

Prevalence of Parasites,  
Especially those with Zoonotic Potential,  
in Wild Dogs/Dingoes in Suburban Fringe Areas of Townsville  
(Queensland, Australia)

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## Abstract

Necropsies were performed on 27 wild dogs professionally culled in the Townsville district of Australia between August and September 2002. The purpose of this study was to define the prevalence of parasitic infections present in the local wild dog population, with special focus on those of zoonotic potential, and to speculate as to the associated risks to the community's health. Collection of ectoparasites revealed the presence of one species of flea, *Ctenocephalides felis* (present in 35% of wild dogs in this study), and two genera of ticks, *Amblyomma sp.* and *Haemophysalis sp.* (60% and 47% of wild dogs infected, respectively). Four species of helminth were found in the intestines: *Ancylostoma caninum* (74% of sample population infected), *Dipylidium caninum* (59% of wild dogs infected), *Spirometra erinacei* (49% of wild dogs infected), and *Echinococcus granulosus* (22% of wild dogs infected). *Dirofilaria immitis* was present in the heart and pulmonary arteries of 75% of the adult dogs. Plasma from these dogs was also tested for dirofilariasis by two different methods, an ELISA and a commercially available kit. Results from a haemagglutination-inhibition test performed on these samples demonstrated antibodies to parvovirus in 27% of the dogs. It was concluded that encroachment of wild dogs into the suburban fringes of Townsville presents a low risk to public health and the health of adequately cared for domestic dogs.

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## Introduction

Arriving on the isolated continent of Australia about 4000 years ago, it is most commonly believed that the dingo was brought by Asian seafarers as an important hunting companion during their terrestrial explorations (Corbett 1995a). Hardly domesticated, it was not uncommon for a dingo to take off roaming from its human overseer and to be left behind on the continent. Dingoes quickly populated Australia and remain to this day the only species of wild dog on the continent. Over the past two centuries, due to hybridization with domestic dogs brought by European settlers, dingoes have become increasingly rare in their pure-bred form (Corbett 1995b). Despite the hybridized genetic composition of many wild dogs, they continue to be referred to as dingoes by local residents. Methods have been devised to differentiate pure dingoes from hybridized wild dogs and to measure the extent of this hybridization. However, the accuracy of these methods continues to be debated. For the purpose of this paper, the dogs in this study will be referred to as wild dogs.

Encroachment of wild dogs into suburban fringe areas of Townsville (Queensland, Australia) has raised concerns that these dogs may carry diseases of zoonotic importance or which may infect domestic dogs. To address this perceived problem, Townsville City Council and several other community institutions funded a short-term culling program of troublesome wild dogs during August-September 2002. This action presented the opportunity to survey parasites carried by the dogs and thus permit an assessment of potential health risks of these parasites to the Townsville community and domestic dogs.

A survey of parasites in wild dogs in the Townsville district has not previously been conducted, and relatively little is known about the prevalence of infectious diseases, including those of zoonotic potential, in Australia's wild dog population. The purpose of this study was to determine the prevalence of parasitic infections, especially those with zoonotic potential carried by these dogs. Furthermore, as no studies have previously been conducted on parvovirus infection in wild dogs, this survey also

included assessment of parvoviral titers to determine if wild dogs may be a source of parvovirus infection for domestic dogs.

## Materials & Methods

Due to concern over the potential public safety and health risk posed by the recent encroachment of wild dogs into suburban fringe areas of Townsville (19°13' S, 146°48' E), the Townsville City Council, Townsville Hospital, James Cook University, and the Australian Army (which holds a large base in the area) collaborated to employ a professional trapper to cull wild dogs in the area. This took place from the first of August to mid-September 2002.

***Wild Dog Collection:*** A total of 27 wild dogs were collected and examined in this study. The majority of these dogs were caught by professional trappers, Mark Goulet and Gavan McKenzie of the company Ferals Out. Traps with smooth metal jaws were used that clamped together when a dog stepped on the trigger plate. The force of these traps was adjusted so as not to break the skin or bones of the leg. Four domestic dogs inadvertently caught were returned unharmed to their owners. Several of the wild dogs pulled their leg out of the trap; consequently, traps for these particular dogs were reset with a slightly greater force. Nineteen of the wild dogs were shot within a few hours of being caught using a .22 "Hornet" with a lead bullet that exploded on impact. Dogs were shot at close range in the neck just behind the skull, but a couple dogs not caught in traps were shot in the chest from a range of about 100 meters. One adult dog and all seven pups in this study were euthanized with pentobarbitone by Bruce Copeman of the Australian Institute of Tropical Veterinary and Animal Science. Euthanasia was chosen over shooting for reasons of expediency. Each body, regardless of the means of death, was sealed in a vinyl body bag and brought to the animal necropsy room at James Cook University, where they were held at 4°C. All dogs were necropsied within 48 hours of death. The age of each dog was initially estimated according to body size, sexual maturation and, to some extent, dental development, although toothware is not a wholly accurate indication of age in wild dogs (Corbett 1995a). In subsequent studies, the dogs were also classified as either pure dingoes or hybrid dingoes on the basis of skull morphology and DNA profile.



### ***Necropsy:***

*Collection of plasma:* The trapper collected 5mL of blood by cardiac puncture immediately following euthanasia of each wild dog. Blood was transferred to heparinized tubes and stored at 4°C. At the time of necropsy the blood was centrifuged at 3500rpm for 10 minutes, and plasma was recovered and stored at -20°C until further use.

*Collection of ectoparasites:* A thorough examination of the hair and skin of the ears, face, neck, shoulders, chest, belly, inner flanks, and rump was made for fleas, lice, and ticks. The inside of the vinyl body bag in which each dead dingo was transported was rinsed with water several times and parasites recovered in a 250- $\mu$ m aperture sieve. Contents of the sieve were grossly examined in a white tray and all parasites found were preserved in 10% formalin for later identification.

*Collection of gastrointestinal helminths:* The stomach, small intestine, and large intestine were each ligated at the junctions between and examined separately. Each was longitudinally cut along its entire length and the contents washed into a 250- $\mu$ m aperture sieve. Stomach washings were grossly examined for presence of worms. A representative sample of stomach contents was preserved in 10% formalin for use in another study to determine the species of prey consumed. The intestines were run between the examiner's thumb and forefinger several times to scrape off any attached worms while visually inspecting the mucosal side. Scrapings were added to the sieve with the intestinal contents for thorough washing. All contents remaining in the sieve were preserved in 10% formalin for later examination.

*Examination of heart & lungs:* The trachea, esophagus and aorta were severed to permit removal of the heart and lungs. A longitudinal incision was made in the right ventricle along the intraventricular septum, extending up into the pulmonary artery and its branches. Heartworms found in the right ventricle and pulmonary arteries were removed and their number and sex recorded.

### ***Identification of Parasites:***

All preserved intestinal contents were examined for parasites grossly and with a dissecting microscope. Helminths and all ectoparasites previously collected were examined under a dissecting microscope and identified using keys (Soulsby 1968; Roberts 1970). Results were tabulated for each wild dog as either a positive or negative result.

### ***Serological Testing:***

*ELISA for detection of dirofilariasis:* The method used was according to TropBio's "ELISA kit for detecting and quantifying *Wuchereria bancrofti* antigen". Each plasma sample was prepared by adding 100 $\mu$ L of plasma to a tube of 300 $\mu$ L of sample diluent in a 100°C water bath for five minutes. Tubes were then centrifuged at 2000g for 15 minutes. Fifty- $\mu$ L aliquots of the supernatant fluid from each tube were added to each of two test wells in a microtiter tray pre-coated with a monoclonal antibody (Og4C3) which binds antigen of *D. immitis*. To prepare the control and standards, 50 $\mu$ L of the conjugate control and the seven standard *D. immitis* antigens supplied were added to the wells in pairs (ie, B11 and B12 each contained 50 $\mu$ L of standard #1). Fifty- $\mu$ L of sample diluent was also added to wells A11 and A12. The tray was placed in a dark, humid container and allowed to incubate overnight; following which it was washed with buffer three times and tapped dry. Fifty- $\mu$ L of diluted rabbit anti-Onchocerca antibody was then added to every well and the tray left to incubate for one hour before being washed with buffer three times and tapped dry. Diluted anti-rabbit conjugate was then added in 50 $\mu$ L aliquots to each well, the plate left to incubate for one hour, and then washed three times with buffer and tapped dry. Finally, 100 $\mu$ L of chromogen was added to each well and the plate was incubated for another hour before reading the results with a spectrophotometer at dual wavelengths of 414nm and 442nm.

*Commercial (AGEN) canine heartworm test:* This antigen detection test utilizes migration of soluble *D. immitis* antigen (in whole blood, serum, or plasma), marked with colloidal gold, along a nitrocellulose strip. Across this strip is attached a line of anti-heartworm antibody and further along another line of anti-canine antibody. In a positive test

colloidal gold will accumulate at both lines whereas in a negative test only the second line will accumulate gold and be visualized. The procedure described in the pamphlet enclosed with the pre-packaged tests was followed. To summarize, a drop of plasma was added to the sample well of the test along with two drops of buffer. After ten minutes the test results were readable.

*Hemagglutination-Inhibition for canine parvovirus:* Canine parvovirus has a protein which causes porcine erythrocytes to agglutinate in the presence of virus. Exposure of dogs to the virus stimulates production of antibodies that prevent this agglutination process. This is the basis of the hemagglutination-inhibition test used here. The method used was as described by Smith (1986) with noted modifications. In summary, 0.2mL of plasma was mixed with 0.6mL of kaolin in mini test tubes and allowed to sit for twenty minutes at room temperature, with occasional shaking. Centrifugation at 2000rpm for ten minutes pelleted the kaolin; 0.05mL of packed washed porcine erythrocytes were added and allowed to sit for an hour, being occasionally shaken. After further centrifugation, the supernatant fluid was transferred to a well in a microtiter tray. This tray remained at 4°C for eight days while awaiting a fresh supply of porcine erythrocytes. When these became available, a 0.05mL aliquot of each sample of plasma was serially diluted two-fold in a fresh microtiter tray with 0.05mL of antigen as diluent and incubated for an hour and a half at room temperature. Controls were also prepared at this time to determine viral titer being used. After incubation, 0.05mL of 0.5% porcine erythrocytes was added to each well of the tray which was gently tapped to ensure mixing and left to incubate overnight.

## Results

Necropsies were conducted on twenty-seven wild dogs caught in the vicinity of Townsville between August 2002 and September 2002. The age and sex distribution of these dogs is shown in Table 1.

**Table 1** Age and sex distribution of twenty-seven wild dogs caught in the vicinity of Townsville and presented for necropsy at JCU.

Sex	Puppies (<3 months)	Adults (>12 months)	Total
<b>Males</b>	1	10	11
<b>Females</b>	6	10	16
<b>Total</b>	7	20	27

Table 2 shows both the overall prevalence of each helminth species and the prevalence with regards to age present in the wild dogs examined. Among the adults, *A. caninum* was the most prevalent species (90%), followed by *D. immitis* (75%). Among puppies *D. caninum* was most prevalent (57%), followed by *A. caninum* (28%); no other helminths were found in this age group.

**Table 2** Prevalence of helminth species, according to age, found in twenty-seven wild dogs presented for necropsy at JCU. (*D. immitis* requires at least six months to develop and thus was not applicable in the survey of puppies.)

Parasite Species	Number Infected			Prevalence (%)		
	Puppies	Adults	Overall	Puppies	Adults	Overall
<i>Dirofilaria immitis</i>	NA	15	NA	NA	75	NA
<i>Ancylostoma caninum</i>	2	18	20	28	90	74
<i>Spirometra erinacei</i>	0	12	12	0	60	44
<i>Dipylidium caninum</i>	4	12	16	57	60	59
<i>Echinococcus granulosus</i>	0	6	6	0	30	22

Six adult wild dogs (3 male; 3 female) were infected with *E. granulosus*. All of these dogs were trapped at the army training area.

Table 3 shows the OD values of the ELISA for dirofilariasis, the ELISA and AGEN test results, and the actual *D. immitis* worm burden found in eighteen of the adult wild dogs examined. As blood was not collected from wild dogs #1 and #2, these dogs were excluded. The ELISA has been standardized by Dr. Janice Smith of James Cook University based upon her previous work so that any OD value greater than 0.300 was accepted as a positive result. Only ten AGEN tests were available for the purpose of examining the relative sensitivity of this test.

**Table 3** Comparison of results of ELISA and AGEN serological tests to detect circulating antigen of *D. immitis* with actual counts of *D. immitis* found at necropsy in eighteen adult wild dogs caught in the vicinity of Townsville. Heartworms were found in all dogs listed; however, no count was recorded for four dogs and is indicated by “NR”. A blank space under AGEN response indicates no AGEN test was run for that dog. P=positive, N=negative

Wild Dog #	<i>D. immitis</i> Worm Count			Mean OD Value	ELISA Result	AGEN Result
	Male	Female	Total			
3	NR	NR	NR	1.164	P	
4	NR	NR	NR	0.163	N	N
10	NR	NR	NR	0.396	P	
11	0	0	0	0.058	N	
12	1	3	4	1.038	P	P
13	2	3	5	1.840	P	
14	NR	NR	NR	1.195	P	
15	7	6	13	1.605	P	P
16	6	16	22	1.327	P	P
17	4	4	8	0.912	P	N
18	9	7	16	1.518	P	
19	0	4	4	1.132	P	P
20	0	0	0	0.058	N	
24	1	3	4	0.189	N	N
25	4	6	10	0.712	P	N
26	1	6	7	0.867	P	P
29	8	13	21	1.094	P	P
30	0	0	0	0.050	N	

The prevalence of ectoparasites is shown in Table 4. Ticks of the genus *Amblyomma* were most prevalent overall (60%). Among the adult population

*Amblyomma sp.* was again most prevalent (62%), followed by another genus of tick, *Haemaphysalis sp.* (50%), and the flea *Ctenocephalides felis* (19%). Among puppies, however, *C. felis* was most prevalent (71%), followed by *Amblyomma sp.* (50%) and *Haemaphysalis sp.* (25%).

**Table 4** Prevalence of ectoparasites, according to age, of wild dogs presented for necropsy at JCU. (\*Due to a lack of examination for ectoparasites in the first few wild dogs caught, the sample number varied and is thus included in the table.)

Parasite Species	*Number Infected/Examined			Prevalence (%)		
	Puppies	Adults	Overall	Puppies	Adults	Overall
<i>Ctenocephalides felis</i>	5/7	3/16	8/23	71	19	35
<i>Amblyomma sp.</i>	2/4	10/16	12/20	50	62	60
<i>Haemaphysalis sp.</i>	1/4	8/16	9/20	25	50	45

Six of the twenty-five dogs tested were positive in the hemagglutination-inhibition test for canine parvovirus antibody. These are an approximate reading of the final results, as autoagglutination occurred for unknown reasons within some of the control wells and thus may have occurred within the test wells, too. Despite the lack of definite results, it appears that at least some of these wild dogs have been exposed to canine parvovirus while the majority has not. This test will be repeated under different conditions with the same serum samples in hopes of obtaining more concrete data.

## Discussion

### **I. *Canis lupus dingo***

Much ambiguity has surrounded the origin and history of the dingo. This has led to a great deal of inconsistency in the dingo's classification – *Canis antarcticus* (est. by Kerr 1792), *Canis dingo* (est. by Meyer 1793), the still quite commonly used *Canis familiaris dingo*, and *Canis lupus dingo*, among many others (Breckwoldt 1988; Corbett 1995a). It is now widely accepted that the dingo is a non-native mammal of Australia that was brought from Asia by man approximately 4000 years ago; however, multiple theories exist as to the details of this migration (Breckwoldt 1988). According to Dr. Laurie Corbett of CSIRO (1995a), the dingo evolved from a cross of the Arabian wolf, *Canis lupus arabs*, and the Indian wolf, *Canis lupus pallipes*, up to 10,000 years ago in parts of Southern Asia. For this reason, he concluded the scientifically correct classification of the dingo is *Canis lupus dingo*, a subspecies of the wolf. Corbett's historical account relates that it was around this time in history that evidence of wolf/dingo domestication began to occur in Israel and, through human pressures of selective breeding, led to what are now over 600 official breeds of the modern domestic dog, *Canis familiaris*. In Southeast Asia, however, the dingo remained free of man's manipulation and thus morphologically unchanged. Among the evidence used by Corbett to support his theory are fossils of the dingo's earliest ancestors in Northeast Thailand and North Vietnam dating back 5000-5500 years which are nearly identical to the structure of the present pure-bred dingo.

Despite such preservation of this sub-species over the past half-millennium, a decline in the number of pure-bred dingoes in Australia has occurred, due to heavy hybridization with the domestic dog (Breckwoldt 1988). Encroachment of wild dogs into suburban fringes of cities, combined with growth of the human population and expansion of residential development into rural areas, provides an increasing opportunity for interaction between wild dogs and domestic dogs. Thus the hybrid population is rapidly replacing the gene pool of the dingo. Corbett (1995b) estimated that only half of Eastern Australia's wild dog population consists of true dingoes.

Distinguishing pure-bred dingoes from dingo/feral dog hybrids or determining the proportion of dingo present in a hybrid dog is difficult. Breckwoldt (1988) concluded that eight anatomical characteristics (including bulla size, jaw width, various tooth sizes, and cranial height) can be used to distinguish pure-bred dingoes from domestic dogs; however, while hybrids show a definite cross of these characteristics from the parents, the extent of this cross is erratic and offspring of hybrids tend to be more dingo-like than the parents (Breckwoldt 1988). Coat color, too, is not completely reliable, although white feet, a white-tipped tail, and fairly solid coloration is typical of most pure-bred dingoes (Breckwoldt 1988).

In an attempt to categorize the dogs in this study as either pure dingoes or hybridized wild dogs, skull morphology and DNA profile of each dog were examined in parallel studies. Dr. Pete Wulf and his students at JCU made eight different measurements on each of twenty-one of the skulls (Wulf, personal communication). They concluded that eighteen were likely to be pure dingoes, two were hybrids and one was of unknown breed. DNA tests by Dr. Alan Wilton at the University of New South Wales, however, suggested a more hybridized population. The technique used examined allele sizes at twenty microsatellite loci and compared them to those of reference samples from dingoes and domestic dogs (Wilton, personal communication). Thus, since the extent of hybridization with domestic dogs of the dogs examined in this survey is unresolved they are referred to as “wild dogs” in this discussion.

## **II. Survey of Parasites and Associated Zoonotic Implications**

Hybridization is only one consequence of overlapping territory between wild dogs and domestic dogs. The potential for spread of canine infectious diseases from wild dogs to domestic dogs and humans has also become an issue. Examination of the twenty-seven wild dogs in this study revealed the presence of six parasites with potential zoonotic capabilities: *E. granulosus*, *A. caninum*, *D. immitis*, *D. caninum*, *S. erinacei*, and *Amblyomma sp.* Comparison of these results with those of past studies, as



well as implications for canine and human health will be discussed. Basic epidemiology and the related pathogenesis of each of these parasites will also be briefly covered.

*Echinococcus granulosus*, a small member of the family Taeniidae, or tapeworm, is responsible for causing hydatid disease in humans. This parasite is passed via a fecal-oral route involving two relatively non-specific mammalian hosts: an herbivore or omnivore acting as the intermediate host and a carnivore as the definitive host. The life cycle is described in Thompson and Lymbery's Echinococcus and Hydatid Disease (1995) and is summarized as follows. Infective eggs passed in the feces of a carnivore are ingested by the intermediate host (including humans) and development of a cystic metacestode (larval stage) occurs within the viscera of this new host. Asexual reproduction occurs within the metacestode giving rise to thousands of protoscolices, each capable of developing further into a mature adult worm. When a carnivore preys upon this infected host, the cysts are ingested; digestive juices within the carnivore free the protoscolices, allowing them to continue development into the adult stage in the intestinal tract, among the crypts of Lieberkuhn.

The most likely source of human infection is intimate contact with an infected domestic dog and the consequent heightened opportunity for infection with eggs from the dog's feces (Roberts & Janvay 2000); however, several studies have investigated cases in which such exposure was unlikely (Hope et al. 1992). Possible explanations have implicated the dingo as a less direct but possible source (Baldock et al. 1985; Banks 1984; Hope et al. 1992; Wallner 1999). While human infections are now relatively rare in Australia due to the success of a campaign over the past three decades to control infection previously maintained in a sheep-dog cycle, human cases are still reported annually. Most current cases probably represent a spillover of infection maintained sylvatically either in a dingo-wallaby cycle or a fox-wallaby cycle (Baldock et al. 1985; Banks 1984; Wallner 1999; Jenkins et al. 2000). In 1999 and again in 2000, the Communicable Disease Intelligence reported 26 cases, equivalent to 0.2 cases/100000 of the Australian population (Australian Dpt. of Health & Aging, 2000). This is likely to be an underestimation of the actual case occurrence for two reasons: 1) the developing

cysts may remain asymptomatic for upwards of 20 years depending on location within the host's body and 2) cases in some high risk groups, such as indigenous communities in which dogs live intimately with their owners, tend to be underreported as people in these rural areas are less likely than others in the Australian community to use the regional medical services (Wallner 1999).

The most important means of dissemination of eggs of *E. granulosus* from canine feces is suspected to be flies (Banks 1984). Eggs present in dog feces are picked up by flies feeding on the feces and then transferred to surrounding objects (Banks 1984). This puts areas in which both infected dogs and humans congregate at a higher risk for zoonotic transmission. The six wild dogs in this study which were infected with *E. granulosus* were all caught on the local army training area. Although this does constitute a potential health risk to the army personnel who use the training area, the risk may be considered low for several reasons: 1) the area is large and inhabited by few wild dogs, 2) burdens of *E. granulosus* found within each dog were relatively low, and 3) direct exposure of feces to the sun probably restricts survival time of the eggs to a few days.

The best form of prevention against *E. granulosus* infection in humans, as with all other zoonotic parasites spread via a fecal-oral route, is through public awareness and education on proper food sanitation. Such practice should be promoted throughout the community, as picnic sites and other recreational areas where people and wild dogs congregate may constitute a risk for transmission of *E. granulosus*. Areas at higher elevations upon Mt. Stewart, centrally located on the army training land, may present a higher risk due to the presence of rock wallaby habitat. Known to harbor viable hydatid cysts (Banks 1984), rock wallabies often play an important part in this parasite's sylvatic life cycle. Based on examination of hairs in fecal scats in the areas where the wild dogs were trapped, agile wallabies, and not rock wallabies, were the most common component of their diet (Palmer, personal communication). This observation, absence of hydatid cysts in numerous necropsies of agile wallabies from the area in the past years, and the low burden of *E. granulosus* in all but one of the wild dogs suggest

that the infected dogs in this survey hunted mainly in lowland areas and may have had only occasional access to an infected wallaby.

The most common parasite of the wild dogs surveyed was *A. caninum*. Eggs of this hookworm are passed in the feces of an infected dog. Transmission occurs when the hatched larvae come in direct contact with the skin of a potential host and penetration occurs. Rather than migrating to the bloodstream, lungs and intestine as would occur in the infection of a dog, most larvae infecting a human host tunnel aimlessly through the skin. This gives rise to itchy, raised red tracks called cutaneous larvae migrans or creeping eruptions, which may become infected with bacteria (Roberts & Janvov 2000). It is also now apparent that some larvae penetrate through the skin and undergo a blood-lung migration to reach the intestine. In some individuals this causes eosinophilic enteritis. The affected portion of small intestine is edematous and heavily infiltrated from mucosa to serosa with eosinophils. The link between infection with *A. caninum* and eosinophilic enteritis was made by a Townsville gastroenterologist, Dr. John Croese, who diagnosed 93 cases of eosinophilic enteritis in patients from the Townsville region between 1988 and 1990 (Provic & Croese 1990). Cases of eosinophilic enteritis are now quickly resolved following therapy with mebendazole, replacing previous symptomatic therapies and, in some patients, surgical removal of affected intestine.

The ninety percent prevalence of infection with *A. caninum* in adult wild dogs is similar to the 87% prevalence in dogs from the local pound (Wallner 1999). If this latter group of dogs may be considered a sample of the domestic dog population in Townsville, encroaching wild dogs would appear to pose a comparable risk to humans; in other words, feces from a wild dog is just as likely to be infective as is the feces from a domestic dog. Parks, picnic areas, and other shaded locations with damp soil that are frequented by both people and domestic or wild dogs provide favorable conditions for the transmission of *A. caninum*. These areas constitute the greatest risk during the wet season when the moist environment allows extended survival of the larvae, otherwise prone to desiccation (Gilles & Ball 1991). Although most human infections are likely a

result of exposure to an infected domestic pet, hookworm in wild dogs may be a significant cause of disease within the wild dog population; enteric hemorrhage and dark tarry feces typical of a clinical level of infection were present in a number of the animals.

The warm, humid climate of Townsville is ideal not only for perpetuating infection with *A. caninum*, but also for promoting the mosquito-transmitted canine heartworm, *D. immitis*. Transmission occurs when a mosquito, the intermediate host, ingests microfilariae present in the blood of an infected dog, the definitive host. Larvae undergo several stages of development within the mosquito (Roberts & Janvov 2000) and are transmitted to a new definitive host during the mosquito's next feed. The infective larvae travel to the right ventricle and pulmonary arteries of the dog where they develop to maturity over the next six months. For this reason, infection is typically undetectable in puppies under about six months of age. Chronic infection may result in coughing, exercise intolerance, circulatory distress, inefficient functioning of the heart valves, and eventually death (Roberts & Janvov 2000; Soulsby 1968).

Prior to the now widespread use of chemoprophylactic drugs, especially macrocyclic lactones, prevalence of heartworm among dogs in North Queensland was demonstrated to be as high as 90% (Welch & Dobson 1974). In 1972 Aubrey and Copeman estimated prevalence in dogs in Townsville was 77%. Since then, prevalence has been greatly reduced. A recent survey of adult dogs from the Townsville pound found 25% were infected (Wallner 1999); furthermore, prevalence is likely to be even lower among domestic dogs receiving adequate care from their owners. The higher prevalence of 75% in the adult wild dogs surveyed may thus provide a reservoir for infection of domestic dogs not receiving chemoprophylactic treatment including puppies.

Cases of infection with *D. immitis* in humans have been reported but are generally asymptomatic (Moorhouse et al. 1971). Diagnosis in the past occurred only after incidental finding of pulmonary nodules in chest x-rays of patients being examined for tuberculosis in the 1960s and early 1970s (Moorhouse et al. 1971). As of

1974, 65 cases of dirofilariasis in man had been reported worldwide, with distribution strongly corresponding to that seen in dogs (Welch & Dobson 1974). Aborigines in communities where canine infection with *D. immitis* is endemic had a higher prevalence of anti-*D. immitis* antibodies (20.1%) than Caucasians (2.6%). This difference is likely due to geographic factors, with prevalence of *D. immitis* infection in dogs of the area reflecting the relative size of the mosquito vector population. Aborigines tend to live in communities much farther north than most Caucasians, where the warmer, more humid climate gives way to a thriving mosquito population to which Aborigines are continually exposed. Surprisingly, of the 65 cases of dirofilariasis mentioned earlier not one occurred in an Aborigine. Welch and Dobson suggest the reason may be due to a gradual immunity built up throughout a lifelong exposure to *D. immitis*. Overall, their study demonstrated that while the potential for human exposure in endemic areas such as Australia exists, *D. immitis* is of negligible risk to public health. It may also be concluded that infection of wild dogs with *D. immitis* will not constitute a significant risk of infection to domestic dogs so long as domestic dogs receive chemoprophylaxis.

Results from the enzyme linked immunosorbent assay (ELISA) for dirofilariasis run on eighteen of these dogs demonstrated the test has comparatively high sensitivity and specificity in relation to worm count. All positive and negative results correlated with presence or absence of worms, respectively, with the exception of two samples, both of which were false negatives; however, one of these dogs had only four immature worms (a worm count was not recorded for the other). The commercially available AGEN test for heartworm was less sensitive than the ELISA. Besides giving a “true” negative result for the two samples negative by ELISA, it also gave two false negative results. The less accurate result of the AGEN test in comparison to ELISA, is offset by the greater convenience of use of the AGEN test which gives a result within ten minutes, whereas the ELISA takes two days to complete.

*Dipylidium caninum* is a common tapeworm found in dogs and cats worldwide. Transmission occurs via ingestion of fleas infected with a cysticercoid. Larval fleas become infected by eating *D. caninum* eggs from the desiccated proglottids passed in

feces of the infected definitive host (Roberts & Janvay 2000). Such infection is detrimental to the health of the flea and this may make it sluggish and more likely to be caught and eaten than a non-infected flea. *D. caninum* was the most common internal parasite found in puppies (57%). As the puppies came from the same den and were heavily infested with fleas, this result was not unexpected. Cases of human infection, which nearly always involve crawling children (Hugh-Jones et al. 1995), are typically asymptomatic. As close contact between children and wild dogs is an unlikely scenario it may be concluded that wild dogs pose no significant human health risk with regard to *D. caninum*.

*Spirometra erinacei*, a relatively large tapeworm of dogs and cats worldwide, was also found in this survey. It typically occurs in animals which feed on the fauna surrounding lakes or rivers. Humans may become infected with proceroids by drinking water contaminated with infected copepods or ingesting raw meat infected with plerocercoids (Hugh-Jones et al. 1995), the infection is usually asymptomatic and not considered to be of zoonotic relevance. Nevertheless, personnel in the army training area should be discouraged from drinking untreated ground water to avoid infection.

Ectoparasites were included in this survey, but only one was of minimal zoonotic importance. Those found were the common cat and dog flea, *Ctenocephalides felis*, and two genera of ticks, *Amblyomma* sp. and *Haemaphysalis* sp. Identification of the ticks was carried out only to the level of genus, as many of the specimens were larvae or nymphs and could not be identified to species using the available key. Adult specimens present were identified as *Haemaphysalis bancrofti* (the common wallaby tick) and *Amblyomma triguttatum* (the common kangaroo tick). While these ticks frequently parasitize macropodids, they may also parasitize humans among other animals. *A. triguttatum* has been incriminated in transmission of Q fever from kangaroos to humans (Pope et al. 1960). Thus, infestation of wild dogs with these ticks may contribute to their persistence in the region, but their role is probably minor in comparison to that of their usual and more numerous macropod hosts. Occasional attachment, especially to personnel in the

army training area, can be expected. The ticks are easily removed and have no toxic effects but their potential to transmit Q fever in this region is unknown.

### **III. Parvovirus Serology**

Canine parvovirus (CPV-2) is a highly virulent virus capable of infecting all canids. Structurally very similar to feline panleukopenia virus (FPV), it has been hypothesized that the intermediate virus between these two developed in foxes (Steinel et al. 2001, Truyen 1999). Canine parvovirus first appeared in Australia in late 1978 (Sabine et al. 1982). The infection is most commonly characterized by syndromes of enteritis and/or CPV myocarditis (Vella & Ketteridge 1991). Since the manufacturing of a highly effective vaccine in 1980 (Sabine et al. 1982), incidence of parvovirus infection in domestic dogs has dropped dramatically; however, vaccinated puppies less than six months of age may continue to be at risk for this infection as demonstrated by a 1982 survey carried out at the University of Sydney (Sabine et al. 1982). Dogs of this age and unvaccinated or immunocompromised dogs are the groups at highest risk for becoming infected with CPV.

It has been speculated that CPV is endemic in many wild carnivore populations (Steinel et al. 2001); random cases of their exposure to the virus have been demonstrated in serum specimens from North Queensland (Smith, 1986), but no studies or surveys of CPV in dingoes has been published. As the virus is shed in high quantities in the feces of infected animals and may be easily spread by rodents, feral cats, or even wind (Studdert et al. 1983), wild dogs could potentially provide a valuable source of infection for susceptible domestic dogs. Additionally, as a very stable virus able to persist in the environment outside the host for upward of several weeks, feces of infected animals remains a serious health threat throughout this time (Mealey, personal communication).

Results from the hemagglutination-inhibition (HI) test run on twenty-five of the collected serum samples indicated only a small proportion of these wild dogs had been exposed to canine parvovirus, as demonstrated by the presence of serum antibodies. While results of the HI were rather difficult to interpret and somewhat subjective in

many cases it was clear that both types of results did exist within the sample population and that negative responses far outweighed the positive responses. The reason for the apparently low prevalence of CPV in the wild dogs may be related to their dispersal singly or in small family groupings over a relatively large territory, reducing the possibility of epidemic spread. While it is possible that infected wild dogs in suburban areas may be a source of infection for unvaccinated domestic dogs, the possibility also exists that infected domestic dogs provide a source of infection for local wild dogs.

#### **IV. General Discussion**

The growing presence of wild dogs in suburban fringe areas of cities like Townsville has raised concern regarding public health and safety. The growing presence of hybrid wild dogs has not only illustrated the increasing proximity of these unwelcome residents to the local community, but has suggested underlying health implications associated with the increased exposure of humans and their pets to these wild dogs.

Although this study did reveal the presence of parasites of zoonotic importance and other canine-communicable diseases within the local wild dog population, it is assessed the overall risk toward the health of the community to be relatively low. Parasites found which were considered to be of most significance to human health in terms of disease brought on by the initial infection were *E. granulosus* and *A. caninum*. While the latter is considered endemic in tropical parts of Australia, especially Townsville, wild dogs pose less of a threat than domestic dogs due to issues of confinement. Public awareness and education regarding proper hygiene are the best methods of prevention. This recommendation also stands for *E. granulosus*, a parasite not nearly as common but of much more serious consequence. Prevention through education is the key.

In other cases, wild dogs may act as a reservoir of infection for diseases no longer able to establish themselves as fully in the domestic dog population as a result of



advancements in preventative therapy. These diseases include canine heartworm and canine parvovirus. Monthly preventative medication and strict vaccination protocols via veterinary care have greatly reduced prevalence among domestic dogs. While some groups remain at risk for developing infection, the untreated wild dog population may be one of the last strongholds of such agents of disease. Continued adherence to preventative therapy may one day render them obsolete among domestic dogs.

This survey provided the prevalence of numerous parasites among the local wild dog population. It also established a rough estimate of the prevalence of parvovirus within this sample; further testing will reveal a more accurate value as well as present information regarding the antibody titer of those exposed wild dogs. Such assessment provides a basis for further studies in a variety of fields, including the surveillance of health among wild dogs, potential monitoring of dog-human infectious disease transmission, and comparing of the status or translocation of such parasites over time.

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## Appendix A

### Wild Dog Survey Results

Sex	Age	Weight (Kg)	Parasites								<i>D. immitis</i>			
			<i>C. felis</i>	<i>Haem. sp.</i>	<i>Amb. sp.</i>	<i>A. canin.</i>	<i>D. canin.</i>	<i>S. erinacei</i>	<i>E. gran.</i>	<i>D. immitis</i>	Worm #	OD Value	ELISA	AGEN
F	0.2	3	P	NR	NR	N	N	N	N	NA	NA	NA	NA	
F	0.2	3	P	NR	NR	N	P	N	N	NA	NA	NA	NA	
F	0.2	3	P	NR	NR	N	P	N	N	NA	NA	NA	NA	
F	0.2	3.2	P	N	N	P	P	N	N	NA	NA	NA	NA	
F	0.2	3.2	P	N	N	N	P	N	N	NA	NA	NA	NA	
F	0.2	3.2	N	N	P	N	N	N	N	NA	NA	NA	NA	
M	0.2	3.4	N	P	P	P	N	N	N	NA	NA	NA	NA	
F	1	NR	NR	NR	NR	P	N	P	N	N	NR	NB	NB	
F	2	14.3	N	P	P	P	N	N	N	N	0	0.058	N	
F	2	14.4	N	N	N	P	N	P	P	P	7	0.867	P	P
F	2	15.2	N	N	N	P	P	N	N	N	0	0.050	N	
M	3	17.8	NR	NR	NR	P	N	P	N	N	NR	NB	NB	
F	3	15	N	P	P	N	P	P	P	P	4	0.189	N	N
M	4	17	NR	NR	NR	N	P	P	N	P	NR	1.164	P	
M	4	19	NR	NR	NR	P	N	P	N	P	NR	0.163	N	N
M	4	19	P	N	P	P	P	P	P	P	NR	0.396	P	
F	4	17.2	N	P	P	P	N	N	P	P	4	1.038	P	P
M	4	19.6	N	N	P	P	P	N	P	P	5	1.840	P	
M	4	17.2	P	P	P	P	P	N	N	P	NR	1.195	P	
F	4	16	P	P	N	P	P	N	N	P	13	1.605	P	P
M	4	17.4	N	P	N	P	P	N	N	P	22	1.327	P	P
F	4	14	N	N	N	P	P	P	N	P	8	0.912	P	N
M	4	21.9	N	P	P	P	N	P	N	P	16	1.518	P	
M	4	19.9	N	N	P	P	P	P	P	N	0	0.058	N	
M	4	20.2	N	P	P	P	P	P	N	P	10	0.712	P	N
F	4	12.7	N	N	N	P	N	N	N	P	21	1.094	P	P
M	7	19.4	N	N	P	P	N	P	N	P	4	1.132	P	P

#### Table Key:

*C. felis* = *Ctenocephalides felis*  
*Haem. sp.* = *Haemaphysalis sp.*  
*Amb. sp.* = *Amblyomma sp.*  
*A. caninum* = *Ancylostoma caninum*

*D. caninum* = *Dipylidium caninum*  
*S. erinacei* = *Spirometra erinacei*  
*E. gran.* = *Echinococcus granulosus*  
*D. immitis* = *Dirofilaria immitis*

F = female  
M = male  
N = negative  
P = positive

NR = not recorded  
NB = no blood collected  
NA = not applicable





## Appendix A

### Wild Dog Survey Results

Sex	Age	Weight (Kg)	Parasites								<i>D. immitis</i>			
			<i>C. felis</i>	<i>Haem. sp.</i>	<i>Amb. sp.</i>	<i>A. canin.</i>	<i>D. canin.</i>	<i>S. erinacei</i>	<i>E. gran.</i>	<i>D. immitis</i>	Worm #	OD Value	ELISA	AGEN
F	0.2	3	P	NR	NR	N	N	N	N	NA	NA	NA	NA	
F	0.2	3	P	NR	NR	N	P	N	N	NA	NA	NA	NA	
F	0.2	3	P	NR	NR	N	P	N	N	NA	NA	NA	NA	
F	0.2	3.2	P	N	N	P	P	N	N	NA	NA	NA	NA	
F	0.2	3.2	P	N	N	N	P	N	N	NA	NA	NA	NA	
F	0.2	3.2	N	N	P	N	N	N	N	NA	NA	NA	NA	
M	0.2	3.4	N	P	P	P	N	N	N	NA	NA	NA	NA	
F	1	NR	NR	NR	NR	P	N	P	N	N	NR	NB	NB	
F	2	14.3	N	P	P	P	N	N	N	N	0	0.058	N	
F	2	14.4	N	N	N	P	N	P	P	P	7	0.867	P	P
F	2	15.2	N	N	N	P	P	N	N	N	0	0.050	N	
M	3	17.8	NR	NR	NR	P	N	P	N	N	NR	NB	NB	
F	3	15	N	P	P	N	P	P	P	P	4	0.189	N	N
M	4	17	NR	NR	NR	N	P	P	N	P	NR	1.164	P	
M	4	19	NR	NR	NR	P	N	P	N	P	NR	0.163	N	N
M	4	19	P	N	P	P	P	P	P	P	NR	0.396	P	
F	4	17.2	N	P	P	P	N	N	P	P	4	1.038	P	P
M	4	19.6	N	N	P	P	P	N	P	P	5	1.840	P	
M	4	17.2	P	P	P	P	P	N	N	P	NR	1.195	P	
F	4	16	P	P	N	P	P	N	N	P	13	1.605	P	P
M	4	17.4	N	P	N	P	P	N	N	P	22	1.327	P	P
F	4	14	N	N	N	P	P	P	N	P	8	0.912	P	N
M	4	21.9	N	P	P	P	N	P	N	P	16	1.518	P	
M	4	19.9	N	N	P	P	P	P	P	P	0	0.058	N	
M	4	20.2	N	P	P	P	P	P	N	P	10	0.712	P	N
F	4	12.7	N	N	N	P	N	N	N	P	21	1.094	P	P
M	7	19.4	N	N	P	P	N	P	N	P	4	1.132	P	P

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*C. felis* = *Ctenocephalides felis*

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*E. gran.* = *Echinococcus granulosus*  
*D. immitis* = *Dirofilaria immitis*

M = male  
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