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Evidence of Feline Immunodeficiency Virus, Feline Leukemia Virus, and *Toxoplasma gondii* in Feral Cats on Mauna Kea, Hawaii

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ABSTRACT: We determined prevalence to feline immunodeficiency virus (FIV) antibodies, feline leukemia virus (FeLV) antigen, and *Toxoplasma gondii* antibodies in feral cats (*Felis catus*) on Mauna Kea Hawaii from April 2002 to May 2004. Six of 68 (8.8%) and 11 of 68 (16.2%) cats were antibody positive to FIV and antigen positive for FeLV, respectively; 25 of 67 (37.3%) cats were seropositive to *T. gondii*. Antibodies to FeLV and *T. gondii* occurred in all age and sex classes, but FIV occurred only in adult males. Evidence of current or previous infections with two of these infectious agents was detected in eight of 64 cats (12.5%). Despite exposure to these infectious agents, feral cats remain abundant throughout the Hawaiian Islands.

Key words: *Felis catus*, feral cat, FeLV, FIV, Hawaii, *Toxoplasma gondii*.

Infectious diseases of introduced predatory mammals are important for at least two reasons: first, diseases may limit populations, thereby reducing the risk of predation on native wildlife. Second, some infectious agents may be transmitted to native wildlife, domestic pets, or humans. Feral domestic cats (*Felis catus*) in Hawaii may carry and transmit feline immunodeficiency virus (FIV), feline leukemia virus (FeLV), and the coccidian protozoan *Toxoplasma gondii*. Feline immunodeficiency virus and FeLV have been evaluated as potential biological control agents in island ecosystems (Courchamp and Sugihara, 1999), and *T. gondii* is known to cause illness in several Hawaiian bird species (Work et al., 2000, 2002), the endangered Hawaiian monk seal (*Monachus schauinslandi*) (Honnold et al., 2005), and humans (Dubey and Beattie, 1988).

Although the pathogenesis of FIV and FeLV in cats has been well studied, and some impacts of *T. gondii* on wildlife are known, seroprevalence of infectious agents in wild felids in remote natural areas of Hawaii is poorly understood. Our objective was to assess three infectious agents in feral cats on Mauna Kea, Hawaii. We examined FIV and FeLV to determine whether these potential biological control agents already existed in feral cats, and we examined toxoplasmosis prevalence to determine whether a risk of transmission exists for native wildlife, particularly for endangered bird species on Mauna Kea, Hawaii.

We captured feral cats on the north and west slopes of Mauna Kea (19°50′N, 155°35′W) from 9 April 2002 to 16 May 2004 as part of efforts to protect and restore an endangered Hawaiian forest bird. Tomahawk® model 106 live traps (23 × 23 × 85 cm) were distributed at 150-m intervals along 15 transects in subalpine woodland from 1,750 to 3,000 m in elevation. We covered traps with a layer of plastic to protect trapped cats from rain and cold, and we placed a cloth rag inside for bedding. We used canned cat food, sardines, and mackerel as bait. We checked set traps daily, and we wired traps open when unattended. Feral cats were euthanized by gunshot to the head according to University of Hawaii IACUC protocol 97–063.

We determined sex, and we determined age by the presence of complete adult dentition. We also collected blood samples. A 21-gauge hypodermic needle was used to draw 10 ml of blood by cardiac puncture postmortem. Snap™ Combo FeLV Anti-

TABLE 1. Prevalence of feline leukemia (FIV) antibodies, feline immunodeficiency (FeLV) antigen, and *Toxoplasma gondii*-specific immunoglobulin G (IgG) and immunoglobulin M (IgM) antibodies in feral cats on Mauna Kea, Hawaii, 2002–04 (percentage with number of age/sex class sample in parentheses).

			T. gondii		
	FIV	FeLV	IgG	IgM	IgG and IgM
Adult					
Male	17 (6/36)	17 (6/36)	29 (10/35)	0 (0/35)	3 (1/35)
Female	0 (0/23)	13 (3/23)	27 (6/22)	9 (2/22)	9 (2/22)
Juvenile					
Male	0 (0/3)	33 (1/3)	67 (2/3)	0 (0/3)	0 (0/3)
Female	0 (0/6)	17 (1/6)	29 (2/7)	0 (0/7)	0 (0/7)
Overall	9 (6/68)	16 (11/68)	30 (20/67)	3 (2/67)	5 (3/67)

gen/FIV antibody enzyme-linked immunosorbent assays (ELISAs) (IDEXX Laboratories, Inc., Portland, Maine, USA), were used in the field with fresh whole blood according to the manufacturer's instructions. These tests are portable kits that provide results in 10 min when a conjugate is added to whole blood. Remaining whole fresh blood was placed in 2-ml serum collection vials, stored on ice, and centrifuged, and plasma/serum was aliquoted, frozen (−10 C), and sent to Colorado Veterinary Diagnostic Laboratories (Colorado State University, Fort Collins, Colorado, USA) for analysis of antibodies to *T. gondii*. Immunoglobulin G (IgG) and immunoglobulin M (IgM) ELISAs confirmed past exposure or recent infection with *T. gondii*, respectively, at threshold titers of 1:64 (Lappin et al., 1989).

We took blood samples from 50 feral cats on the west slope and from 21 cats on the north slope of Mauna Kea. We measured exposure/infection to FIV/FeLV in 68 cats and *T. gondii* in 67 cats, with 64 cats assayed for all three agents. Six and 11 of 68 cats were serologically or antigen positive to FIV or FeLV, respectively (Table 1). Feline immunodeficiency virus occurred only in adult males. Twenty-five of 67 cats were seropositive to toxoplasmosis. Of 64 cats tested for all three agents, four cats were positive to *T. gondii* and FIV, three cats to *T. gondii* and FeLV, and one cat was positive for both viruses.

Feral cats on Mauna Kea lead a solitary existence, frequently roaming over great distances far from human habitation (Tomich, 1986). In contrast to urban animals leading more colonial lifestyles, Mauna Kea cats probably do not have as much conspecific contact, thereby limiting opportunities for horizontal disease transmission. The presence of FIV only in adult males, however, is consistent with the suspected primary mode of transmission, which is biting (Yamamoto et al., 1988). We failed to detect FIV in eight adult males among 21 cats from the north slope, but this result is probably due to inadequate sample size rather than a lack of geographic mixing. We suspect mixing is not a problem because a male cat with a radio collar repeatedly traveled 25 km between the west and northeast slopes, and the genetic structure of feral cats suggests substantial gene flow between Mauna Kea and Mauna Loa populations, which are >50 km apart (USGS-BRD, unpubl. data).

Feline leukemia virus may be transmitted through contaminated saliva, blood, or other body fluids (Maruyama et al., 2003), but the virus is extremely labile, surviving only 24–48 hr in a moist environment at room temperature (Cotter, 1998). Therefore, close contact is required for transmission. Kittens also may be infected transplacentally, through nursing, or by licking (Cotter, 1998). The cat flea

(*Ctenocephalides felis*) is also a potential vector of FeLV (Vobis et al., 2003), but it has yet to be recovered from cats on Mauna Kea, possibly due to the cool, dry climate. These modes may explain how the virus is maintained in a population that lacks a colonial social structure.

Cats are the definitive host of *T. gondii* (Wallace, 1973), and toxoplasmosis has been reported in Hawaii since the 1950s (Tilden, 1953). Toxoplasmosis has caused mortality of native Hawaiian birds such as captive Nēnē (*Nesochen sandvicensis*), wild Red-footed Booby (*Sula sula*) (Work et al., 2002), and critically endangered ʻAlalā (*Corvus hawaiiensis*) (Work et al., 2000). Moreover, *T. gondii* oocysts may enter marine environments in municipal sewage or storm water runoff, sporulate in seawater (Lindsay et al., 2003) and thereby infect a variety of marine mammals, including dolphins (Migaki et al., 1990) and seals (Holshuh et al., 1985).

Vertical transmission of *T. gondii* to offspring can occur transplacentally or when kittens consume infected milk (Kenny et al., 2002), but cats typically become infected by eating raw meat, birds, or rodents containing cysts (Acha and Szyfres, 1980). Humans and wildlife primarily develop toxoplasmosis after ingesting sporulated oocysts shed in cat feces (Dubey and Beattie, 1988). Work et al. (2000) suspected that free-ranging ʻAlalā may have contracted fatal toxoplasmosis by ingesting *T. gondii* oocysts from infected feral cat feces, tissue cysts from transport hosts, or from invertebrates that ingested oocysts. Other ground-feeding species such as the endangered Nēnē and the introduced Erckel's Francolin (*Francolinus erckelii*) may become infected after consuming forage contaminated with sporulated oocysts (Work et al., 2002).

The degree to which feline diseases limit or regulate cat populations in the wild is not well understood. Feline leukemia virus may strongly limit populations (Courchamp and Sugihara, 1999). Cat populations were depressed by 7–20% in a population model with only 4.3–12.4% FeLV prevalence (Fromont et al., 1997). The cat population on Mauna Kea may be depressed by feline diseases, but their presence suggests they would not be effective agents in a biological control strategy. Feral cats remain abundant throughout most of the Hawaiian Islands (Winter, 2003), and they are difficult to control in the remote wilderness.

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