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Serosurvey of Free-ranging Amur Tigers in the Russian Far East

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ABSTRACT: Wild Amur tigers (*Panthera tigris altaica*, $n=44$) from the Russian Far East were tested for antibodies to feline leukemia virus, feline corona virus (FCoV), feline immunodeficiency virus, feline parvovirus (FPV), canine distemper virus (CDV), *Toxoplasma gondii*, and *Bartonella henselae*. Antibodies to FCoV, CDV, FPV, and *T. gondii* were detected in 43, 15, 68, and 42% of tigers, respectively. No differences were detected in antibody prevalence estimates between tigers captured as part of a research program and those captured to mitigate human-tiger conflicts. Domestic dogs (*Canis familiaris*) were tested as a potential source for CDV; 16% were vaccinated against CDV and 58% of unvaccinated dogs were antibody positive for CDV. A high percentage of tigers were exposed to potential pathogens that could affect the survival of this species. We recommend continued monitoring of wild tigers throughout Asia, development of standardized sampling and postmortem examination procedures, and additional research to better understand potential domestic and wild animal sources for these pathogens.

Key words: Amur tiger, canine distemper virus, feline coronavirus, feline parvovirus, infectious disease, *Panthera tigris altaica*, Siberian tiger, Toxoplasma.

Infectious disease may reduce carnivore survival and reproduction (Woodroffe, 1999), and catastrophic die-offs of carnivores are frequently related to disease (Young, 1994). Even small decreases in survival and reproduction in small populations of slowly reproducing species like tigers (*Panthera tigris*) could increase extinction risk (Chapron et al., 2008). Only about 3,500 wild tigers survive in just 7% of their historic range in small (mostly <100 individuals), isolated populations that are particularly prone to extinction (Walston et al., 2010). There are no published data on infectious disease in wild tigers beyond a few case reports (Nowell and Jackson, 1996). With fewer than 400 in the wild, disease could significantly affect Amur tiger (*Panthera tigris altaica*) populations. To better understand disease threats to Amur tigers, we conducted a serosurvey of wild tigers in the Russian Far East (RFE) and assessed whether infectious disease resulted in infirmity leading to human-tiger conflict (HTC).

We collected serum from tigers as part of a telemetry-based research program on the Sikhote-Alin Biosphere Zapovednik (SABZ; 44°46′N, 135°48′E; hereafter, "research tigers"; Miquelle et al., 2010), and in human-tiger conflicts ("conflict tigers"; Goodrich et al., 2011) in Russia, 1992–2004 (Fig. 1; Table 1). Most tigers were captured in Aldrich foot-snares or darted from a helicopter (Goodrich et al., 2001, 2010), but some conflict tigers were captured or killed by local people or Inspection Tiger (Russian Ministry of Natural Resources; Miquelle et al., 2005; Goodrich et al., 2011). Tigers were aged on the basis of tooth eruption and wear, gum recession, and known birth dates (Goodrich et al., 2001). The 4,000-km^2 SABZ is strictly protected, with access limited to staff and visiting scientists. Domestic animals are prohibited, so potential for disease transmission from them is lower than in surrounding areas, although most tigers had home ranges extending beyond SABZ (Goodrich et al., 2010). Most of the remaining habitat consisted of state lands, where livestock, dogs (*Canis familiaris*), and tiger–domestic animal encounters were common (Miquelle

FIGURE 1. Area where tigers were sampled in the Russian Far East, 1992–2004. Asterisks indicate capture sites of tigers from human-tiger conflicts, and circles indicate towns where dogs were sampled. Research tigers were sampled on and near the Sikhote-Alin Zapovednik (reserve).

et al., 2005). To assess the potential for disease transmission from dogs, we collected blood and recorded vaccination histories (from owner records) of nonferal dogs from three villages, including one bordering our research site (Fig. 1). For feline parvovirus (FPV), feline corona virus (FCoV), and toxoplasmosis, we tested for a relationship between age and antibody titer with a nonparametric linear regression (Conover, 1980). Antibody prevalences were compared using Fisher's exact test.

Mean age of sampled tigers was 4.1 years (SD=3.6, range, 0.3–12, n=44) and sex ratio was 1.35 (male:female). All tigers were negative for antibody to feline immunodeficiency virus (FIV), feline leukemia virus (FeLV), and *Bartonella henselae*, but some were positive for other pathogens (Table 1). There were no differences in prevalences between sexes (P>0.21). Only FPV antibody prevalence changed significantly with age (R^2=0.04, P=0.006, n=41), with all animals >7 yr old positive. Three tigers that initially were negative for FCoV had antibody when retested 0.75 to 4 yr later. Although the prevalence for canine distemper virus (CDV) antibody was more than twice as high for conflict tigers (30% vs. 13%), the difference was not significant (P=0.37; Table 1). We detected no differences between research and conflict tigers for antibody to other pathogens (P>0.19; Table 1). No samples collected before 2000 were positive for CDV antibody (n=27). One antibody-positive tiger died from canine distemper (CD; Quigley et al.,

TABLE 1. Results of tests for antibodies to infectious diseases (Washington Area Disease and Diagnostic Laboratory, Pullman, Washington, USA) in blood collected from wild Amur tigers captured in the Russian Far East, 1992–2004.

	Research tigers		Conflict tigers		Total	
Disease agent	% Positive	N[a]	% Positive	n	% Positive	n
Feline leukemia virus[b]	0	29	0	15	0	44
Feline coronavirus[c]	41	29	47	15	43	44
Feline immunodeficiency virus[b]	0	29	0	15	0	44
Canine distemper virus[d]	11	27	23	13	15	40
Feline parvovirus[c]	68	28	69	13	68	41
Toxoplasma gondii[e]	62	29	46	13	62	42
Bartonella henselae[f]	0	17	0	0	0	17

[a] n = total number of animals captured.

[b] Antigen (FeLV) and antibody (FIV) enzyme-linked immunosorbent assays (ELISAs).

[c] Immunofluorescent antibody tests (positive titer>1:25).

[d] Virus-neutralizing antibodies using the Rockborn strain of canine distemper virus.

[e] Hemaglutination inhibition assay (positive titer>1:64).

[f] Western blot (National Veterinary Laboratory Inc, Franklin Lakes, New Jersey, USA).

2010) after 39 days in captivity, during which it was supported with intravenous fluids and force feeding. Of the remaining five positive animals, one's fate was unknown, one was shot during a conflict 1 month after capture, one survived 7 months before being killed by a poacher, one survived for ≥10 yr in a zoo, and one survived for 2 yr before we discontinued monitoring. Of 45 dogs sampled, only 16% were vaccinated and, of those that were not, 58% had antibody to CDV.

We did not detect antibody to FeLV or FIV, and only one of 24 mortalities of radio-collared tigers for which cause of death was known was attributable to infectious disease (Goodrich et al., 2008), suggesting that these agents were rare in wild Amur tigers. The high prevalence of antibody to FCoV, FPV, and *Toxoplasma gondii* may reflect that these pathogens are endemic in the population or the presence of widespread exposure through contact with infected domestic or wild animals. Opportunities for exposure of tigers to these infectious agents are many, given their extensive movements (Goodrich et al., 2010) and widespread human activity outside of protected areas (Miquelle et al., 2005).

Canine distemper virus is of concern because of catastrophic die-offs of other carnivores (Munson, 2001), high rates of exposure of tigers to dogs (Goodrich et al., 2011), high antibody prevalence (58%) and low rates of vaccination (16%) in dogs, and the transmissible nature and high case-fatality rate of CDV (Williams, 2001). There are reports of CD in captive tigers (Munson, 2001), and CD killed a wild tiger; however, this is the first report of survival of CDV exposure by wild tigers. CDV appeared in our samples only after 1999, suggesting CD may be an emerging disease.

Unvaccinated dogs were implicated as CDV reservoirs, causing outbreaks in wild carnivores (Funk et al., 2001), and they may be the source for Amur tigers. This would explain high infection rates in conflict tigers, which commonly kill dogs (Goodrich et al., 2011). Education to encourage vaccination of dogs using vaccines readily available through State veterinarians may decrease threats. Furthermore, increasing wild prey will reduce dependence of tigers on dogs (Goodrich et al., 2011).

Other potential CDV reservoirs include wolves (*Canis lupus*), red fox (*Vulpes vulpes*), raccoon dogs (*Nyctereutes procyonoides*), and badgers (*Meles meles*; Munson, 2001). Tigers prey on all of these species (Miquelle et al., 1996). Canine distemper virus is unlikely to be sustained in low-density populations with small group sizes (Almberg et al., 2010), which applies to all these carnivores, including tigers (Goodrich et al., 2010). However, the protection offered by low-density populations may be offset in winter because CDV survives for long periods at low temperatures (Appel, 1987), making indirect transmission possible. Sampling is needed to determine whether wild species are reservoirs and whether CDV is endemic. Disease risk in tigers should be monitored throughout Asia with the use of standardized sampling and postmortem examination protocols. Whenever tigers are handled, blood samples should be collected for serologic investigations, and thorough postmortem examinations should be conducted on all dead tigers.

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