## Genotype identification of toxoplasma gondii in macropods from a zoological park in Florida, USA.

Spriggs M, Jiang T, Gerhold R, Stedman N, López-Orozco N, Su C.

Journal of Zoo and Wildlife Medicine. 2020 Mar;51(1):131-9.

**Abstract:** There are limited reports of the genetic characterization of Toxoplasma gondii infecting captive macropods in North America. A novel genotype, ToxoDB PCR-RFLP genotype 263, was reported from six wallabies at a zoological facility in Virginia, USA, prompting an investigation into the genotypes from T. gondii strains infecting macropods at a zoological park in Florida, USA. Cardiac muscle and/or lung samples from an agile wallaby (Macropus agilis, n = 1), red kangaroos (Macropus rufus, n = 8), red-necked wallaby (Macropus rufogriseus, n = 1), and a tammar wallaby (Macropus eugenii, n = 1) that died between 2014 and 2018 were collected. All 11 cases were confirmed to have died from systemic toxoplasmosis by histopathology and immunohistochemical staining. Multilocus PCR-RFLP genotyping of T. gondii was performed directly on tissue samples or on parasites isolated from myocardium by mouse bioassay. Two cases of toxoplasmosis were identified as the reported novel genotype, ToxoDB PCR-RFLP genotype 263, but no common source of exposure could be identified. Five cases were identified as genotype 2 (type III strain, haplogroup 3), and four cases were identified as genotype 216, which has been previously reported in North American wildlife. There were no overt differences in lesion severity or distribution related to genotype. These results suggest that the premise was contaminated with at least three genotypes of T. gondii causing systemic toxoplasmosis in macropods. The largest cluster of fatal toxoplasmosis in macropods in the study period occurred following severe rainfall flooding of the exhibit, suggesting the transmission of T. gondii by water and pointing out the importance of this transmission mechanism. In summary, our study revealed three T. gondii outbreaks that caused significant loss of macropods within 5 yr in a zoological facility in Florida. More studies are needed to understand transmission and prevention of toxoplasmosis in sensitive zoo animals.

**Background:**

* *Toxoplasma gondii* = intracellular coccidian parasite that causes disease in birds and mammals
	+ Definitive host = felids
* Felids shed oocysts → hosts ingest oocysts
	+ Vertical transmission of tachyzoites is possible
* Genotypes 1 and 3 have been found in domestic animals
* Genotypes 4 and 5 have been found in wildlife
* Macropods in Virginia died after infection with genotype 4 or a novel genotype 263
* Atavaquone has successfully treated some Bennet’s wallabies

**Key Points:**

* In a Florida zoo, 20 wallabies/kangaroos died from systemic toxoplasmosis after peracute illness
* Clinical signs = sudden death or diarrhea, ataxia, shaking, recumbency, seizures for <3 days prior to death
* Lesions were common in brain, lung, myocardium, and digestive tract
* Most were positive on serology, but not an effective antemortem test
* Three different genotypes sequenced but no differences in illness
	+ Genotype 263 associated with macropod deaths in Virginia zoo
	+ Genotype 2 and 216 seen in wildlife
* An Australian freckled duck also died from genotype 2 infection in this collection
* Largest cluster of mortalities associated with large amount of rainfall
	+ Macropod exhibits should have good drainage to prevent storm water from collecting

**Conclusions:** Toxoplasma gondii can cause a rapidly fatal infection in wallabies, especially associated with increased rainfall.

## PREVALENCE AND POTENTIAL IMPACT OF TOXOPLASMA GONDII ON THE ENDANGERED AMARGOSA VOLE (*MICROTUS CALIFORNICUS SCIRPENSIS*), CALIFORNIA, US

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J Wildl Dis, 53 (1), 62-72 Jan 2017

**Taxa:** Mammalia → Rodentia → Myomorpha (suborder) → Cricetidae

**Abstract:** We investigated the prevalence of Toxoplasma gondii in 2011-15 to assess its potential threat on the endangered Amargosa vole (*Microtus californicus scirpensis*) in California, US. Surveillance was simultaneously performed on populations of syntopic rodent species. We detected antibodies to T. gondii in sera from 10.5% of 135 wild-caught Amargosa voles; 8% of 95 blood samples were PCR-positive for the T. gondii B1 gene, and 5.0% of 140 sympatric rodent brain samples were PCR-positive. Exposure to T. gondii did not change the probability that an animal would be recaptured in the field study. Behavioral response to domestic cat (*Felis catus*) and bobcat (*Lynx rufus*) urine was evaluated in five :nonendangered Owens Valley voles (*Microtus californicus vallicola*) as surrogates for Amargosa voles and seven uninfected controls. Voles showed mild attraction to mouse urine and had neutral reactions to domestic cat urine whether or not infected. Time spent near bobcat urine was approximately twice as high in infected than in uninfected voles (although not statistically significant). The presence of T. gondii in wild Amargosa vole and sympatric rodent populations may hinder the endangered Amargosa vole population's ability to recover in the wild.

**Background:**

* Endangered Amargosa voles were found to have *Toxoplasma gondii* DNA (13%) and antibodies (8%)
* *T. gondii* is a zoonotic protozoan
	+ In humans, mostly asymptomatic but can be fatal
	+ Sexual reproduction in felines (definitive host)
	+ Infective oocysts passed feces and can remain viable to up to years in the environment
	+ Latent tissue cysts in muscle and brain
	+ Can also be transmitted transplacentally
* In rodents, *T. gondii* infection can be subclinical or cause muscle or respiratory issues

**Key Points:**

* Wild Amargosa voles had a moderate seroprevalence (<10%) and DNA prevalence blood PCR (8.4%)
* Sympatric rodents had a similar DNA presence on brain PCR (10%)
* Adult Amargosa voles were 8x more likely to be exposed than subadults, but no age factor in sympatric rodents
* In sympatric rodents, higher rates of infection in the wet season
* Most experimentally exposed Owen Valley voles were seropositive and brain PCR positive but PCR negative on spleen and heart
* Owen Valley voles were attracted to house mice urine but not to domestic cat or bobcat urine
* Infected voles were more likely to have spent time near bobcat urine

**Conclusions:** *Toxoplasma gondii* is prevalent in Amargosa voles and sympatric rodents.

Mize, Erica L., Shaun M. Grassel, and Hugh B. Britten. "Fleas of black-footed ferrets (mustela nigripes) and their potential role in the movement of plague." *Journal of wildlife diseases* 53.3 (2017): 521-531.

**Abstract:**  Sylvatic plague is one of the major impediments to the recovery of the black-footed ferret (Mustela nigripes) because it decimates their primary prey species, prairie dogs (Cynomys spp.), and directly causes mortality in ferrets. Fleas are the primary vector of Yersinia pestis, the causative agent of sylvatic plague. The goal of this research was to better understand the flea fauna of ferrets and the factors that might influence flea abundance on ferrets. **Fleas from ferrets were tested for Y. pestis in a post hoc assessment to investigate the plausibility that some ferrets could act as incidental transporter hosts of fleas infected with Y. pestis.** Fleas were collected from ferrets captured on the Lower Brule Indian Reservation in central South Dakota, US from 2009 to 2012. A total of 528 fleas collected from 67 individual ferrets were identified and tested for the presence of Y. pestis with a nested PCR assay. **The predominant flea recovered from ferrets was Oropsylla hirsuta**, a species that comprises 70–100% of the fleas recovered from prairie dogs and their burrows in the study area**. Yersinia pestis was detected at low levels in fleas collected from ferrets with prevalence ranging from 0% to 2.9%; male ferrets harbored significantly more fleas than female ferrets.** Six of 67 ferrets vaccinated against plague carried fleas that tested positive for Y. pestis, which suggests ferrets vaccinated against plague could inadvertently act as incidental transporter hosts of Y. pestis–positive fleas.

**Key Points:**

* Endangered ferret rediscovered in 1988 after presumed extinction
	+ Obligate predator of prairie dogs – both highly susceptible to sylvatic plague – *Yersinia pestis*
* Prairie dogs commonly parasitized by Orophylla hirsuta & O. tuberculate – both transmit plague
* Plague exposure documented in badgers, coyotes, raccoons, red fox, skunks, and swift fox
* Only coyotes have similar fleas (O. hirsuta) to prairies dogs (not the most common coyote flea – 5%)
* Larger flea numbers on male ferrets likely due to larger body size
* Ferrets may transmit infected fleas
* 6/67 ferrets vaccinated against plague carried fleas that tested positive for Y. pestis
	+ Ferrets vaccinated against plague could act as incidental transporter hosts of Y. pestis–positive fleas.
* Deltamethrin treatments did not seem to help reduce incidence of fleas

**Take-Home:** O. hirsute is the most common flea of the black footed ferret which can transmit plague to prairie dogs

Hoogland, John L., et al. "Plague in a colony of Gunnison's prairie dogs (Cynomys gunnisoni) despite three years of infusions of burrows with 0.05% deltamethrin to kill fleas." *Journal of wildlife diseases* 54.2 (2018): 347-351.

ABSTRACT: **At Valles Caldera National Preserve in New Mexico, US, infusing Gunnison’s prairie dog (Cynomys gunnisoni) burrows with an insecticide dust containing 0.05% deltamethrin killed fleas which transmit bubonic plague**. The reduction in the number of fleas per prairie dog was significant and dramatic immediately after infusions, with a suggestion that the reduction persisted for as long as 12 mo. **Despite the lower flea counts, however, a plague epizootic killed .95% of prairie dogs after 3 yr of infusions (once per year).** More research is necessary for a better understanding of the efficacy of insecticide dusts at lowering flea counts and protecting prairie dogs from plague

Intro

* Prairie dogs are highly susceptible to plague (yersinia pestis)
	+ Epizootic outbreaks of plague typically kill .95% of residents within infected colonies of all four species of prairie dogs that inhabit the western US
* Transmitted by fleas
	+ Common species include *Oropsylla hirsuta, Oropsylla labis,* and *Oropsylla tuberculata cynomuris*
* In this report they infused Gunnison’s prairie dog (Cynomys gunnisoni) burrows with an insecticide dust containing 0.05% deltamethrin
	+ No control group
	+ Infusions given yearly for 3 years
* The reduction in the number of fleas per prairie dog was significant and dramatic immediately after infusions (given in September)
	+ flea prevalence in September–October was reduced from 69% to 5% in 2013 and from 55% to 1% in 2014
	+ Long term reduction in flea intensity, including in juveniles that had not been alive for the infusion
* No adverse effects noted in prairie dog health
* Despite the lower flea counts, a plague epizootic killed 95% of prairie dogs after 3 yr of infusions
* Possible that this outbreak was caused by a different mode of transmission (ie aerosol from an infected carcass) or could have been caused by low level of fleas present
* **Takeaway:** Deltamethrin was effective at reducing the prevalence of fleas in the Gunnison’s prairie dog, but did not prevent an outbreak of yersinia pestis

**Leptospira, parvovirus, and toxoplasma in the North American river otter (*Lontra canadensis*) in North Carolina, USA.**

Sanders, C.W., Olfenbuttel, C., Pacifici, K., Hess, G.R., Livingston, R.S. and DePerno, C.S.

*Journal of wildlife diseases*, 2020;56(4):791-802.

The North American river otter (Lontra canadensis) is the largest mustelid in North Carolina, US, and was once extirpated from the central and western portions of the state. Over time and after a successful reintroduction project, otters are now abundant and occur throughout North Carolina. However, there is a concern that diseases may have an impact on the otter population, as well as on other aquatic mammals, either through exposure to emerging diseases, contact with domestic animals such as domestic cats (Felis catus), or less robust condition of individuals through declines in water quality. **We tested brain and kidney tissue from harvested otters** for the pathogens that cause leptospirosis, parvovirus, and toxoplasmosis. Leptospirosis and toxoplasmosis are priority zoonoses and are maintained by domestic and wild mammals. Although parvovirus is not zoonotic, it does affect pets, causing mild to fatal symptoms. Across the 2014–15 and 2015–16 trapping seasons, **we tested 220 otters (76 females, 144 males) using real-time PCR for Leptospira interrogans, parvovirus, and Toxoplasma gondii.** Of the otters tested, 1% (3/220) were positive for L. interrogans, 19% (41/220) were positive for parvovirus, and 24% (53/220) were positive for T. gondii. Although the pathogens for parvovirus and toxoplasmosis are relatively common in North Carolina otters, the otter harvest has remained steady and the population appears to be abundant and self-sustaining. Therefore, parvovirus and toxoplasmosis do not currently appear to be negatively impacting the population. However, subsequent research should examine transmission parameters between domestic and wild species and the sublethal effects of infection.

**Background**

* IUCN 5/13 otter species endangered, only North American river otter is least concern, stable
* *Leptospira interrogans*: aerobic spirochete, mammals, reptiles, amphibians; shed leptospires in urine
	+ Fatal to sea otters
* Canine parvovirus: lethal to Asian small-clawed otters
* *Toxoplasma gondii*: eukaryote parasite, zoonotic, most asymptomatic, outdoor cats are definitive host, many species can be intermediate hosts - transmission by ingestion of meat or water contaminated with cat feces, vertical transmission also possible
	+ Sea otters get exposed by freshwater runoff; cause of death of sea otters in central California
	+ Human population density has been connected to *T condii* rates in sea otters and southern river otters

**Key Points**

* Carcasses collected across the entire state of North Carolina during trapping season
	+ Lower canine tooth used for cementum annuli aging
* Considered positive if either brain or kidney were positive
* No effect of year on prevalence rates for any pathogens
* 1% tested positive for *L interrogans*
* 19% tested positive for parvovirus
	+ Highest in yearlings and southern coastal plain (not detected in the mountains)
	+ Significant influence of age, sex, and location (fur-bearer management unit)
	+ Primary probability of occurrence 19%
	+ Only 10/41 were positive on both kidney and brain
* 24% tested positive for *T gondii*
	+ Highest in females and >4 yos, highest in western state
	+ Significant influence of age and river basin on occurrence of parvovirus
	+ Primary probability of occurrence 24%
	+ Only found in brain tissue samples

**Conclusions**

* *L interrogans* occurred at low levels in the NA river otter in North Carolina
	+ *L interrogans* 1%; parvovirus 19%; *T gondii* 24%
* Higher probability of *T gondii* in female and older otters
* Higher prevalence of parvovirus in yearling otters
* Recommend continuing to test brain and kidney for parvovirus (often detected in only 1)

**Flea parasitism and host survival in a plague-relevant system: theoretical and conservation implications.**

Eads, D. A., Abbott, R. C., Biggins, D. E., & Rocke, T. E.

*The Journal of Wildlife Diseases*, 2020;56(2):378-387.

Plague is a bacterial zoonosis of mammalian hosts and flea vectors. The disease is capable of ravaging rodent populations and transforming ecosystems. Because plague mortality is likely to be predicted by flea parasitism, it is critical to understand vector dynamics. It has been **hypothesized that paltry precipitation and reduced vegetative production predispose herbivorous rodents to malnourishment and flea parasitism, and flea parasitism varies directly with plague mortality**. We evaluated these hypotheses on **five colonies of Utah prairie dogs (UPDs; *Cynomys parvidens*), on the Awapa Plateau, Utah, US, in 2013–16. Ten flea species were identified** among 3,257 fleas from UPDs. These 10 flea species parasitize prairie dogs, mice, rats, voles, ground squirrels, chipmunks, and marmots, all known hosts of plague. The abundance of fleas on individual UPDs (1,198 observations) varied inversely with UPD body condition; fleas were most abundant on lightweight, malnourished UPDs. Flea abundance on UPDs was highest in dry years that were preceded by wet years. Increased precipitation and soil moisture in the prior year might generate humid microclimates in UPD burrows (that could facilitate flea survival and reproduction) and paltry precipitation in the current year could predispose UPDs to malnourishment and flea parasitism. Annual re-encounter rates for UPDs (1,072 observations) were reduced in wetter years preceded by drier years; reduced precipitation and vegetative production might kill UPDs, and increased flea densities in drier years could provide conditions for plague transmission (and UPD mortality) when moisture returns. Re-encounter rates were reduced for UPDs carrying at least one flea compared to UPDs with no detected fleas. These results support the hypothesis that reduced precipitation in the current year predisposes UPDs to flea parasitism. Our results also suggest a link between flea parasitism and UPD mortality. Given documented connections between flea parasitism and plague transmission, our results point toward an effect of flea parasitism on plague-related deaths for individual UPDs, a phenomenon rarely investigated in nature.

**Background**

* Plague: *Yersinia pestis* bacterial zoonosis, flea vector, emerging infectious disease
	+ Threat to prairie dogs - keystone species, federally threatened in Utah
	+ Prevalent at high elevations in Utah - possibly because of diversity of rodents and fleas

**Key Points**

* Live-trapped, anesthetized, collected fleas and tagged; recorded re-encounters in subsequent years
* Carcasses tested for *Y pestis*: Found in 0-3 colonies depending on the year
* Flea species diversity: 10 species *Thrassis francisi* > *Oropsylla tuberculata* > *Oropsylla idahoensis* > *Oropsylla labis*
	+ *O idahoensis* most prevalent at highest elevations
* Flea abundance:
	+ Adults > juveniles
	+ Males > females
	+ Poor BCS > Good BCS
	+ High in dry years preceded by wet years
* Prairie dog survival (re-encounter rates):
	+ Adult > juvenile
	+ Females > Males
	+ Prairie dogs that did not have at least one flea the prior year
	+ Lowest re-encounters in wet years preceded by dry year - when fleas had moderate abundance

**Conclusions**

* Large diversity of flea species on prairie dogs in Utah
	+ Three most prevalent species: *Thrassis francisi*, *Oropsylla idahoensis*, *Oropsylla tuberculata*
* Negative correlation between flea parasitism, prairie dog body condition and prairie dog survival (possibly due to plague)
* Fleas were most abundant in wet-to-dry year and re-encounter was lower in dry-to-wet year
	+ Sequences of dry-to-wet years may facilitate plague transmission
* Encourages the use of flea control measures for plague management

Guthrie, Amanda, et al. "Newly described Toxoplasma gondii strain causes high mortality in red necked wallabies (Macropus rufogriseus) in a zoo." *Journal of Zoo and Wildlife Medicine* 48.3 (2017): 694-702.

**Abstract:** This manuscript describes an outbreak of fatal toxoplasmosis in wallabies. Ten adult red necked wallabies (*Macropus rufogriseus*) were imported from New Zealand to the Virginia Zoo. **Agglutination testing upon admission into quarantine showed all animals to be negative for antibodies to Toxoplasma gondii. Nine of these wallabies died from acute toxoplasmosis within 59–565 (average 224) days after being moved onto exhibit.** *Clinical signs included lethargy, diarrhea, tachypnea, and ataxia that progressed rapidly; death without premonitory signs occurred in one case*. **Histopathologic examination revealed interstitial pneumonia, encephalomyelitis, myositis, enteritis, and myocarditis.** The diagnosis was confirmed through serologic, histopathologic, and polymerase chain reaction (PCR) testing. Multilocus PCR-RFLP (restriction fragment length polymorphism) **genotyping revealed that the first six animals were infected by a previously undiscovered Toxoplasma gondii genotype, designated as ToxoDB PCR-RFLP genotype No. 263.** These six cases survived for an average of 118 days on exhibit before succumbing to toxoplasmosis. The other three wallabies were infected with a Toxoplasma gondii strain of ToxoDB PCR-RFLP genotype No. 4, which is a common strain type circulating in wild animals in North America. These three cases survived for an average of 435 days on exhibit before succumbing to toxoplasmosis. The outbreaks of toxoplasmosis in these wallabies are likely from two different sources. Furthermore, the results highlight Toxoplasma gondii PCR-RFLP genotyping in parasite diagnosis and understanding parasite transmission and potential mitigation procedures.

Key Points:

* *Toxoplasma gondii* – Obligate intracellular, apicomplexan parasites, infects mammals and birds.
	+ New World monkeys, lemurs, Pallas’ cats, slender-tailed meerkats, Australian marsupials particularly susceptible to clinical disease and high mortality.
	+ Rats, cattle, horses, Old World monkeys fairly resistant to clinical infection.
	+ Distributed worldwide – Felids are definitive host.
		- Environmental contamination with TG oocysts from domestic cats and wild felids generally considered main source of infection in herbivorous marsupials.
			* Sporulated oocysts survive for long periods in environment, years.
			* Transmission is fecal oral, consumption of infected meat, and transplacental transfer from mother to fetus.
			* Transplacental or transmammary infection may occur in joeys.
* 9 wallabies imported to a zoo in VA from New Zealand. Died within 565 days (avg 224).
* Clinical signs – ocular, neurologic, respiratory, and GI manifestations or peracute death
	+ **Tachypnea was one of the earliest clinical signs observed and prolonged or increasing tachypnea was a reliable indicator of impending disease progression.**
	+ Attempted therapies in some; prophylactic weekly ponazuril and IV plasma transfusions from wallabies with known high titers, animals died. TMS, clindamycin, atovaquone, supportive care.
	+ One case died without clinical signs, 8 received treatments, all died.
	+ **Dz confirmed through serology, histo, and PCR-RFLP testing.**
		- Serology – Acute infection indicated by high IgG and low IgM or paired increasing IgM.
		- **PCR-RFLP testing was positive in all 9 cases confirming infections.**
			* **Previously undescribed genotype No 263 in 6 cases, common genotype No 4 in NA wildlife in 3 cases.**
* Common clin path findings – Azotemia, elevated muscle and liver enzymes. Hypocalcemia due to renal failure.
* Common necropsy findings – Dark lungs, dark muscles, friable liver, peritoneal effusion.
	+ **In general, pulmonary congestion and edema are the most commonly reported lesions.**
	+ Pneumonia, meningoencephalitis, uveitis, chorioretinitis, myositis, myocarditis, gastroenteritis, hepatitis.
	+ Tachyzoites and bradyzoites may be observed in affected tissues on histo.
* **Common histo findings – Hepatitis, myocarditis, encephalitis, enteritis, pneumonia often with intralesional protozoal tachyzoites and bradyzoites.**
* **Atovaquone – Antimicrobial compound with activity against both tachyzoites and tissue cysts and is synergistic in combination with pyrimethamine and clindamycin or sulfadiazine.**

Take-Home: Newly identified T. gondii train highly pathogenic to red necked wallabies. Wildlife and cats around the zoo likely source of environmental contamination.

Other References:

* Portas TJ. Toxoplasmosis in macropodids: a review. J Zoo Wildl Med. 2010;41:1–6.

Girling, Simon J., et al. "USE OF CLINDAMYCIN IN PALLAS'CATS [OTOCOLOBUS (FELIS) MANUL] TO REDUCE JUVENILE TOXOPLASMOSIS-ASSOCIATED MORTALITY RATES." *Journal of Zoo and Wildlife Medicine* 51.1 (2020): 39-45.

**Abstract:** Pallas' cat [*Otocolobus* (*Felis*) *manul*] experiences a high mortality rate from toxoplasmosis. During the period 2006–2016, the overall mortality rate for this species from all causes during the first year of life was 71.59% in European Association of Zoos and Aquaria institutions, with the most significant infectious cause from systemic toxoplasmosis (20.6%) as confirmed by postmortem examination and histopathology. Clindamycin was used starting in 2014 in two collections that had previously experienced 100% mortality rates by toxoplasmosis in kittens less than one year of age, covering key *Toxoplasma gondii* exposure periods for kittens (*n* = 17) as a prophylactic measure. This protocol resulted in a 67.03% (95% confidence interval 41.76–78.61%) reduction in the first year mortality rate over a two-year period to 5.88% in those animals treated.

* Introduction:
	+ Toxoplasma gondii.
		- Felids definitive host, only spp that pass oocysts.
		- Most felid infections via ingestion of cysts from IM host tissues, or direct ingestion of oocysts.
		- Adult felids typically subclinical.
			* Immune system suppresses infection, shedding typically occurs once in the hosts life.
			* Fatal infection from replication of organism in tissues, resulting in granulomatous inflammation and necrosis.
				+ Lesions most common in lungs, liver, heart, spleen, pancreas, mesenteric LN.
	+ **Palas cat – Indigenous to central Asia.**
		- **Far greater susceptiblility to T. gondii than other felids and chronic seropositivity with ongoing veritical transmission being frequently reported in captive animals.**
			* Low seropositivity < 13% in the wild.
			* **Reported to cause high mortality rates in managed care ~60%.**
				+ **Kittens around weaning, when exposure is greatest and immune system not fully developed.**
				+ Losses also late in gestation or after parturition with infection of kittens postnatally via felid feces, prey items, in utero.
				+ Domestic cats – Lactational transmission reported experimentally,
		- Once clinical, treatment generally unsuccessful.
	+ Most common infectious dz assoc with mortality in annual mort records of European Assoc Zoo and Aquaria = toxo (20.6%) kittens.
	+ Prophylactic medical management regime with clindamycin formulated and tested at two institutions experiencing high kitten mortality.
* M+M:
	+ **Pregnant females at the two collections that were seropositive for toxo were treated with clindamycin 10-12.5 mg/kg PO q12h x 7-10 days preparturition until 3-4 wks postparturition.**
		- **Once eating solid foods, ~3-4wks, kittens administered clindamycin at same dose for 3-4 wk to cover exposure period where previous losses had been reported in both collections.**
		- **Clindamycin capsules opened and powder spread onto food items, target fed to individuals.**
* Results:
	+ **2012-2013 period without clindamycin protocol – 9 born, 9 died.**
	+ **2014-2016 period with clindamycin protocol – 17 born, 1 died.**
* Discussion:
	+ Toxo considered primary pathogen in Pallas’ cats because of their evolutionarily naïve immune systems.
	+ Other dz that impact Pallas cats – Trypanosoma manulis, Cytauxzoon felis, Mycoplasma sp, FHV-1, FIV, FeLV, influenza A.
	+ Periods of high exposure risk for toxo:
		- Late term gestation and around weaning when maternal immunity is waning, permanent immunity has not yet fully developed, and kittens are exposed to additional sources of infection i.e. consumption of wild rodents.
			* Pallas cats also known to die beacause of either immunosuppression or failure to develop immunity through lack of exposure in early life.
	+ Historically used combo tx with sulfadiazine and pyrimethamine for tx of domestic felids.
		- Pyrimethamine may exacerbate negative effects of sulfonamide drugs, leads to anorexia, leukopenia, anemia, hypersalivation.
		- Pyrimethamine alone assoc with myelosuppression in felids, contraindicated.
		- Diclazuril also suggested as preventative therapy for toxo in Pallas cats.
			* Combination with clindamycin may have better success.
			* Formulation of diclazuril in Eu predominantly sold as in-feed mix for poultry and farmed rabbits. Hard to titrate dose for kittens.
	+ Clindamycin = tx of choice for clinical toxoplasmosis in the domestic cat.
		- Long term administration associated with dysbiosis, vomiting, diarrhea, predisposition to Clostridium spp and yeast in GIT.
		- Generally well tolerated unless liver or kidney dz in domestic cats.
		- Clindamycin hindered recovery for a single case of ocular toxo in domestic felids, possibly by inhibition of natural phagocyte function.
	+ TMS has also been used as a prophylactic or preemptive therapy but serious side effects in felids.
	+ Ponazuril shown to reduce mortality rates of toxo in mice. Also toltrazuril metabolite.
		- Both have GI side effects. Excreted metabolite may be toxic to plants, survives for long periods in the environment.
			* Requires dilution or removal of urine and feces of treated animals.
	+ Reduction of exposure risk – Freezing, defrosting fed foods to reduce viability of bradyzoites present in prey items.
	+ Consider housing animals indoors to avoid hunting wild rodents and infection via that route.
		- Deaths have occurred in completely indoor-housed cats later moved to outdoor enclosures.

**Resistance to deltamethrin in prairie dog (Cynomys ludovicianus) fleas in the field and in the laboratory**

JWD 2018 54(4) 745-754

Abstract: Sylvatic plague poses a substantial risk to black-tailed prairie dogs (*Cynomys ludovicianus*) and their obligate predator, the black-footed ferret (*Mustela nigripes*). The effects of plague on prairie dogs and ferrets are mitigated using a deltamethrin pulicide dust that reduces the spread of plague by killing fleas, the vector for the plague bacterium. In portions of Conata Basin, Buffalo Gap National Grassland, and Badlands National Park, South Dakota, US, 0.05% deltamethrin has been infused into prairie dog burrows on an annual basis since 2005. We aimed to determine if fleas (*Oropsylla hirsuta*) in portions of the Conata Basin and Badlands National Park have evolved resistance to deltamethrin. We **assessed flea prevalence, obtained by combing prairie dogs for fleas, as an indirect measure of resistance. Dusting was ineffective in two colonies treated with deltamethrin for >8 yr; flea prevalence rebounded within 1 mo of dusting.** We used a bioassay that exposed fleas to deltamethrin to directly evaluate resistance. Fleas from colonies with >8 yr of exposure to deltamethrin exhibited survival rates that were 15% to 83% higher than fleas from sites that had never been dusted. All fleas were paralyzed or dead after 55 min. After removal from deltamethrin, 30% of fleas from the dusted colonies recovered, compared with 1% of fleas from the not-dusted sites. Thus, **deltamethrin paralyzed fleas from colonies with long-term exposure to deltamethrin, but a substantial number of those fleas was resistant and recovered.** Flea collections from live-trapped prairie dogs in Thunder Basin National Grassland, Wyoming, US, suggest that, **in some cases, fleas might begin to develop a moderate level of resistance to deltamethrin after 5–6 yr of annual treatments.** Restoration of black-footed ferrets and prairie dogs will rely on an adaptive, integrative approach to plague management, for instance involving the use of vaccines and rotating applications of insecticidal products with different active ingredients.

* Key points:
	+ Deltamethrin pulicide dust reduces spread of plague by killing fleas.
	+ Has been used in prairie dog burrows annually in South Dakota – Badlands NP and Conata Basin.
	+ Study to determine if fleas in these areas have evolved resistance.
	+ Assessed flea prevalence as indirect measure of resistance.
	+ Dusting ineffective in two colonies with flea rebound within 1 month of dusting.
	+ Fleas from dusted colonies exhibited survival rates higher than fleas from sites that had never been dusted.
	+ Fleas might begin to develop a moderate level of resistance to deltamethrin after several years (5-6) annual treatments.
	+ Integrative approach to plague management using vaccines, rotating applications of insecticidal products with different active ingredients recommended.

**TRANSMISSION DYNAMICS OF TOXOPLASMA GONDII IN ARCTIC FOXES (VULPES LAGOPUS): A LONG-TERM MARK-RECAPTURE SEROLOGIC STUDY AT KARRAK LAKE, NUNAVUT, CANADA**

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**ABSTRACT**: Transmission dynamics of Toxoplasma gondii, a parasite of importance for wildlife and human health, are enigmatic in the Arctic tundra, where free-ranging wild and domestic felid definitive hosts are absent and rarely observed, respectively. Through a multiyear mark-recapture study (2011– 17), serosurveillance was conducted to investigate transmission of T. gondii in Arctic foxes (Vulpes lagopus) in the Karrak Lake region, Nunavut, Canada. Sera from adult foxes and fox pups were tested for antibodies to T. gondii by using serologic methods, including the indirect fluorescent antibody test, direct agglutination test, and modified agglutination test. The **overall seroprevalence was 39% in adults and 17% in pups**. **Mature foxes were more likely to be exposed** (seroconvert) than young foxes (less than 1 yr old), with the **highest level of seroprevalence in mid-aged foxes (2-4 yr old)**. Pups in two different litters were seropositive on emergence from the den, around 5 wk old, which could have been due to passive transfer of maternal antibody or vertical transmission of T. gondii from mother to offspring. The seropositive pups were born of seropositive mothers that were also seropositive the year before they gave birth, **suggesting that vertical transmission might not be limited to litters from mothers exposed to T. gondii for the first time in pregnancy**. **All recaptured seropositive foxes remained seropositive** on subsequent captures, suggesting that antibodies persist or foxes are constantly re-exposed or a combination of both. The results of this study provided insights into how foxes were likely exposed to T. gondii, the dynamics of antibody persistence and immune response, and how the parasite was maintained in a terrestrial Arctic ecosystem in the absence of felid definitive hosts.

Summary:

* Toxoplasma gondii is a protozoan that infects birds and mammals
	+ Often asymptomatic, if immunocompromised or pregnant 🡪 neurologic, ocular, reproductive problems
	+ Felid definitive hosts
	+ Intermediate host by ingesting oocysts in felid feces, tissue cysts in prey, or vertical transmission
* High mortality in captive fox pups from experimentally infected mothers
* Vertical transmission or ingestion of tissue cysts from migratory birds are suspected in artic foxes
* N = 55, trapped by box traps, indirect fluorescent antibody test, direct agglutination, or modified agglutination
	+ Considered positive if both agglutination and IFAT were positive
* Adult higher seroprevalence (39%) >> pups (17%)
	+ **Mid-aged foxes (2-4yo) highest level of seroprevalence**
* Pups as young as **5 weeks old** seropositive
	+ Indicates maternal antibody passive transfer or vertical transmission
	+ **Some seropositive females had seronegative pups 🡪** loss of maternal antibody likely explanation
* Four recaptured foxes seroconverted to seropositive 🡪 remained seropositive
	+ Antibody thought to persist for life due to antigenic stimulation from persistent tissue cysts

**Take home**: Toxoplasma seroprevalence is more likely in adult artic foxes with vertical transmission and maternal antibody passive transfer likely