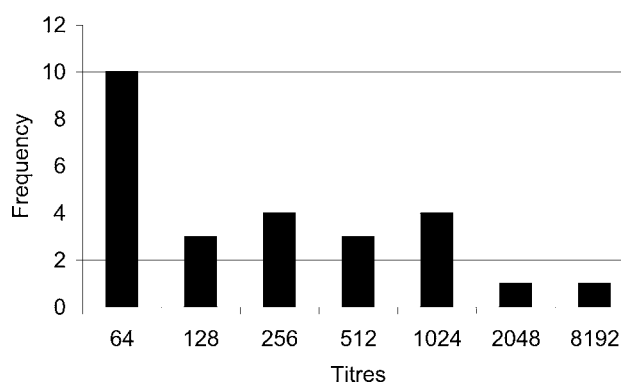
Fig. 2. Frequency of titres to *Toxoplasma gondii* found in dogs using indirect haemagglutination. Titres ≥1:1024 are considered to be indicative of active or recent infection.

and canine parvovirus (CPV) were very common; over 90% of the dogs were positive. Canine adenovirus (CAV) antibodies were found in over three-quarters of the dogs, while canine herpesvirus (CHV) and canine coronavirus (CCV) were less common, each of the latter occurring in about one-third of the dogs sampled. Antibodies to one or two serovars of *Leptospira interrogans* were found in about one-fifth of dogs. These serovars were identified as *hardjo* and *canicola* (Table 5). Antibodies to *Toxoplasma gondii* were found in 62% of dogs, of which six (36%) had high titres (≥1:1024) indicative of active infection (Fig. 2).

Table 4. Number of dogs in each town with positive serological results for each pathogen over the number of dogs tested

Town	Canine distemper virus	Canine herpes-virus	Canine adeno-virus	Canine corona-virus	Canine parvo-virus	*Leptospirosis*	*Toxoplasmosis*	*Dirofilaria*	*Sarcoptes*
San Buena-ventura *n* (%)	5/5 (100)	3/5 (60)	3/5 (60)	2/5 (40)	5/5 (100)	1/5 (20)	3/5 (60)	5/6 (83)	6/14 (43)
Tumupasa *n* (%)	8/9 (89)	2/9 (22)	5/9 (56)	3/9 (33)	7/9 (78)	2/9 (22)	6/9 (67)	4/10 (40)	12/13 (92)
Ixiamas *n* (%)	11/12 (92)	4/12 (33)	12/12 (100)	3/12 (25)	12/12 (100)	5/12 (42)	7/12 (58)	2/12 (17)	4/13 (31)
Total *n* (%)	24/26 (92)	9/26 (35)	20/26 (77)	8/26 (31)	24/26 (92)	8/26 (31)	16/26 (62)	11/28 (39)	22/40 (55)

The figures in brackets are the % of dogs that had a positive serological result for the pathogen being tested for.

Table 6. Number of cats in each town with positive serological tests for each pathogen over number tested

Town	Feline parvovirus	Feline leukaemia virus	Feline calicivirus	*Leptospirosis*	*Toxoplasmosis*	*Dirofilaria*
San Buenaventura n (%)	3/3 (100)	0/3	3/3 (100)	1/3 (33)	3/3 (100)	0/3
Tumupasa n (%)	6/6 (100)	0/6	5/6 (83)	0/6	6/6 (100)	1/6 (17)
Ixiamas n (%)	5/5 (100)	1/5 (20)	5/5 (100)	0/5	4/5 (80)	0/5
Total n (%)	14/14 (100)	1/14 (7)	13/14 (93)	1/14 (7)	13/14 (93)	1/14 (7)

Antibodies to feline herpesvirus, feline coronavirus, and feline immunodeficiency virus were not found in any cats.
The figures in brackets are the % of cats that had a positive serological result for the pathogen being tested for.

Dogs with high *T. gondii* titres were found in all three towns; one from San Buenaventura, three from Ixiamas and two from Tumupasa. Heartworm antigen was found in 39% of dogs and antibodies to *Sarcoptes scabiei* were present in 55% of dogs.

All cats had antibodies to feline parvovirus and over 90% had antibodies to feline calicivirus (Table 6). Antibodies to feline immunodeficiency virus, feline coronavirus, and feline herpesvirus were not detected in any cats ($n = 14$). One cat was positive for FeLV antigen. *Leptospira interrogans* antibodies were found in one cat; these were against the *canicola* serovar. Most cats (93%) had antibodies to *T. gondii*, although none had titres high enough to suggest recent infection (Fig. 3). One cat was positive for heartworm antigen.

Heartworm disease in dogs was more common in San Buenaventura (G-test; $P = 0.0198$), while sarcoptic mange in dogs was more common in Tumupasa (G-test; $P = 0.0016$). No other significant differences were found (see Table 4). There were also no significant differences between villages in the expected prevalences of any of the pathogens between dogs that were and were not vaccinated against rabies. Sample sizes were insufficient to explore differences between rabies-vaccinated and unvaccinated cats.
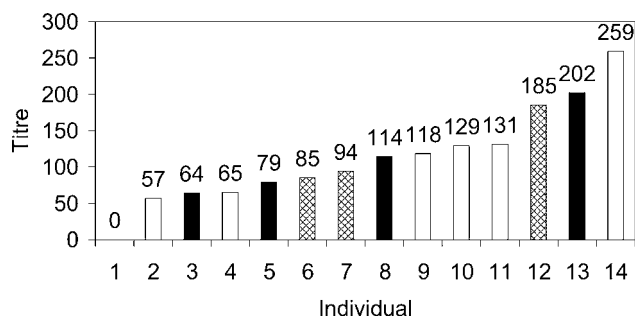
Fig. 3. *Toxoplasma gondii* titre for each cat sampled, using kinetic enzyme-linked immunosorbent assay (ELISA). Titres 1:750 are considered to be indicative of active or recent infection. Open bars, Tumupasa; cross-hatched bars, Ixiamas; solid bars, San Buenaventura.

DISCUSSION

This survey indicates that the potential for spillover of pathogens from domestic to wild carnivore populations in this region of Bolivia is real. Exposure to several disease agents was documented at relatively high prevalences, including CDV, CPV, feline parvovirus, and *T. gondii*. It is possible for spillover of these pathogens from regionally large domestic populations to less dense wild populations to result in disease epidemics in populations of endangered carnivores (e.g. Gascoyne *et al.*, 1993; Roelke-Parker *et al.*, 1996; Laurenson *et al.*, 1998). Whether this will occur depends on the exposure and immunological status of the wild populations, which we did not investigate, but our evidence does show that domestic carnivores are exposed to disease agents to which wild species are susceptible.

Of the disease agents we identified, CDV and CPV are perhaps of greatest concern. CDV is a generalist pathogen that has been responsible for serious epidemics and population declines in wild carnivores (Alexander & Appel, 1994; Roelke-Parker *et al.*, 1996; Cleaveland *et al.*, 2000; Kennedy *et al.*, 2000). It is spread most commonly by close contact between individuals *via* aerosolised respiratory secretions, although it can remain viable in the environment for hours or even weeks in cool temperatures (Greene & Appel, 1998: see Table 1). The climate of the Amazon basin probably precludes environmental survival, leaving direct contact as the probable means of transmission. CPV, however, is transmitted *via* the faecal–oral route and can survive for months in the environment (Mann *et al.*, 1980; Hoskins, 1998: see Table 1). Although natural parvovirus infections of wild canids have been reported (Hoskins, 1998; Peterson *et al.*, 1998), the potential effects on wild populations are poorly understood (Mech & Goyal, 1993; Johnson, Boyd & Pletscher, 1994).

Over 90% of the domestic dogs we sampled were seropositive for CDV and CPV, indicating that these viruses are endemic in this population (Table 4). The level of prevalence found in this study differs sharply from that found in a recent study in the Amazon basin of Brazil. Using similar methodologies, Courtenay, Quinnell & Chalmers (2001) found CDV and CPV antibodies in only 9% and 13%, respectively, of 23 dogs. Those authors

also sampled sympatric crab-eating foxes (*Cerdocyon thous*). Despite a large amount of peridomestic activity by the foxes, documented with radiotelemetry, none of the 24 foxes tested had antibodies to either virus (Courtenay *et al.*, 2001). While these findings might seem reassuring, it is not surprising given the low prevalence of these diseases in that domestic dog population.

The other canine viruses we investigated occurred at moderate to high seroprevalence, but are less likely to be of epidemic significance in wild carnivores (Holzman, Conroy & Davidson, 1992; Hoskins, 1998; Woods, 2001). The presence of these viruses is noteworthy because co-infection with one of these agents and CDV or CPV may cause increased morbidity and mortality and may prolong viral shedding in infected canids (Evermann *et al.*, 1980; Pratelli *et al.*, 1999, 2001).

The results of the feline virus survey were more encouraging, although our sample size was small. The lower prevalence of pathogen exposure in cats may be a result of their apparently low density in this region, or their less social nature. Antibodies to three viruses were not found at all, including feline immunodeficiency virus and feline coronavirus, two of the more worrisome pathogens for wild felid populations. We cannot conclude that these viruses are not present in the domestic cat population, but even if every family owned one cat – highly unlikely given the unpopularity of cats in the region – we can be 95% confident that these viruses are present in less than 20% of the population (Cannon & Roe, 1982). In addition, the single positive result for FeLV antigen was considered a weak positive, indicating the need for a retest in 3–4 weeks to confirm infection. Because a retest was not possible, we cannot confirm the presence of this virus in this animal. Antibodies to feline panleukopaenia were found in all 14 cats (Table 6). This virus is of conservation concern because it can cause clinical disease in felids, procyonids and mustelids and it persists in the environment (Greene, 1998*a*; Steinel *et al.*, 2000: Table 1). Infection with feline calicivirus, a pathogen known to cause disease in wild felids (Sabine & Hyne, 1970; Kadoi *et al.*, 1997), was also widespread in the cats we sampled. In most cases, this virus causes high morbidity but low mortality in domestic cats (Gaskell & Dawson, 1998); however, more virulent strains that cause high mortality do exist (Pedersen *et al.*, 2000).

A striking result was the high seroprevalence of *T. gondii* exposure in dogs and the large number of dogs with titres suggestive of active infection (Fig. 2). Although most cats had antibodies (Table 6), their titres were generally quite low (Fig. 3). This is not surprising, since cats are the definitive host for this parasite. They are probably infected as kittens and may have either partial or complete immunity to reinfection. Both dogs and cats are most probably infected *via* the ingestion of raw meat, including rodents and other wild mammals. Humans are also commonly exposed by this route, but children may be more at risk for infection by ingestion of contaminated soil (Tenter, Heckeroth & Weiss, 2000). In these semi-rural areas of Bolivia, cats are allowed to roam freely and indoor litter boxes are not provided. Cats are therefore defaecating in soil where children can be readily exposed.

Leptospirosis is a zoonotic bacterial disease considered by some to be a re-emerging infectious disease in the tropics (Brandling-Bennett & Pinheiro, 1996). Many serovars exist and serological testing often reveals some degree of cross-reactivity among the different serovars. Only the serovars *hardjo* and *canicola* were recognised in our samples (Table 5). Dogs are the primary host of *canicola*, so this serovar is probably endemic in this area (Greene, Miller & Brown, 1998). The serovar *hardjo* is most often found in cows and other hoofed animals, including wild bovids. Interestingly, most positive dogs were found in Ixiamas, which has a large dairy farm (Tables 4 & 5). However, the serovar *canicola* was found in four out of the five Ixiamas dogs with titres, so the presence of cattle may be coincidental. Two dogs from Ixiamas had titres ≥ 1600, which is considered suggestive of active or recent infection. The few dogs with positive titres from San Buenaventura and Tumupasa had titres of 100, all to serovar *hardjo*. Unfortunately, our cross-sectional data are insufficient to document active infection; paired titres separated by 2–3 weeks would be necessary. The low seroprevalence of *Leptospira* found in domestic cats is probably related to their smaller ranging patterns and their natural dislike of standing water. Data concerning human *Leptospira* infection from the region at this time would be helpful in determining if dogs could serve as sentinels for human and wildlife infections.

Heartworm disease caused by *Dirofilaria immitis* is found worldwide in tropical, subtropical and temperate climates, although it has a higher prevalence in warmer regions (Anderson, 2001). In general, wherever heartworm disease occurs in domestic dogs, it also occurs in domestic cats, but at a lower prevalence (Ralston, Stemme & Guerrero, 1998). It has been diagnosed in many wild carnivore species, including canids, felids, mustelids and pinnipeds (Anderson, 2001). In the present study, we found a significantly higher prevalence of this disease in dogs from San Buenaventura, which is the only community we sampled that is located near a large body of water (Table 4). Foci of heartworm disease around rivers have been previously reported (Okoniewski & Stone, 1983) and are probably related to proximity to mosquito breeding areas. Our results show, however, that even in Tumupasa and Ixiamas, which are more distant from a major river, the disease is present.

Over half of the dogs tested were positive for *Sarcoptes scabiei* antibodies (Table 4). This is a zoonotic parasite, which causes clinical disease in free-ranging Bolivian carnivores (Deem *et al.*, 2002; C. V. Fiorello, unpublished results) and disease epidemics resulting in population declines in other carnivore species (Lindström *et al.*, 1994; Pence & Windberg, 1994; Shibata & Kawamichi, 1999). Antibodies to this parasite wane and are not detectable by about 2 months after resolution of infection (Arlian *et al.*, 1996; Lower *et al.*, 2001). The presence of antibodies therefore indicates a current or very recent infection, unlike antibodies to most viral pathogens, which persist for years (Greene, 1998*b*). In Tumupasa, all but one of

the dogs tested were positive, an alarming statistic. Why the prevalence of mange was higher in Tumupasa is not clear. The overall health of the dogs from Tumupasa did not seem to differ from that of the other two towns and no other disease agents were found disproportionately more often in Tumupasa.

The use of serology, which documents infection but not disease, may be viewed as a limitation or an advantage of this study. The stratified prevalence survey employed here is commonly used to estimate the frequency of infection in a population (Greiner & Gardner, 2000*a*). Disadvantages to serology include the possibility of cross-reaction of reagents to antibodies against other agents (false positives) as well as the (less likely) possibility for false negatives. The presence of antibodies is dependent on numerous factors, including the immune status of the host, the timing of the initial infection and the outcome of the infection (Greiner & Gardner, 2000*a*). The detection of antibodies is further dependent on the accuracy of the test, the presence of cross-reacting substances in the host, sample handling and proper test execution (Barr, 1996; Greiner & Gardner, 2000*a*). Even if the test is carried out correctly, determining what constitutes a positive result may be somewhat arbitrary, since cut-off values often vary between laboratories (Barr, 1996; Greiner & Gardner, 2000*b*). The interpretation of a positive test – one in which antibody was detected – varies as well. For example, antibodies to feline immunodeficiency virus found in the serum of an adult cat using a Western blot nearly always indicates that the cat is currently infected with the virus (Barr, 1996). However, antibodies to feline coronavirus found in the serum of a cat by any method indicates that the cat was probably infected by a feline coronavirus, or a related coronavirus, at some point in the past (Barr, 1996). Neither positive result tells you with certainty if the cat in question is, or will become, clinically ill, nor does it tell you which viral strain infected the cat or if that strain was particularly virulent. Paired titres from the same individual at different times would have been useful to determine the presence and timing of infection, but this was not possible for this study. While serology has its drawbacks, it provides information on the exposure history of a given individual, so that it is not necessary to sample an animal during the short period of clinical disease to document infection.

At least five of the dogs we sampled were purchased or bred specifically for hunting and these dogs go into the forest regularly. Even those dogs that were not considered to be hunting dogs by their owners sometimes accompanied them hunting and all dogs were unconfined and free to roam at will. Direct contact with wildlife did occur; owners occasionally pointed out scars or healing wounds on their dogs that they attributed to encounters with peccaries (although when questioned, all of the owners admitted that they had not actually witnessed the encounter). Even if hunting dogs infrequently have direct contact with wild carnivores, they are urinating and defaecating in the forest where wildlife may be exposed. Because many carnivore species use urine and faeces to mark their territories, they are likely to investigate any dog urine and faeces they encounter in the forest. Given that dog and cat pathogens can and do cross taxonomic lines, this situation allows for disease transmission. Dogs from a large population in which many diseases are endemic enter the forest and may contact susceptible wildlife species, which may have little or no immunity to these pathogens. In the dense rainforest of the Amazon basin, outbreaks of infectious diseases in carnivores may go unnoticed or unrecognised, leading to population declines of these already threatened species.

Acknowledgements

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