Zoonotic and economically significant pathogens of peri-urban wild dogs across north-eastern New South Wales and south-eastern Queensland, Australia

Lana Harriott [iD] [A,B,E], *Matthew Gentle* [B], *Rebecca Traub* [C], *Ricardo J. Soares Magalhães* [A,D] *and Rowland Cobbold* [A]

[A]School of Veterinary Science, University of Queensland, Gatton, Qld 4343, Australia.

[B]Pest Animal Research Centre, Biosecurity Queensland, Department of Agriculture and Fisheries, 203 Tor Street, Toowoomba, Qld 4350, Australia.

[C]Faculty of Veterinary and Agricultural Science, University of Melbourne, Parkville, Vic. 3010, Australia.

[D]Child Health Research Centre, University of Queensland, South Brisbane, Qld 4101, Australia.

[E]Corresponding author. Email: Lana.Harriott@daf.qld.gov.au

Abstract

Context. Peri-urban wild dogs are known to reside within high-risk and densely populated regions and are capable of harbouring a variety of zoonotic pathogens. Despite recognising the potential of peri-urban wild dogs to carry zoonotic pathogens, limited prevalence data are currently available to assist in understanding the potential risks that peri-urban wild dogs pose within developed communities.

Aims. The aim of the present research was to establish the current status of key zoonotic and economically significant pathogens in peri-urban wild dogs.

Methods. Two hundred and one peri-urban wild dog cadavers were collected from south-eastern Queensland and northern New South Wales. In addition, whole blood, serum and faecal samples were also collected. Pathogens were identified through several morphological, microbiological and molecular methods.

Key results. Helminth parasites were detected within 79.6% of peri-urban wild dogs; *Echinococcus granulosus* was the most common pathogen, with adult worms being detected within $50.7 \pm 6.9\%$ of intestines, followed by *Spirometra erinacei* $(36.6 \pm 6.4\%)$; hookworms, including *Ancylostoma caninum* and *Uncinaria stenocephala* $(28.8 \pm 7.1\%)$; *Toxocara canis* $(5.4 \pm 3.1\%)$ and *Taenia* spp., including *T. serialis* and *T. pisiformis* $(4.5 \pm 2.8\%)$. Bacterial pathogens detected included methicillin-resistant *Escherichia coli* $(20.0 \pm 10.1\%)$, *Salmonella* spp. $(3.7 \pm 4.0\%)$ and methicillin-sensitive *Staphylococcus aureus* $(3.3 \pm 2.7\%)$.

Conclusions. The present study is the most comprehensive investigation of zoonotic pathogen carriage in peri-urban wild dogs in Australia. Parasitic infections in peri-urban wild dogs are common, with tapeworms representing the majority of intestinal pathogens. Important zoonotic bacterial pathogens are carried by peri-urban wild dogs, although at a much lower prevalence than are parasites.

Implications. The presence of these pathogens in free-ranging peri-urban dog populations suggests a strong potential for public health risk, most notably from *E. granulosus.* These data are inherently important as baseline information, which is essential to guide risk-based management of peri-urban wild dog impacts.

Additional keywords: disease, dingoes, parasites, bacteria, *Echinococcus granulosus*, free-ranging dogs.

Introduction

Several wildlife species around the world have successfully adapted to peri-urban and urban habitats (Koenig *et al.* 2001; Prange *et al.* 2003; Statham and Statham 1997). Peri-urban areas are commonly classified as a transitional zone between urban and rural landscapes (Low Choy *et al.* 2007). In Australia, wild dogs (*Canis familiaris*) are free ranging across the majority of the mainland (Fleming *et al.* 2014), including within urban and peri-

urban regions (Allen *et al.* 2013; McNeill *et al.* 2016). Peri-urban wild dogs and other urban carnivore species are known to utilise much smaller home-ranges than do their rural counterparts (Bateman and Fleming 2012; McNeill *et al.* 2016), which suggests that peri-urban areas have a higher resource abundance (Newsome *et al.* 2013). Importantly, peri-urban wild dogs can remain within 700 m (Allen *et al.* 2013) or 1000 m (McNeill *et al.* 2016) of houses or buildings at all

times, as well as frequently utilise and traverse public environments such as recreational ovals, parklands, school yards and residential backyards. This is concerning not only for potential human–wildlife conflicts, including attacks on the public, their pets, livestock animals, and native wildlife, but also introduces the potential for transmission of zoonotic pathogens.

The extent to which peri-urban wild dogs harbour zoonotic pathogens is currently unknown. Previous research has largely focused on domestic household, rural working or pound populations of animals (Palmer *et al.* 2008; Jenkins *et al.* 2014*a*). For many of the common pathogens, the epidemiology, modes of transmission, routes of infection, parasite life cycles and pathogenicity of their infections are well described (Deplazes *et al.* 2011; Chen *et al.* 2012). However, the significance of infection in domestic canines is often reduced by the fact that most animals are treated with broad-scale anthelmintic therapy. In Australia, this is also assisted by the requirement and facilitation of owners to remove any faeces left by their dog in public-use areas. As a result, contamination of environmental sources with infectious pathogens is more likely to be attributed to untreated wild populations of animals. Therefore, peri-urban wild dogs could contribute towards the transmission of zoonotic pathogens to humans, either via direct transmission or enhanced epizootic cycling.

Peri-urban regions play a significant role in agricultural production around Australia (Choy and Buxton 2013). Impacts from wild dogs carrying zoonotic pathogens in agricultural settings may result in reduced production levels, morbidity and mortality of livestock and devaluing damaged product at slaughter. Pathogens may be significant to both humans and livestock, but many are specific to each group. Therefore, it remains important to consider both the human-health aspects and potential economic impacts on the livestock industry from peri-urban wild dogs.

The aim of the present study was to investigate the carriage of pathogens by peri-urban wild dogs that are a risk to public health and to livestock industries. Beyond providing valuable estimates of infection burden to assist in evaluating risk, this knowledge is essential in supporting epidemiological studies to inform strategies for management.

Materials and methods

Study population

In all, 201 whole wild-dog carcasses were collected between August 2012 and May 2015 (Harriott 2018). Males and females were equally represented within the dataset at 49% and 51% respectively. Peri-urban wild dogs provided for the present study were trapped either as a part of routine council control programs or private pest-management programs. We classify peri-urban regions as a transitional zone adjacent to, and influenced by, urban centres (Low Choy *et al.* 2007). They are classified neither as urban nor rural and are comprised of a fragmented mixture of rural, residential and commercial land uses. Trapping locations were influenced by public complaints or active management plans for control of feral animals. Wild dogs were humanely caught with foothold traps. They were captured from south-eastern Queensland (SEQ), a small section of northern New South Wales, and regions just north of SEQ within close

proximity to human populations (Fig. 1). Human population density was sourced from the Center for International Earth Science Information Network (CIESIN), Columbia University, 2016; gridded population of the world, version 4 (GPWv4): population density; Palisades, NY: NASA Socioeconomic Data and Applications Center (SEDAC), at http://doi.org/10.7927/H4NP22DQ, accessed 5 November 2018. The present study was completed as part of a postgraduate research program (Harriott 2018) and approved by the University of Queensland Animal Ethics Committee (Approval number SVS/145/13).

Sample collection

Exposure to and identification of zoonotic pathogens was conducted using blood and faecal samples, combined with examination of intestinal tracts. Blood samples were collected via cardiac puncture and transferred to EDTA (EDTA) and serum-separator (SST) vials (Becton, Dickinson and Co., Sydney, NSW, Australia) and kept cool. Blood vials were kept in a portable fridge or on ice (if possible) until fieldwork was completed. EDTA samples were immediately placed in the freezer and SST tubes were refrigerated at 4°C until the serum had separated. Once separated, the supernatant was collected off the SST tubes and stored frozen. Faecal samples were collected from the rectum and stored in three separate tubes, each one containing 10% neutral buffered formalin (NBF), 2.5% potassium dichromate and 20% glycerol. Faeces in formalin and potassium dichromate were stored at room temperature (~23°C) and faeces in glycerol were stored at –80°C as soon as possible. After samples had been collected, wild dog cadavers were placed into individual large body bags and frozen, within reasonable time (on the same morning as capture), at –18°C for later necropsy. At necropsy, the entire small intestine was removed from all dogs ($n = 201$) and its contents were flushed with water and expelled into a dish. The intestinal contents were then transferred into a disposable container for transport to the laboratory and kept refrigerated (3°C) until examination within 24 h. The intestinal tract was then excised longitudinally and the mucosa was macroscopically examined for the presence of hookworms or other parasites. The upper jaw was removed for the purpose of collecting the two canine teeth to age the wild dogs for future studies. Throughout this process, the nasal cavities were assessed for the presence of tongue worm (*Linguatula serrata*).

Parasite isolation and identification

Intestinal contents were filtered initially through a 918-µm sieve and washed into a beaker to isolate larger parasites. Parasites were placed into a Petri dish for initial identification, and then stored in 70% ethanol. All adult helminths were identified on the basis of morphological characteristics (Bowman 2013). Contents of the beaker were then washed through a 200-µm-mesh sieve. All items trapped on the mesh were washed into a clean beaker and the total volume was adjusted to 1000 mL. Contents were stirred and two 50-mL subsamples were collected. Each subsample was individually examined in small amounts under a dissecting microscope in a Petri dish marked with ~1-cm squares. Parasites other than those discovered in the coarse-sieving stage were identified. For *Echinococcus granulosus*,
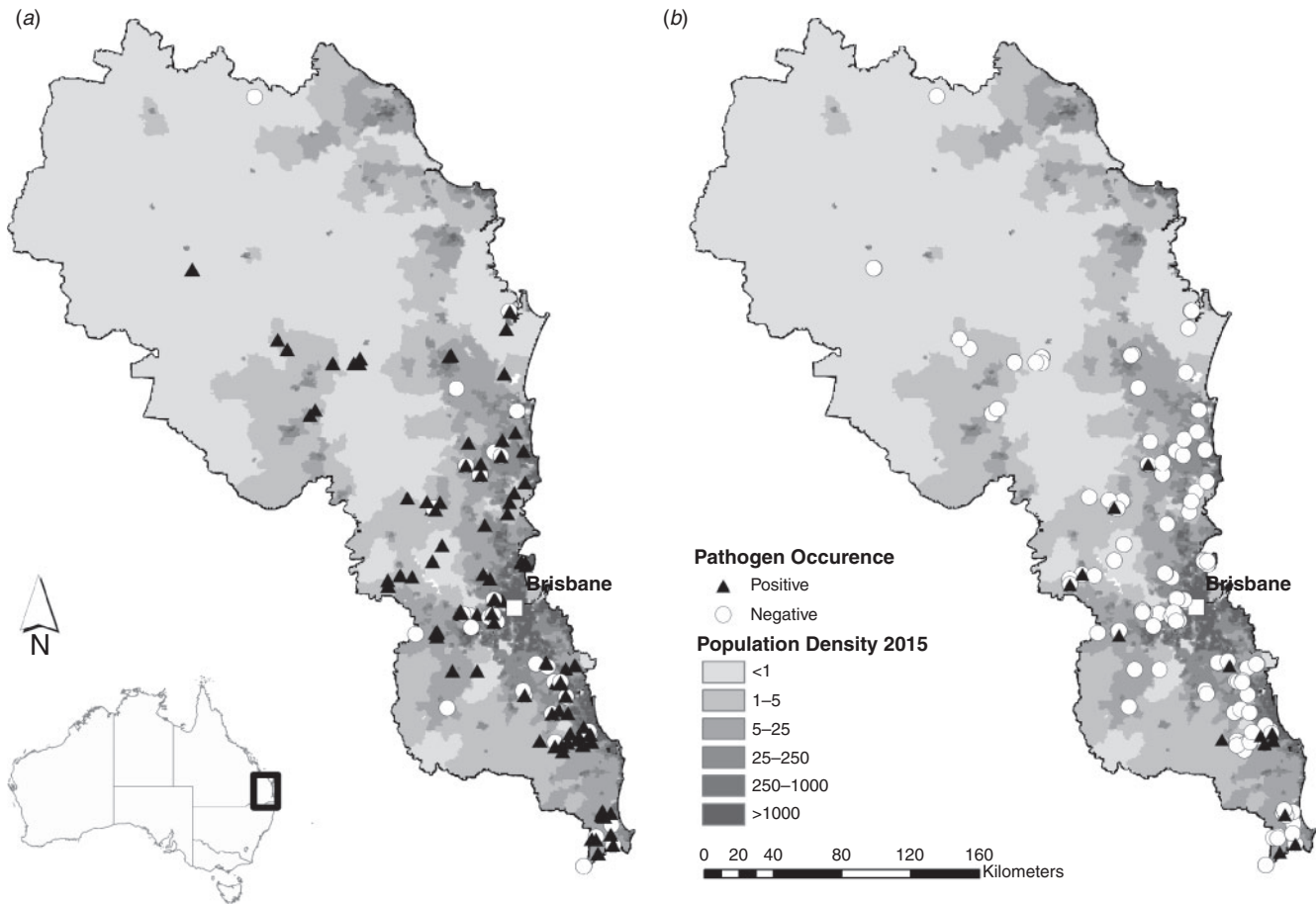
Fig. 1. Human population density and the geographical locations of the 201 trapped peri-urban wild dogs with (▲) or without (○) the presence of (*a*) parasitic pathogens and (*b*) bacterial pathogens. Gridded population density is in the scale of persons per square kilometre.

worms were counted for two 50-mL subsamples as previously described by Jenkins *et al*. (2008), so as to provide an estimate of the total *E. granulosus* worm burden for each dog. Faecal samples in 10% formalin (*n* = 156) were washed and were subject to sodium nitrate (specific gravity (SG) 1.20; Inpankaew *et al*. 2014) and zinc sulfate (SG1.18; Faust *et al*. 1938) flotation for egg and/or oocyte identification and quantification. The entire coverslip was examined under a light microscope at ×10 and ×40 magnification and species of eggs and/or oocytes were noted.

Genomic DNA extraction from faecal samples

Aliquots of ~2 g of faeces (from the potassium dichromate sample) were washed twice with phosphate-buffered saline and centrifuged at 2000*g* for 3 min at room temperature. A 200-mg subsample of faeces was transferred to an empty 2-mL tube with ~1 g of silica/zirconia 0.5-mm beads (Daintree Scientific, St Helens, Tas., Australia) and 370 µL of Mo Bio Powerbead solution from the PowerSoil DNA isolation kit (Mo Bio Laboratories Inc., Carlsbad, California, USA) The remaining DNA-extraction protocol was performed as per the Mo Bio protocol (https://mobio.com/media/wysiwyg/pdfs/protocols/12888.pdf, accessed 1 May 2017) with minor modifications. A volume of 50 µL of C6 solution was used at

Step 20 to increase DNA concentration. DNA concentration was measured using a nano-drop 1000 spectrophotometer (Thermo Fisher, Waltham, MA, USA).

Genomic DNA extraction from blood samples

DNA was extracted from whole-blood samples collected in EDTA tubes. The DNeasy Blood and tissue kit (Qiagen, Hilden, Germany) was used following the manufacturer's protocol, with only minor modifications. The blood volume was increased to 200 µL and the final elution volume was decreased to 50 µL for more concentrated DNA. Total DNA concentration was measured and adjusted (if needed) to ~50 ng per 1 µL before polymerase chain reaction (PCR).

Genomic DNA extraction from worms

DNA was extracted from adult worms of *Taenia* spp., and morphologically identified according to Bowman (2013). The DNeasy Blood and Tissue kit (Qiagen) was used following the manufacturer's protocol with minor modifications. Approximately 0.25 g of worm tissue was incubated at 56°C with 180 µL of ATL buffer and 20 µL of proteinase K until completely lysed. The final elution volume was decreased to 50 µL. Total DNA concentration was measured and adjusted (if needed) to ~50 ng per 1 µL before the PCR.

Polymerase chain reaction (PCR)

All DNA samples were screened for eukaryotic DNA to ensure the presence of amplifiable DNA. Samples were tested using universal primers 18SEUDIR 5′-TCTGCCCTATCAACTTTC GATGG-3′ and 18SEUINV 5′-TAATTTGCGCGCCTGCTG-3′ for amplification of a 140-bp fragment of the nuclear 18S gene from eukaryotic DNA (Fajardo *et al.* 2008). The annealing temperature was modified to 60°C (Wang *et al.* 2013).

Samples were tested for hookworm by using previously published protocols to amplify a section of the internal transcribed spacer (ITS) regions of *A. caninum*, *A. ceylanicum*, *A. braziliense* and *U. stenocephala* (Traub *et al.* 2004, 2007). Amplified ITS PCR products were subjected to direct digestions as described in Palmer *et al.* (2007), so as to differentiate among species. PCR products that were too faint to visualise using restriction fragment-length polymorphism were subjected to purification and bidirectional DNA sequencing for species confirmation by the Animal Genetics Laboratory (School of Veterinary Science, University of Queensland). DNA sequences were analysed on Finch TV 1.4.0 (Geospiza Inc. Seattle, WA, USA) and compared with published sequence data on GenBank, using the basic logical alignment search tool (BLAST) (http://blast.ncbi.nlm.nih.gov/Blast.cgi, accessed 27 February 2019). To test for *Taenia* spp., we used a singleplex PCR protocol to amplify a 350-bp region of the *Cox*1 gene as published by Ng-Nguyen *et al.* (2017). DNA sequences were analysed on Finch TV 1.4.0 (Geospiza Inc.) and compared with published sequence data on GenBank using BLAST. Whole-blood samples were tested for spotted fever group rickettsia by amplification of a 297-bp region of the outer membrane protein B (ompB) gene of spotted fever group *Rickettsia* according to Hii *et al.* (2011). To test for *Neospora caninum*, we used previously published primers and the cycling protocol of Fish *et al.* (2007).

Isolation of bacterial species

To test for *Salmonella* spp., faecal samples had been stored in 20% glycerol at –80°C until testing. Faeces were swabbed and suspended in 5 mL of Rappaport–Vassiliadis (RV) broth (Oxoid, Altrincham, Cheshire, England) and incubated at 37°C for 18–24 h. A total of 10 µL of the enrichment broth was aspirated and streaked onto brilliant green sulfa agar (BGA) and incubated at 37°C for 18–24 h. Isolates represented by red–pink–white colonies surrounded by a red/pink zone were subcultured onto sheep blood agar (SBA) and MacConkey agar (MCA). All suspect *Salmonella* organisms were subject to a microbact™ test (Oxoid). Identification of serotype was conducted by Queensland Health Forensic and Scientific Services, Brisbane, Queensland. To test for *Campylobacter* spp., faecal samples stored in 20% glycerol at –80°C were transferred using a sterile dry swab, using contact methods that aimed to maximise sample exposure on the basis of the nature of the respective sample, onto modified charcoal cefoperazone deoxycholate agar (Oxoid) and incubated in an atmosphere consisting of 5–6% oxygen, 10% carbon dioxide and 84–85% nitrogen at 41.5°C for 48 h. Detection methods for *Escherichia coli* focussed on the identification of multi-drug resistant strains, as indicators for resistant Gram-negative bacteria. Faecal samples were partially defrosted before using a dry swab and streaking onto ampicillin–MacConkey (AMP–MCA) plates (Oxoid) and incubated at 37°C for 18–24 h. Three suspect colonies were selected and plated individually onto SBA plates, which were again incubated for 18–24 h at 37°C. Growth on SBA plates were subject to rapID™ Spot Indole (Remel, San Diego, CA, USA) and pyrrolidonyl arylamidase (PYR) testing to confirm suspect colonies as *E. coli*. Confirmed *E. coli* colonies were subjected to sensitivity testing. Three to four colonies, per sample, confirmed as *E. coli* were selected and grown in 4 mL of Mueller–Hinton Broth at 37°C for 3–4 h or until denser than 0.5 McFarland standard. Incubated broth was added to 4 mL of saline until equivalent to a 0.5 McFarland standard. Two drops of the solution were dropped onto Mueller–Hinton agar (MH) and streaked evenly across the surface of the plate. Another drop was placed onto SBA for purity testing. Antimicrobial disks were stamped onto the streaked MH plate. Both the MH and SBA plates were incubated at 37°C for 18–24 h. Chosen antimicrobials are commonly used in human or veterinary medicine and included cephalothin (KF), amoxicillin clavulanate 2 : 1 (AMC), ampicillin (AMP), cefoxitin (FOX), ceftazidime (CAZ), ciprofloxacin (CIP), trimethoprim-sulfamethoxazole (SXT), tetracycline (TE), amikacin (AK), gentamicin (CN), tulathromycin (TUL) and florfenicol (FFC) (Oxoid). To test for both methicillin-resistant and -sensitive *Staphylococcus aureus* (MRSA and MSSA), nasal swabs were collected at necropsy and were suspended in 1.5–2.0 mL of Mueller–Hinton broth containing 20% (v/v) glycerol and stored at –80°C. A total of 100 µL of the broth was aspirated and added to 2.0 mL of Mueller–Hinton broth containing 6.5% (w/v) sodium chloride (NaCl) and incubated at 37°C for 18–24 h. To test for MSSA, 10 µL of broth was aspirated and streaked onto mannitol salt agar containing 6.5% (w/v) NaCl. Plates were incubated aerobically at 37°C for 18–24 h. Suspect MSSA colonies were subjected to a Gram stain, catalase test and staph latex-agglutination test for confirmation. To test for MRSA, 10 µL of broth was aspirated and added to 2–5 mL of tryptic soy broth containing 2.5% (w/v) NaCl, 3.5 mg L^{-1} cefoxitin and 20 mg L^{-1} aztreonam and incubated at 37°C for 18–24 h. A total of 10 µL of the selective enrichment broth was aspirated and streaked onto brilliance MRSA agar.

Serology

Serum *Brucella suis* testing was performed by Biosecurity Sciences Laboratory, Brisbane (Queensland). Samples underwent an initial rose bengal (RB) test with brucellosis antigen (IDEXX, Westbrook, ME, USA). Positive samples were confirmed using compliment fixation (CF) testing.

Analysis

The prevalence of pathogens and 95% confidence intervals (CI) were calculated for all pathogens. The overall hookworm prevalence was calculated utilising more than one method of detection. The total number of positive samples across any method was divided by the total number of samples tested within all methods (excluding samples that were negative at intestinal sampling and corresponding faecal samples were not available for flotation or PCR to confirm).

Table 1.   Number (and percentage) of wild dogs testing positive for parasitic pathogens for each sampling technique
PCR, polymerase chain reaction; n.a., not available

Parasite	Samples positive in intestine ($n = 201$), n (% ± CI)	Samples positive by faecal floatation ($n = 156$), n (% ± CI)	Samples positive by PCR ($n = 82$), n (% ± CI)	Samples positive on the basis of combined methods (% ± CI)
Echinococcus granulosus	102 (50.7 ± 6.9)[A]	7 (4.3 ± 3.1)	n.a.	50.7 ± 6.9
Hookworms	11 (5.5 ± 3.1)	28 (17.2 ± 5.9)	32 (39.0 ± 10.5)[A]	28.8 ± 7.1
Ancylostoma caninum			32 (39.0)	
Uncinaria stenocephala			4 (4.9)	
Spirometra erinacei	73 (36.3 ± 6.4)[A]	42 (25.7 ± 6.8)	n.a.	36.6 ± 6.4
Taenia spp.	9 (4.5 ± 2.8)[A]	7 (4.3 ± 3.1)	–	4.5 ± 2.8
T. serialis			6 (3.0)[B]	
T. pisiformis			1 (0.5)[B]	
Taenia spp.			2 (1.0)[B]	
Toxocara canis	11 (5.4 ± 3.1)[A]	7 (4.3)	n.a.	5.4

[A]Gold standard testing method.
[B]PCR conducted on DNA from the adult worm, not faecal sample.

Results

Zoonotic parasites

A total of 79.6% of dogs sampled had a parasitic infection and, of those, 28.1% were infected with multiple species (Table 1). Mixed infections consisted most commonly of *E. granulosus* ($n = 40$) and *S. erinacei* ($n = 29$). Two wild dogs were infected with three different parasites, and another two wild dogs were infected with four species of intestinal parasites. *Echinococcus granulosus* was the most common parasite, detected in 50.7% (CI 43.8–57.7) of all dogs (results not shown for *E. granulosus* intensity), followed by *S. erinacei* and *A. caninum* at 36.3% (CI 29.7–43.0) and 28.8% (CI 21.7–36.0), respectively. Adult hookworms were identified within the intestinal tracts of 5.5% of the wild dogs at necropsy. Faecal-floatation methods increased the prevalence to 17.2% and molecular methods further increased prevalence to 39%. All samples that were positive at faecal floatation and had a corresponding sample for PCR were also positive at PCR. Several samples that were negative at faecal floatation returned positive at PCR. Taking into consideration the variations in sample size across the different testing methods, the overall combined hookworm prevalence was 28.8%. Four dogs were also shown to be infected with *Uncinaria stenocephala* and DNA sequences of these isolates showed 99% or greater similarity to previously published sequences of GenBank accession number HQ262054.1 for *U. stenocephala*. *Toxocara canis* and *Taenia* spp. infections were found in 5.4% (CI 2.3–8.6) and 4.5% (CI 1.6–7.3) of wild dogs respectively. Of the nine adult worms of *Taenia* spp. subjected to PCR and DNA sequencing, seven returned clean and readable sequences. BLAST results showed six of seven to have 100% sequence similarity to previously published sequences of GenBank accession number AB731674.1 for *T. serialis* and one of seven to have 94% sequence similarity to previously published sequence of GenBank accession number JN870104.1 for *T. pisiformis*. PCR did not detect *Neospora caninum*. No adult *L. serrata* individuals were detected at postmortem examination.

Zoonotic bacteria

The bacterial pathogens of interest were not commonly identified within the faecal material and nasal passages of peri-urban wild dogs (Table 2). *Salmonella* spp. were found at low levels (3.7 ± 4%), although 20% (CI 9.9–30.1) of faeces sampled ($n = 60$) contained ampicillin (AMP)-resistant *E. coli*. Of those samples, 33.3% were resistant only to AMP, 41.6% were resistant to AMP plus one other antimicrobial and 25% were resistant to AMP plus two other antimicrobials. The additional antimicrobials to which resistance was demonstrated included AMC, STX, TE and/or KF. *Proteus mirabilis* was also identified in a single faecal sample. *Brucella suis* was not detected within the sampled population. Multiple samples ($n = 7$) were suspected positive on the RB test, but were all negative at CF. All samples were also negative for *Rickettsia felis* and methicillin-resistant *S. aureus*; however, low levels of methicillin-sensitive *S. aureus* were detected (3.3 ± 2.7%).

Discussion

Given the comprehensive nature of the present study, both in terms of sample size and the number of pathogens screened, some conclusions can be drawn about pathogen infection in wild dogs in a large peri-urban area of eastern Australia. Parasitic infections in peri-urban wild dogs are common. Previous studies have generally focussed on rural populations, where a high prevalence of pathogens such as *S. erinacei* (Coman 1972; Jenkins *et al*. 2008), *Taenia* spp. (Coman 1972; Jenkins *et al*. 2014*b*) and *E. granulosus* (Baldock *et al*. 1985; Morrison *et al*. 1988; Jenkins and Morris 1991; Jenkins *et al*. 2008) has been reported in parasite carriage in wild dogs across Australia. Using a sample of peri-urban wild dogs from south-eastern Queensland (SEQ) and northern New South Wales (NSW), the present study demonstrated that *E. granulosus*, hookworms and *S. erinacei* also commonly infect these populations. Existing studies of *E. granulosus* infection in peri-urban wild-dog populations are discordant in the reported endemicity (Brown and Copeman 2003; Jenkins *et al*. 2008; Smout *et al*. 2013). Consistently high temperatures are likely to be major factors

Table 2. The presence of bacterial pathogens in peri-urban wild dogs

The number (*n*) of samples tested, and number (*n*) identified as positive (% ± CI), local government area where positive samples were collected and the presence or absence of co-infections are shown. MRSA, methicillin-resistant *Staphylococcus aureus*; MSSA, methicillin-sensitive *Staphylococcus aureus*

Species	Tested *n*	Positive *n* (% ± CI)	Local government area	Co-infections
Brucella suis	39	0 (0)	–	–
Campylobacter spp.	82	0 (0)	–	–
Ampicillin-resistant *Escherichia coli*	60	12 (20 ± 10.1)	Gold Coast	*Echinococcus*, *Toxocara*
			Gold Coast	Hookworm
			Gold Coast	*Echinococcus*, *Toxocara*
			Gold Coast	*Echinococcus*
			Sunshine Coast	Hookworm, *Spirometra*
			Somerset	*Spirometra*
			Somerset	*Echinococcus*, *Spirometra*
			Somerset	*Spirometra*
			Byron	*Echinococcus*, Hookworm
			Byron	*Echinococcus*, MSSA
			Byron	*Echinococcus*
			Ipswich	*Echinococcus*, *Salmonella* sp.
MRSA	164	0 (0)	–	–
MSSA	164	5 (3.3 ± 2.7)	Gold Coast	*Echinococcus*, Hookworm
			Gold Coast	*Echinococcus*, *Spirometra*
			Gold Coast	*Echinococcus*
			Sunshine Coast	Hookworm, *Spirometra*
			Byron	*Echinococcus*, *E. coli*
Rickettsia felis	82	0 (0)	–	–
Salmonella spp.	82	3 (3.7 ± 4.0)	Ipswich	*Echinococcus*, hookworm, *E. coli*
S. Potsdam			Byron	Hookworm
S. Birkenhead			Gold Coast	Unknown tapeworm
S. subsp. 1				

in the reduced prevalence of *E. granulosus* in northern Australia (Wachira *et al*. 1991; Torgerson and Heath 2003). Although Jenkins *et al*. (2008) reported a prevalence somewhat similar to that in the present study, all of the infected dogs were located towards the northern and western regions of Australia, with those closest to residential areas being free of the pathogen. In contrast, our results showed that wild dogs infected with *E. granulosus* are commonly found in the close proximity to residential areas, and *E. granulosus* is broadly distributed throughout SEQ, and parts of north-eastern NSW. The ability of infective eggs to travel and spread via wind, flies and within soil via accidental hosts and fomites (e.g. on shoes) enhances the range of the parasite. This is a significant concern for public health. SEQ is a highly populated area, being home to an estimated 3.4 million people in 2017 (ABS 2018). Within our study area, wild dogs can live within 1000 m of residential houses at all times, and repeatedly traverse roads, suburban backyards and frequently utilised public areas such as school yards and parklands (McNeill *et al*. 2016). In Switzerland, an emerging epidemic of human alveolar echinococcosis, caused by *Echinococcus multilocularis* (not present in Australia), was linked with a recorded increase in the urban fox population 10–15 years prior (Schweiger *et al*. 2007). *Echinococcus granulosus* is similar in that in can take several years for the disease to manifest in humans. Although current human cases in Australia are rare, it is important to conduct long-term surveillance to detect any developing impacts from the recent high prevalence detected in peri-urban wild dogs.

The low prevalence of hookworm (5.5%) recorded during postmortem examination increased markedly following further examination using faecal-floatation (17.2%) and molecular (39%) methods. Despite this, the prevalence of hookworms remained much lower than the 70.7% previously detected in scats from far-northern Queensland (FNQ; Smout *et al*. 2013) and the 74% previously detected in Townsville (Brown and Copeman 2003). Our estimates were also lower than the 37% prevalence in south-eastern Queensland (Jenkins *et al*. 2008). Climatic conditions such as increased humidity and temperature favour the presence of *Ancylostoma* spp. The reason for the low prevalence of hookworm in our study could be attributed to the ability of *A. caninum*, the most commonly detected species, which undergoes hypobiosis during the colder months, whereas, in tropical regions of Australia, the parasite will shed year round (Gibbs 1982). Hookworms are reportedly common in the domestic-dog population in Australia (Palmer *et al*. 2008), with the majority of these being *A. caninum* (Palmer *et al*. 2007). Similarly, *A. caninum* represents the main species detected in wild dogs (Brown and Copeman 2003; Jenkins *et al*. 2008; Smout *et al*. 2013). Although detected in wild-dog populations in FNQ (Smout *et al*. 2013), *A. ceylanicum* was not identified within our study area. Four wild dogs were found to have co-infections of *A. caninum* and *Uncinaria stenocephala*, which has not previously been reported as far north as SEQ. *Uncinaria stenocephala* is generally associated with the southern regions of Australia where temperatures are lower and

winters, in particular, are colder (Palmer *et al.* 2007). Dogs from our study with *U. stenocephala* were all trapped either in the month of May or August, which had respective average temperature ranges (minimum–maximum) of 13.9–24.0°C and 9.8–23.3°C (Brisbane: http://www.bom.gov.au/climate/data/index.shtml?bookmark=200, accessed May 2017), well within the ideal temperature for larval development of *U. stenocephala* (Gibbs and Gibbs 1959). Educating adults on the transmission routes of hookworm (percutaneous infection) and appropriate hygiene (no bare feet in parklands and hand-washing after children playing) as well as the clinical signs in humans (pruritic self-resolving rash, abdominal signs) is likely to contribute significantly to our ability to mitigate the potential risk of hookworm infections posed by wild dogs.

Two species of *Taenia* (*T. serialis*, *T. pisiformis*) were detected in the present study. Urbanisation may limit the exposure of wild dogs to pathogens that are more frequent in livestock than wildlife. The intermediate stages of *T. serialis* and *T. pisiformis* are commonly found in rabbits and are probably more likely to be detected among the peri-urban wild-dog population than are those of *T. hydatigena* and *T. ovis*, which use cattle, sheep and goats as intermediate hosts (Arundel 1972). Humans are accidental intermediate hosts for both species of *Taenia* detected in the present study, although records of cases are very rare (Ing *et al.* 1998). Therefore, the presence of *Taenia* species within the peri-urban wild-dog population is unlikely to be of concern to public health or be of major economic significance for livestock industries.

The high prevalence of *S. erinacei* that we have reported in the peri-urban wild-dog population could reflect its prevalence in feral-pig populations, which could impact on the international trade of feral-pig meat through condemnations of infected carcasses. Most feral-pig meat is exported to Europe, with exports between 2006 and 2007 being responsible for approximately AU$13 million into the economy (Bengsen *et al.* 2014). Feral-pig meat is commonly consumed in aboriginal communities around Australia (Koichi *et al.* 2012), but consumption by the general Australian public is generally very low and the local commercial market is small. Human infection can also result from consumption of pleuroceroids in the muscles of other intermediate or paratenic hosts (e.g. tadpoles, frogs, toads) or through accidental consumption of procercoids within copepods in fresh water (Lee *et al.* 1990). However, people in peri-urban regions have access to treated drinking water and often carry bottled water when spending time outdoors. As a result of this, as well as Australia's rigorous meat-hygiene inspection and food-safety assurance systems, public health implications from *S. erinacei* are likely to be minimal. However, profitability for the sector may be affected.

Toxocara canis was found at low numbers within the intestines of the peri-urban wild dogs. These results are similar to previous findings on the Sunshine Coast (Jenkins *et al.* 2008). However, prevalence was lower than that that detected in foxes in Western Australia, where 14.9% were infected (Dybing *et al.* 2013). Geographic and demographic risk factors of both human (Congdon and Lloyd 2011) and canine (Regis *et al.* 2011) toxocarosis are well described; however, spatial factors and environmental distribution of infective eggs are not as well understood. Although young

dogs in Bristol were found to shed the highest number of eggs, their movements were limited and, hence, contribution to environmental contamination was primarily through fox faeces (Morgan *et al.* 2013). This suggests that animals that shed the highest number of eggs may not be the most significant factors in the spread and/or maintenance of pathogen if their spatial movements are limited. Within the current study area, foxes (*n* = 102) were found to have a prevalence of *T. canis* (8.8%) similar to that in wild dogs (L. Harriott, unpubl. data). The low prevalence in both the fox and wild-dog populations may be from a lack of infective eggs in the environment (owing to an increase in anthelmintic use in domestic dogs) or a lack of paratenic hosts in the diet. There is also the potential for age-related immunity to be present because most infections in dogs greater than 6 months old could be somatic (Claerebout *et al.* 2009). Despite this, the potential impacts on human health are substantial and should be recognised by pest managers and health authorities.

The variety of parasitic species co-occurring in sampled dogs was lower than that in similar studies, and unlike previous reports, there were no uncommon parasites detected (Jenkins *et al.* 2008; Dybing *et al.* 2013). Peri-urban wild dogs across SEQ are mostly exposed to similar dietary items and exhibit similar preferences (Allen *et al.* 2016), and food items are usually readily available. Abundant and available food items may reduce the need for dogs to explore outside of their home range, and consume additional food resources that are outside of their preferred prey species. Hence, the exposure to different environmental sources of pathogen is probably limited.

Our results indicated that carriage of zoonotic bacterial pathogens is uncommon. As *E. coli* is a common bacterium than can be found naturally in the gastrointestinal tract of all animals and is frequently commensal rather than pathogenic, our analyses were focussed on detecting dogs that carried multi-drug-resistant (MDR) *E. coli*. In each of the local government areas north, south and west of Brisbane, at least one dog was found to be carrying MDR *E. coli*, although the Brisbane City local government area did not show any positive samples. Multiple samples were resistant to two or more drugs and, although the sample size was small, the presence of bacteria resistant to one or several antimicrobials in the peri-urban wild-dog population proves a significant risk to human health, because it enables the spread of resistance genes and bacteria into the environment and to other species of animals. It suggests value in future research into this topic. We also recorded three peri-urban wild dogs infected with *Salmonella*. Stafford and Bell (2011) suggested that many human *Salmonella* cases tend to be sporadic infections rather than outbreaks, which our preliminary data would also support for peri-urban wild-dog populations. *Staphylococcus aureus* has been detected in domestic dogs, which are often infected with lineages of human origin, rather than the dog-specific clonal complex (Malik *et al.* 2006; Strommenger *et al.* 2006; Weese *et al.* 2006). Although the number of detected wild dogs carrying MSSA was small, it warrants further investigation into MSSA and MRSA carriage by peri-urban wild dogs, because the potential public-health implications are significant.

There are some limitations to the present study. First, sampling could not be considered random. The collection of

wild dogs was influenced by public complaints or existing wild-dog management programs. Second, the logistics of field collection and storage of samples (particularly blood) made it difficult to refrigerate and process samples quickly. This may have reduced our ability to detect some pathogens, particularly bacteria. There were also discrepancies between the testing methods, suggesting that parasites, at least, are often best identified by locating the adult within the definitive host. However, molecular and faecal-floatation methods were more sensitive in the detection of hookworms than was identifying adults within the intestines. Hookworms are small and fragile, and the freeze–thaw process may have affected the ability to detect adults. Finally, temporal aspects of epidemiology are not accounted for in here, although sampling was conducted throughout the year. These limitations are minor because they are unlikely to affect the interpretation of the data or the overall conclusions.

Our results provided estimates of pathogen prevalence in peri-urban wild-dog populations in eastern Australia, which will support the strategic management of wild-dog impacts in these areas (Fleming *et al*. 2014). Public awareness of health in domestic dogs (e.g. correct deworming protocols), and communication on the importance of hygiene (particularly for trappers), can assist in mitigating the risk posed by pathogens carried by the peri-urban wild-dog population. Future research should focus on the risk posed by individual pathogens and their management, including multi-drug-resistant strains. This could include environmental screening for these same pathogens within public areas utilised by peri-urban wild dogs.

Conflicts of interest

The authors declare no conflicts of interest.

Acknowledgements

References

ABS (2018). '3218.0: Regional Population Growth, Australia, 2016–17.' Released 24 April 2018. Available at http://www.abs.gov.au/AUSSTATS/abs@.nsf/Lookup/3218.0Main+Features12016-17?OpenDocument [Verified 30 September 2018].

Allen, B. L., Goullet, M., Allen, L. R., Lisle, A., and Leung, L. K. P. (2013). Dingoes at the doorstep: preliminary data on the ecology of dingoes in urban areas. *Landscape and Urban Planning* **119**, 131–135. doi:10.1016/j.landurbplan.2013.07.008

Allen, B. L., Carmelito, E., Amos, M., Goullet, M. S., Allen, L. R., Speed, J., Gentle, M., and Leung, L. K. P. (2016). Diet of dingoes and other wild dogs in peri-urban areas of north-eastern Australia. *Scientific Reports* **6**, 23028. doi:10.1038/srep23028

Arundel, J. H. (1972). A review of cysticercoses of sheep and cattle in Australia. *Australian Veterinary Journal* **48**, 140–155. doi:10.1111/j.1751-0813.1972.tb09262.x

Baldock, F. C., Thompson, R. C., Kumaratilake, L. M., and Shield, J. (1985). *Echinococcus granulosus* in farm dogs and dingoes in south eastern Queensland. *Australian Veterinary Journal* **62**, 335–337. doi:10.1111/j.1751-0813.1985.tb07653.x

Bateman, P. W., and Fleming, P. A. (2012). Big city life: carnivores in urban environments. *Journal of Zoology* **287**, 1–23. doi:10.1111/j.1469-7998.2011.00887.x

Bengsen, A. J., Gentle, M. N., Mitchell, J. L., Pearson, H. E., and Saunders, G. R. (2014). Impacts and management of wild pigs *Sus scrofa* in Australia. *Mammal Review* **44**, 135–147. doi:10.1111/mam.12011

Bowman, D. D. (2013). 'Georgis' Parasitology for Veterinarians.' 10th edn. (Elsevier, Saunders: St Louis, MI.)

Brown, B., and Copeman, D. B. (2003). Zoonotic importance of parasites in wild dogs caught in the vicinity of Townsville. *Australian Veterinary Journal* **81**, 700–702.

Chen, J., Xu, M. J., Zhou, D. H., Song, H. Q., Wang, C. R., and Zhu, X. Q. (2012). Canine and feline parasitic zoonoses in China. *Parasites & Vectors* **5**, 152–159.

Choy, D. L., and Buxton, M. (2013). Farming the city fringe: Dilemmas for peri-urban planning. In 'Food Security in Australia: Challenges and Prospects for the Future'. (Eds Q. Farmar-Bowers, V. Higgins, and J. Millar.) pp. 397–412. (Springer US: Boston, MA.)

Claerebout, E., Casaert, S., Dalemans, A. C., De Wilde, N., Levecke, B., Vercruysse, J., and Geurden, T. (2009). Giardia and other intestinal parasites in different dog populations in northern Belgium. *Veterinary Parasitology* **161**, 41–46. doi:10.1016/j.vetpar.2008.11.024

Coman, B. J. (1972). Helminth parasites of the dingo and feral dog in Victoria with some notes on diet of the host. *Australian Veterinary Journal* **48**, 456–461. doi:10.1111/j.1751-0813.1972.tb02281.x

Congdon, P., and Lloyd, P. (2011). *Toxocara* infection in the United States: the relevance of poverty, geography and demography as risk factors, and implications for estimating county prevalence. *International Journal of Public Health* **56**, 15–24. doi:10.1007/s00038-010-0143-6

Deplazes, P., van Knapen, F., Schweiger, A., and Overgaauw, P. A. M. (2011). Role of pet dogs and cats in the transmission of helminthic zoonoses in Europe, with a focus on echinococcosis and toxocarosis. *Veterinary Parasitology* **182**, 41–53. doi:10.1016/j.vetpar.2011.07.014

Dybing, N. A., Fleming, P. A., and Adams, P. J. (2013). Environmental conditions predict helminth prevalence in red foxes in Western Australia. *International Journal for Parasitology. Parasites and Wildlife* **2**, 165–172. doi:10.1016/j.ijppaw.2013.04.004

Fajardo, V., Gonzalez, I., Martin, I., Rojas, M., Hernandez, P. E., Garcia, T., and Martin, R. (2008). Real-time PCR for detection and quantification of red deer (*Cervus elaphus*), fallow deer (*Dama dama*), and roe deer (*Capreolus capreolus*) in meat mixtures. *Meat Science* **79**, 289–298. doi:10.1016/j.meatsci.2007.09.013

Faust, E. C., D'Antoni, J. S., Odom, V., Miller, M. J., Peres, C., Sawitz, W., Thomen, L. F., Tobie, J., and Walker, J. H. (1938). A critical study of clinical laboratory technics for the diagnosis of protozoan cysts and helminth eggs in feces: I. Preliminary communication. *The American Journal of Tropical Medicine and Hygiene* **s1–18**, 169–183.

Fish, L., Mazuz, M., Molad, T., Savitsky, I., and Shkap, V. (2007). Isolation of *Neospora caninum* from dairy zero grazing cattle in Israel. *Veterinary Parasitology* **149**, 167–171. doi:10.1016/j.vetpar.2007.08.009

Fleming, P. J. S., Allen, B. L., Allen, L. R., Ballard, G.-A., Bengsen, A., Gentle, M. N., McLeod, L., Meek, P. D., and Saunders, G. R. (2014). Management of wild canids in Australia: free-ranging dogs and red foxes. In 'Carnivores of Australia: Past, Present and Future'. (Eds A. Glen and C. Dickson.) pp. 149–207. (CSIRO Publishing: Melbourne.)

Gibbs, H. C. (1982). Mechanisms of survival of nematode parasites with emphasis on hypobiosis. *Veterinary Parasitology* **11**, 25–48. doi:10.1016/0304-4017(82)90119-4

Gibbs, H. C., and Gibbs, K. E. (1959). The effects of temperature on the development of the free-living stages of *Dochmoides stenocephala* (Railliet, 1884) (Ancylostomidae: Nematoda). *Canadian Journal of Zoology* **37**, 247–257. doi:10.1139/z59-030

Harriott, L. (2018). Prevalence, risk factors and geographical distribution of zoonotic pathogens carried by peri-urban wild dogs. PhD Thesis, School of Veterinary Science, The University of Queensland, Gatton, Qld.

Hii, S. F., Kopp, S. R., Abdad, M. Y., Thompson, M. F., O'Leary, C. A., Rees, R. L., and Traub, R. J. (2011). Molecular evidence supports the role of dogs as potential reservoirs for *Rickettsia felis*. *Vector Borne and Zoonotic Diseases (Larchmont, N.Y.)* **11**, 1007–1012. doi:10.1089/vbz.2010.0270

Ing, M. B., Schantz, P. M., and Turner, J. A. (1998). Human coenurosis in North America: case reports and review. *Clinical Infectious Diseases* **27**, 519–523. doi:10.1086/514716

Inpankaew, T., Schär, F., Khieu, V., Muth, S., Dalsgaard, A., Marti, H., Traub, R. J., and Odermatt, P. (2014). Simple fecal flotation is a superior alternative to quadruple Kato Katz smear examination for the detection of hookworm eggs in human stool. *PLoS Neglected Tropical Diseases* **8**, e3313. doi:10.1371/journal.pntd.0003313

Jenkins, D. J., and Morris, B. (1991). Unusually heavy infections of *Echinococcus granulosus* in wild dogs in south-eastern Australia *Australian Veterinary Journal* **68**, 36–37. doi:10.1111/j.1751-0813.1991.tb09844.x

Jenkins, D. J., Allen, L., and Goullet, M. (2008). Encroachment of *Echinococcus granulosus* into urban areas in eastern Queensland, Australia. *Australian Veterinary Journal* **86**, 294–300. doi:10.1111/j.1751-0813.2008.00327.x

Jenkins, D. J., Lievaart, J. J., Boufana, B., Lett, W. S., Bradshaw, H., and Armua-Fernandez, M. T. (2014*a*). *Echinococcus granulosus* and other intestinal helminths: current status of prevalence and management in rural dogs of eastern Australia. *Australian Veterinary Journal* **92**, 292–298. doi:10.1111/avj.12218

Jenkins, D. J., Urwin, N. A. R., Williams, T. M., Mitchell, K. L., Lievaart, J. J., and Armua-Fernandez, M. T. (2014*b*). Red foxes (*Vulpes vulpes*) and wild dogs (dingoes (*Canis lupus dingo*) and dingo/domestic dog hybrids), as sylvatic hosts for Australian *Taenia hydatigena* and *Taenia ovis*. *International Journal for Parasitology. Parasites and Wildlife* **3**, 75–80. doi:10.1016/j.ijppaw.2014.03.001

Koenig, J., Shine, R., and Shea, G. (2001). The ecology of an Australian reptile icon: how do blue-tongued lizards (*Tiliqua scincoides*) survive in suburbia? *Wildlife Research* **28**, 214–227. doi:10.1071/WR00068

Koichi, K., Sangha, K. K., Cottrell, A., and Gordon, I. J. (2012). Aboriginal rangers' perspectives on feral pigs: are they a pest or a resource? A case study in the wet tropics World Heritage Area of northern Queensland. *Journal of Australian Indigenous Issues* **15**, 2–19.

Lee, S. H., We, J. S., Sohn, W. M., Hong, S. T., and Chai, J. Y. (1990). Experimental life history of *Spirometra erinacei*. *Korean Journal of Parasitology* **28**, 161–173. doi:10.3347/kjp.1990.28.3.161

Low Choy, D., Sutherland, C., Scott, S., Rolley, K., Gleeson, B., Dodson, J., and Sipe, N. (2007). 'Change and Continuity in Peri-urban Australia. Peri-urban Case Study: South East Queensland.' Urban Research Program. (Griffith University: Brisbane, Qld.)

Malik, S., Coombs, G. W., O'Brien, F. G., Peng, H., and Barton, M. D. (2006). Molecular typing of methicillin-resistant staphylococci isolated from cats and dogs. *The Journal of Antimicrobial Chemotherapy* **58**, 428–431. doi:10.1093/jac/dkl253

McNeill, A. T., Leung, L. K., Goullet, M. S., Gentle, M. N., and Allen, B. L. (2016). Dingoes at the doorstep: home range sizes and activity patterns of dingoes and other wild dogs around urban areas of north-eastern Australia. *Animals (Basel)* **6**, e48. doi:10.3390/ani6080048

Morgan, E. R., Azam, D., and Pegler, K. (2013). Quantifying sources of environmental contamination with *Toxocara* spp. eggs. *Veterinary Parasitology* **193**, 390–397. doi:10.1016/j.vetpar.2012.12.034

Morrison, P., Stanton, R., and Pilatti, E. (1988). *Echinococcus granulosus* infection in wild dogs in south-eastern New South Wales. *Australian Veterinary Journal* **65**, 97–98. doi:10.1111/j.1751-0813.1988.tb07376.x

Newsome, T. M., Ballard, G.-A., Dickman, C. R., Fleming, P. J. S., and van de Ven, R. (2013). Home range, activity and sociality of a top predator, the dingo: a test of the resource dispersion hypothesis. *Ecography* **36**, 914–925. doi:10.1111/j.1600-0587.2013.00056.x

Ng-Nguyen, D., Stevenson, M. A., Dorny, P., Gabriël, S., Vo, T. V., Nguyen, V.-A. T., Phan, T. V., Hii, S. F., and Traub, R. J. (2017). Comparison of a new multiplex real-time PCR with the Kato Katz thick smear and copro-antigen ELISA for the detection and differentiation of *Taenia* spp. in human stools. *PLoS Neglected Tropical Diseases* **11**, e0005743. doi:10.1371/journal.pntd.0005743

Palmer, C. S., Traub, R. J., Robertson, I. D., Hobbs, R. P., Elliot, A., While, L., Rees, R., and Thompson, R. C. A. (2007). The veterinary and public health significance of hookworm in dogs and cats in Australia and the status of *A. ceylanicum*. *Veterinary Parasitology* **145**, 304–313. doi:10.1016/j.vetpar.2006.12.018

Palmer, C. S., Thompson, R. C. A., Traub, R. J., Rees, R., and Robertson, I. D. (2008). National study of the gastrointestinal parasites of dogs and cats in Australia. *Veterinary Parasitology* **151**, 181–190. doi:10.1016/j.vetpar.2007.10.015

Prange, S., Gehrt, S. D., and Wiggers, E. P. (2003). Demographic factors contributing to high raccoon densities in urban landscapes. *The Journal of Wildlife Management* **67**, 324–333. doi:10.2307/3802774

Regis, S. C., Mendonca, L. R., Silva Ndos, S., Dattoli, V. C., Alcantara-Neves, N. M., and Barrouin-Melo, S. M. (2011). Seroprevalence and risk factors for canine toxocariasis by detection of specific IgG as a marker of infection in dogs from Salvador, Brazil. *Acta Tropica* **120**, 46–51. doi:10.1016/j.actatropica.2011.05.011

Schweiger, A., Ammann, R. W., Candinas, D., Clavien, P.-A., Eckert, J., Gottstein, B., Halkic, N., Muellhaupt, B., Prinz, B. M., Reichen, J., Tarr, P. E., Torgerson, P. R., and Deplazes, P. (2007). Human alveolar echinococcosis after fox population increase, Switzerland. *Emerging Infectious Diseases* **13**, 878–882. doi:10.3201/eid1306.061074

Smout, F. A., Thompson, R. C. A., and Skerratt, L. F. (2013). First report of *Ancylostoma ceylanicum* in wild canids. *International Journal for Parasitology. Parasites and Wildlife* **2**, 173–177. doi:10.1016/j.ijppaw.2013.04.003

Stafford, R. J., and Bell, R. (2011). Monitoring the incidence and causes of disease potentially transmitted by food in Australia: annual report of the OzFoodNet Network, 2011. *Communicable Diseases Intelligence* **39**, e236–e264.

Statham, M., and Statham, H. L. (1997). Movements and habits of brushtail possums (*Trichosurus vulpecula* Kerr) in an urban area. *Wildlife Research* **24**, 715–726. doi:10.1071/WR96092

Strommenger, B., Kehrenberg, C., Kettlitz, C., Cuny, C., Verspohl, J., Witte, W., and Schwarz, S. (2006). Molecular characterization of methicillin-resistant *Staphylococcus aureus* strains from pet animals and their relationship to human isolates. *The Journal of Antimicrobial Chemotherapy* **57**, 461–465. doi:10.1093/jac/dki471

Torgerson, P. R., and Heath, D. D. (2003). Transmission dynamics and control options for *Echinococcus granulosus*. *Parasitology* **127**, S143–S158. doi:10.1017/S0031182003003810

Traub, R. J., Robertson, I. D., Irwin, P., Mencke, N., and Thompson, R. C. A. (2004). Application of a species-specific PCR–RFLP to identify *Ancylostoma* eggs directly from canine faeces. *Veterinary Parasitology* **123**, 245–255. doi:10.1016/j.vetpar.2004.05.026

Traub, R. J., Hobbs, R. P., Adams, P. J., Behnke, J. M., Harris, P. D., and Thompson, R. C. A. (2007). A case of mistaken identity: reappraisal of the species of canid and felid hookworms (*Ancylostoma*) present in Australia and India. *Parasitology* **134**, 113–119. doi:10.1017/S0031182006001211

Wachira, T. M., Macpherson, C. N., and Gathuma, J. M. (1991). Release and survival of *Echinococcus* eggs in different environments in Turkana, and

their possible impact on the incidence of hydatidosis in man and livestock. *Journal of Helminthology* **65**, 55–61. doi:10.1017/S002214 9X00010440

Wang, W., Cuttell, L., Bielefeldt-Ohmann, H., Inpankaew, T., Owen, H., and Traub, R. J. (2013). Diversity of *Blastocystis* subtypes in dogs in different geographical settings. *Parasites & Vectors* **6**, 215. doi:10.1186/1756-3305-6-215

Weese, J. S., Dick, H., Willey, B. M., McGeer, A., Kreiswirth, B. N., Innis, B., and Low, D. E. (2006). Suspected transmission of methicillin-resistant *Staphylococcus aureus* between domestic pets and humans in veterinary clinics and in the household. *Veterinary Microbiology* **115**, 148–155. doi:10.1016/j.vetmic.2006.01.004