Toxoplasmosis in New Zealand: trying to fill the knowledge gaps around transmission to livestock and native wildlife

Chris N. Niebuhr



Toxoplasmosis

- Toxoplasma gondii
- Protozoan parasite
- Infects most warm-blooded animals
 - Humans
 - Domestic/agricultural animals
 - Wildlife
- Complex lifecycle

Toxoplasmosis in wildlife

Type X strains of *Toxoplasma gondii* are virulent for southern sea otters (*Enhydra lutris nereis*) and present in felids from nearby watersheds

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Last week, ZEALANDIA welcomed home a Little Spotted Kiwi that was successfully treated for toxoplasmosis. After a 4.5 month stay with the vet team at <u>Wellington Zoo</u> this little bird is settling back in to life at the Sanctuary.





Contents lists available at SciVerse ScienceDirect

Veterinary Parasitology



journal homepage: www.elsevier.com/locate/vetpar

An atypical genotype of *Toxoplasma gondii* as a cause of mortality in Hector's dolphins (*Cephalorhynchus hectori*)

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Toxoplasmosis on sheep farms

- Significant cause of abortion in sheep, goats, and pigs
- The timing of infection is critical in terms of impacts on lambs
 - Before pregnancy: transient fever (ewe)
 - Early pregnancy: likely death (lamb)
 - Late pregnancy: possible death or weak at birth (lamb)
- Following infection, ewes develop robust immunity
- Source of infection in sheep is contaminated pasture, food supplies and water
- Fields treated with manure or hay from farm buildings where cats live and defaecate pose a significant risk of disease transmission



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- Hawke's Bay region estimates (Walker 2014)
 - Potential loss of lambs due to toxoplasmosis (up to \$18 million)
 - The cost to vaccinate all replacements* in the region (~\$1 million)
 - *The vaccine is administered to replacement ewes each year



Toxoplasma gondii lifecycle



Fig. 1. The life cycle of Toxoplasma gondii; printed with permission [®]Marcia Hartsock MA, CMI.

Transmission

- *T. gondii* infections in accidental hosts are primarily a result of transmission from the environment
- Therefore, to reduce oocyst loading in the environment, we should manage cat populations







Stopping transmission

- *T. gondii* infections in accidental hosts are primarily a result of transmission from the environment
- Therefore, to reduce oocyst loading in the environment, we should manage cat populations







Is it that simple?

Preliminary evaluation of the effect of predator control on *T. gondii* seroprevalence in sheep

as part of the Cape to City Programme, Hawke's Bay Regional Council

- Seroprevalence of one-year-old ewes was compared prior to, and after commencement of, large-scale control of predators, including feral cats.
- Sheep seroprevalence was highly variable among sites and over time.
- A knockdown of feral cat abundance was observed, but did not last.
- Conclusions: Likely more targeted and/or longterm control needed





Environmental persistence

- A single cat may shed >20 million oocysts into the environment
- Oocysts can remain infective in soil and fresh water for at least 1 year, and in seawater up to 2 years





Cat definitive hosts

- Oocyst shedding typically occurring early in life
- Unowned cats (stray, feral)
 - have shorter life spans (a few years, compared with 15+ years for pet cats)
 - breed more rapidly
 - Therefore, feral cats would be priority for control

(Gillies & Fitzgerald 2005)





Intermediate hosts

- Maintain infections for multiple generations (vertical transmission)
- Infection in cats occurs at a much higher rate from predation on infected intermediate hosts than from ingestion of oocysts from environment
- Varying predation rates (unowned vs owned cats)





Disease Management



Is it that simple?

Disease Management



Is it that simple?



Fig. 1. The life cycle of Toxoplasma gondii; printed with permission [©]Marcia Hartsock MA, CMI.

Epidemiological modelling



- Several modelling studies have investigated impact of different management practises on toxoplasmosis in farm systems.
- Turner at al. (2013)
 - differential equation model that explores
 - transmission pathways
 - o potential control mechanisms



Fig. 2. A schematic representation of the transmission of *T. gondii* between the definitive host (cat), intermediate host (mouse), and the environment.



$$\begin{cases} \dot{N}_c = r_c N_c \left(1 - \frac{N_c}{K_c}\right) \\ \dot{S}_m = b_m S_m + b_m \left(1 - p_m\right) I_m \\ - \left(m_m + \left(b_m - m_m\right) \frac{N_m}{K_m}\right) S_m - a N_c S_m - H S_m \\ \dot{I}_m = b_m p_m I_m - \left(m_m + \nu m_m + \left(b_m - m_m\right) \frac{N_m}{K_m}\right) I_m \\ - \theta a N_c I_m - H I_m. \end{cases}$$

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Epidemiological modelling

• Carrying capacity of cats



Environmental infection in cats

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Epidemiological modelling

- Carrying capacity of cats
- Predation rates



Environmental infection in cats

The predator-prey cycle

Epidemiological modelling

- Carrying capacity of cats
- Predation rates
- Vertical transmission in intermediate hosts



Environmental infection in cats







Vertical transmission in mice

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Epidemiological modelling

- Environmental risk to cats increases as the carrying capacity (K) of cats is increased
 - e.g. for densely populated regions, no other life cycle necessary for infection to persist



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- For moderately high predation, the predator-prey cycle is all that is necessary to sustain infection



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Epidemiological modelling

- Environmental risk to cats increases as the carrying capacity (K) of cats is increased
 - e.g. for densely populated regions, no other life cycle necessary for infection to persist
- For moderately high predation, the predator-prey cycle is all that is necessary to sustain infection
- For sparsely populated regions with low predator-prey life cycle (e.g. urban areas, following control), vertical transmission in IH is a deciding factor for disease persistence

- "a parasite struggling for survival would benefit most by increasing vertical transmission in mice"







May 21

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Disease modelling

- Turner at al. (2013) modelling simulation example: (discussed in Tompkins 2014)
 - Controlling disease in 90% of the cat population each year is predicted to result in local disease eradication.



Fig. 7. Control program of constant rate cat vaccination and/or mouse harvesting. The dashed line represents the maximum allowable harvesting rate H_a . The virulence to mice is $\nu = 0.3$. All other parameters take the default values in Table 1.

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Disease modelling

- Turner at al. (2013) modelling simulation example: (discussed in Tompkins 2014)
 - Controlling disease in 90% of the cat population each year is predicted to result in local disease eradication.
 - With as little as 6% reductions in mouse populations each year, the amount of yearly cat control needed to result in predicted disease eradication was reduced to 20%
 - In other words, the model suggests less-intensive cat control is necessary if mouse populations are reduced concurrently



Fig. 7. Control program of constant rate cat vaccination and/or mouse harvesting. The dashed line represents the maximum allowable harvesting rate H_a . The virulence to mice is v = 0.3. All other parameters take the default values in Table 1.

Create a NZ specific model

- transmission pathways
- potential control mechanisms



Figure: Emmanuelle Gilot-Fromont et al. (2012)



Fig. 2. A schematic representation of the transmission of *T. gondii* between the definitive host (cat), intermediate host (mouse), and the environment.

$$\begin{cases} \dot{S}_{c} = b_{c}N_{c} - \left(m_{c} + (b_{c} - m_{c})\frac{N_{c}}{K_{c}}\right)S_{c} \\ -\beta_{c}ES_{c} - g(\nu)\theta aS_{c}I_{m} - VS_{c} \\ \dot{I}_{c} = -\left(m_{c} + (b_{c} - m_{c})\frac{N_{c}}{K_{c}}\right)I_{c} + \beta_{c}ES_{c} \\ +g(\nu)\theta aS_{c}I_{m} - \gamma I_{c} \\ \dot{R}_{c} = -\left(m_{c} + (b_{c} - m_{c})\frac{N_{c}}{K_{c}}\right)R_{c} + \gamma I_{c} + VS_{c} \\ \dot{E} = \lambda I_{c} - d_{0}E \\ \dot{S}_{m} = b_{m}S_{m} + b_{m}(1 - p_{m})I_{m} \\ -\left(m_{m} + (b_{m} - m_{m})\frac{N_{m}}{K_{m}}\right)S_{m} - \beta_{m}ES_{m} - aN_{c}S_{m} - HS_{m} \\ \dot{I}_{m} = b_{m}p_{m}I_{m} - \left(m_{m} + \nu m_{m} + (b_{m} - m_{m})\frac{N_{m}}{K_{m}}\right)I_{m} \\ +\beta_{m}ES_{m} - \theta aN_{c}I_{m} - HI_{m}. \end{cases}$$

$$\begin{cases} \dot{N}_{c} = r_{c}N_{c}\left(1 - \frac{N_{c}}{K_{c}}\right) \\ \dot{S}_{m} = b_{m}S_{m} + b_{m}\left(1 - p_{m}\right)I_{m} \\ -\left(m_{m} + (b_{m} - m_{m})\frac{N_{m}}{K_{m}}\right)S_{m} - aN_{c}S_{m} - HS_{m} \\ \dot{I}_{m} = b_{m}p_{m}I_{m} - \left(m_{m} + \nu m_{m} + (b_{m} - m_{m})\frac{N_{m}}{K_{m}}\right)I_{m} \\ -\theta aN_{c}I_{m} - HI_{m}. \end{cases}$$

M. Turner et al. / Theoretical Population Biology 86 (2013)

Future plans for Toxoplasmosis studies

- Role of intermediate hosts in transmission dynamics of *T. gondii*
 - Rodents (mice, multiple rat species)
 - Other (e.g. rabbits?)
- Geographic and host distribution of *T. gondii* genotypes in NZ
- Research investigating potential management regimes to reduce (or eliminate) toxoplasmosis impacts
 - Bioeconomics: cost/benefit of wildlife control on impact to livestock
- Infection status of other wildlife in NZ
 - Wild game animals; risk to humans?
 - Acting as sentinel species?





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Questions?



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