Companion Animal Zoonoses Guidelines

Australian Companion Animal Zoonoses Advisory Panel (ACAZAP)

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HOW DO I NAVIGATE THESE GUIDELINES?

Antimicrobial Resistance

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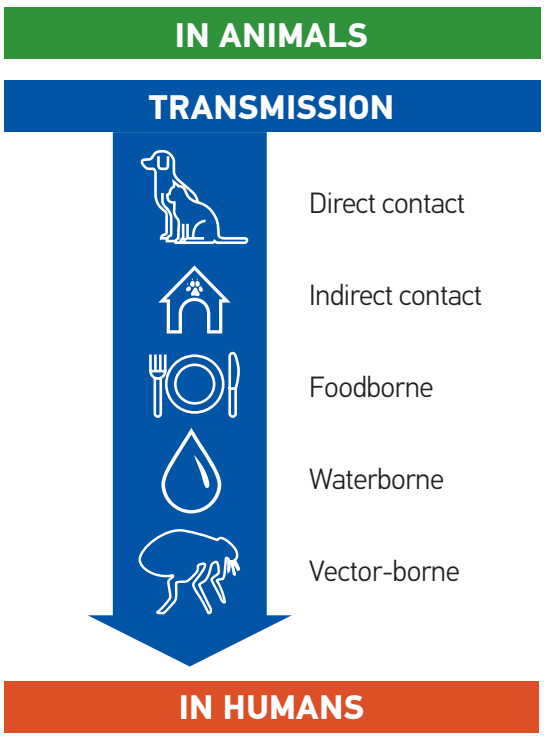
- infection control practices (inclusive of PPE and isolation requirements) to be implemented. The AVA Guidelines for Veterinary Industry Personal Biosecurity are a useful resource in this regard.

- Hyperlinks within the text are indicated in blue text and will take you to additional resources.



- The icons opposite are used as a quick guide to the modes of transmission of the pathogens listed in the guidelines.

Cover image: Medical illustration of nontyphoidal *Salmonella* spp. bacteria. (Public Health Image Library, CDC)

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COMPANION ANIMAL ZOONOTIC DISEASES

"Animals are such agreeable friends
– they ask no questions, they pass no criticisms"

Mary Ann Evans (aka George Eliot)

The agreeable nature of many domestic animals has seen them become indispensable companions to many around the world. In Australia, 60% of households are reported to have a pet, with an estimated 5.1 million pet dogs and 3.7 million pet cats.[1] The reasons for pet ownership, like the pets themselves, are many and varied. In addition to companionship, pet ownership has a range of positive emotional, physical, and psychological benefits including improved mental wellbeing, increased independence, and increased physical activity.[2,3]

Dogs and cats, both healthy and sick, may carry a range of different zoonotic organisms. Given the close relationship between pets and people and their shared living environment, it is not surprising that interspecies transfer may occur occasionally, either directly or indirectly. Whilst transmission of zoonotic pathogens has always been a risk, increasing anthropomorphism of companion animals and the associated high-intensity human-animal interactions make such infections more likely as opportunities for transmission increase. Coupled with this is an increase in the population of those at greatest risk for severe consequences of these infections, including people with compromised immune systems (e.g. HIV, organ transplants, cancer), pregnant women, the very young and the elderly.

With zoonotic diseases, there is no such thing as a "no-risk" pet, or a "no-risk" owner. It is important however to consider the risks in a rational and evidence-based manner. By implementing appropriate risk mitigation strategies, the benefits of pet ownership can be enjoyed safely in the vast majority of circumstances.

AUSTRALIAN COMPANION ANIMAL ZOONOSES ADVISORY PANEL (ACAZAP)

In February 2020, Boehringer Ingelheim brought together an expert panel of veterinary and human infectious disease experts to review and discuss the latest research and make evidence-based recommendations around the control of zoonotic diseases in dogs and cats.

THE AIMS OF THE PANEL WERE TO:

- Provide recommendations and strategies to minimise the risk of zoonotic disease transfer from dogs and cats in the veterinary clinic and community setting
- Facilitate discussion and collaboration between human and veterinary medical professionals to optimise health outcomes, both for pets and people
- Promote awareness of zoonotic diseases and strategies to control them to pet owners

The pathogens included in these guidelines were chosen by the panel based on consideration of their significance in the Australian context. In this regard, significance is a broad term encompassing factors such as the probability and/or consequences of infection. In reviewing each pathogen, the panel considered animal factors, environmental factors, and human factors that contribute to zoonotic disease. Inclusion of a pathogen in these guidelines does not imply that companion animals are the sole or even primary source of infection for people. In some cases, the contribution of dogs and cats to the disease burden in humans may be small and overshadowed by other potential routes of transmission. In such instances, an understanding of the minor role companion animals play remains important as it allows veterinarians and pet owners to fully evaluate the risk and implement a proportional management response.

Whilst it is not necessary for veterinarians to treat or manage human zoonotic infections, a knowledge of risk factors and the consequences of infection in humans allows for a more considered analysis of risk for themselves, their staff, and their clients. From a medicolegal perspective, veterinarians have a duty of care for their staff and clients and are obligated to provide advice and protective strategies to protect people under their guidance. However, veterinarians must use caution not to exceed the scope of their veterinary registration while fulfilling their public health responsibilities. Information concerning veterinary or public health aspects of zoonoses should be provided to clients as indicated and requested, with all recommendations clearly documented in clinical records. Veterinarians should not diagnose or treat diseases in humans or make recommendations about those issues.

On the other side of the zoonoses coin, for human medical professionals, an understanding of the epidemiology of these pathogens in animals, and the associated risk factors in animals, will assist in assessing and managing potential cases, and providing advice to patients about minimising the risk of zoonoses from companion animals. In this regard there is much to be gained by facilitating greater interaction between the medical and veterinary professions to help prevent, diagnose, and treat zoonotic diseases.[4]

References: 1. Animal Medicines Australia, Pets in Australia: A national survey of pets and their people. 2019. 2. Smith, B., (2012) The 'pet effect': Health related aspects of companion animal ownership. *Aust Fam Physician*, 41(6), 439. 3. McConnell, A.R., et al (2011) Friends with benefits: on the positive consequences of pet ownership. *J Pers Soc Psychol*, 101(6), 1239. 4. Steele, S.G., et al (2019) What makes an effective One Health clinical practitioner? Opinions of Australian One Health experts. *One Health*, 8, 100108.

AUSTRALIAN COMPANION ANIMAL ZOONOSES ADVISORY PANEL MEMBERS

Associate Professor Katrina Bosward

BSc (Vet) Hons 1, BVSc, PhD, Grad Dipl Vet Clin Sci, Grad Cert (Higher Ed), MASM, MASID

Associate Professor in Veterinary Microbiology, Sydney School of Veterinary Science, University of Sydney

Following the completion of a BSc (Vet) in 1990 and BVSc in 1991 at the University of Sydney, Katrina worked in mixed and small animal veterinary practice. Katrina returned to the University of Sydney in 1995 to undertake a PhD in collaboration with CSIRO, Animal Production entitled "Eosinophils and Interleukin 5 in Sheep". On completion of her PhD, Katrina commenced training in Clinical and Anatomical Veterinary Pathology and Microbiology at the University Veterinary Centre, Camden earning a Graduate Diploma in Veterinary Clinical Studies.

Since 2002, Katrina has been an academic staff member at the Sydney School of Veterinary Science, University of Sydney where her current teaching within the Doctor of Veterinary Medicine degree is centred on the pathogenesis of infectious diseases (including those considered zoonotic) and the biosecurity practices associated with controlling and preventing those diseases. Her current research projects follow the same themes with a general interest in zoonotic diseases. Her true passion however is all things concerning *Coxiella burnetii* and she is involved in many projects investigating this intriguing pathogen in a wide variety of species including Q fever in humans.

Dr Timothy Gilbey

MBBS, FRACP, BSc (phys)

Infectious Disease Visiting Medical Officer (VMO), Murrumbidgee Local Health District (MLHD). Conjoint Associate Lecturer, University of New South Wales

Dr Timothy Gilbey is a Fellow of the Royal Australasian College of Physicians in Infectious Diseases, a member of the Australasian Society for Infectious Diseases, and is currently Infectious Disease Visiting Medial Officer (VMO) for the Murrumbidgee Local Heath District (MLHD). In addition to clinical responsibilities, Tim has been heavily involved in education and training in medicine and infectious diseases; he holds a Conjoint Associate Lecturer position with the University of New South Wales where he is responsible for teaching clinical aspects of infectious disease to medical students; is involved in the Royal Australasian College of Physicians as a Regional Examiner; and was the founding chair of *"Bug School"*, a teaching program specifically designed to bridge experience gaps in the teaching of infectious diseases to registrars. Tim has a specific interest in antimicrobial stewardship, serving as co-chair of the Antimicrobial Stewardship Committee for MLHD. Tim's research interests include antimicrobial resistance and the use of bacteriophages to treat severe bacterial infections, having presented and published in this area. Tim is also passionate about rural medicine and One Health.

Associate Professor Thomas Gottlieb

MBBS, FRACP, FRCPA

Senior Staff Specialist Microbiology and Infectious Diseases, Concord Hospital. Clinical Associate Professor, University of Sydney

Associate Professor Gottlieb is an Infectious Diseases physician and microbiologist, Head of the Infectious Diseases and Microbiology Department at Concord Hospital, and Senior Lecturer at the University of Sydney. He is a past president and honorary member of the Australasian Society for Infectious Diseases (ASID) and a past president of the Australian Society for Antimicrobials (ASA).

Thomas is an executive member of the Australian Group on Antimicrobial Resistance (AGAR) and ASA. He has represented ASA on the Australian Strategic and Technical Advisory Group on Antimicrobial Resistance (ASTAG), responsible for the development and implementation of Australia's National Antimicrobial Resistance (AMR) Strategy.

He has been chair of advisory committees supervising training in Infectious Diseases and Microbiology. He has participated in the writing groups for the Australian Infection Control Guidelines, the national Therapeutic Guidelines for Antibiotic Use, Australian recommendations for the control of carbapenemase-producing *Enterobacteriaceae* (CPE) in acute care health facilities and guidelines for Antimicrobial Stewardship in Australian Healthcare. He is a member of the NPS Antibiotic Resistance Reference Group and the National Antimicrobial Stewardship Advisory Committee for the Australian Commission on Safety and Quality in Health Care (ACSQHC).

Associate Professor Jane Heller

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Associate Head of School /Associate Professor in Veterinary Epidemiology and Public Health, School of Animal and Veterinary Sciences, Charles Sturt University

After completing a BSc with a major in psychology and mathematics, Jane graduated with a BVSc(Hons) from the University of Sydney. Following some years of clinical work in private practice and at the Universities of Sydney (where she also obtained two additional postgraduate qualifications, DipVetClinStud and MVetClinStud) and Glasgow, she completed her training in Veterinary Epidemiology and Public Health through a PhD and DiplECVPH residency at the University of Glasgow.

In 2009 Jane took up a faculty position at Charles Sturt University and progressed to work as an Associate Professor in Veterinary Epidemiology and Public Health, currently still holding this position as a part time appointment. Jane also works as a consultant epidemiologist within her business 'Heller Consulting'. Jane has been involved in numerous research projects, acting as principal investigator for many of these, has published over 70 journal articles and delivered over 100 scientific presentations at national and international conferences. Jane's main research interest is in infectious disease epidemiology, with particular reference to antimicrobial resistance and the potential for zoonotic transfer of pathogens between animals and humans.

Professor Peter Irwin

BVetMed, PhD, FANZCVS, MRCVS

Emeritus Professor, Murdoch University. Founding Director, Co-Chair and Hon. Treasurer, Tropical Council for Companion Animal Parasites (TroCCAP)

Peter graduated in veterinary science from the Royal Veterinary College, London University in 1982 and has a PhD from James Cook University (1991) for studies into canine babesiosis in Australia. He is a Fellow of the Australian and New Zealand College of Veterinary Scientists and is a registered specialist in canine medicine. He is currently Emeritus Professor at Murdoch University in Perth.

Peter has worked in academia in Australia and overseas for 30 years as a teacher of companion animal medicine and as a researcher in the fields of veterinary parasitology and medical microbiology. He is an internationally recognised expert in vector-borne diseases and is a director of the Vector and Waterborne Pathogens Research Group (the Cryptick Laboratory) at Murdoch University.

Professor Jacqueline Norris

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Professor of Veterinary Microbiology & Infectious Diseases, Associate Head of Research, Sydney School of Veterinary Science, University of Sydney

Jacqui is a Professor of Veterinary Microbiology and Infectious Diseases, and Associate Head of Research at the Sydney School of Veterinary Science, at the University of Sydney. She is a registered practicing veterinarian and is passionate about practical research projects and education programs for veterinary professionals, animal breeders and animal owners.

Her main research areas include: 1) Development of diagnostics and treatments for companion animal viral diseases; 2) Q fever; 3) Multidrug resistant (MDR) *Staphylococcus* species; 4) Infection prevention and control in veterinary practices; 5) Chronic renal disease in domestic and zoo felids and 6) Factors influencing antimicrobial prescribing behaviour of vets and health professionals.

Professor Rebecca J. Traub

BSc, BVMS (Hons), PhD

Professor of Veterinary Parasitology, Faculty of Veterinary and Agricultural Sciences, The University of Melbourne. Founding Director, Tropical Council for Companion Animal Parasites (TroCCAP)

Prof. Traub graduated as a veterinarian from Murdoch University, WA in 1997 and subsequently worked in small animal practice. In 2004, she completed her PhD on canine parasitic zoonoses for which she was awarded the John Adrian Sprent Prize by the Australian Society for Parasitology. Prof. Traub was subsequently awarded a fellowship to continue her research in this field by the Australian Research Council. In 2006, she gained employment as a lecturer in Veterinary Public Health at the University of Queensland and in 2014 moved to the Melbourne Veterinary School, where she currently works as a Professor of Veterinary Parasitology and Australian Research Council Future Fellow (2021-2025).

Prof. Traub has published over 145 international peer-reviewed papers and book chapters covering the diagnosis, zoonotic potential, epidemiology and control of canine endoparasites and vector-borne diseases, with much of her research based in the Asia Pacific. Dr Traub's research expertise has been formally recognized through consultations for the WHO, FAO, OIE, The Gates Foundation, the veterinary pharmaceutical industry, and not-for-profit organisations. In 2019, she was awarded the Bancroft Mackerras Medal of Excellence by the Australian Society for Parasitology. In 2015, Prof. Traub founded the Tropical Council for Companion Animal Parasites and currently serves as the President Elect of the Australian Society for Parasitology (President, 2021-2023).

ANTIMICROBIAL RESISTANCE

- Antimicrobial resistance (AMR) is a critical global health challenge in human medicine and an emerging problem in companion animal medicine.
- In addition to rendering some animal infections more difficult, or even impossible to treat, the development of AMR in pets poses a risk to human health. The close relationship between companion animals and humans facilitates the transfer, directly or indirectly, of shared resistant organisms or genetic determinants. There is potential for bi-directional flow, with the transfer of resistant organisms/genes from animal-to-human or vice versa, and thus a One Health approach to the problem is essential.
- The role and contribution of companion animals to AMR in humans is complex and incompletely understood. It is clear however that antimicrobial use in animals, as in humans, is a risk factor for colonisation or infection with resistant pathogens. Prudent use of antimicrobials by the veterinary profession is an important component of addressing the threat of AMR in animals, and by extension in minimising the contribution animals may play in human AMR. Surveys of companion animal veterinarians and a review of veterinary antimicrobial prescribing practices report the regular use of broad-spectrum antibiotics of high importance to human health, highlighting a need for an increased focus on the principles of prudent use in the profession.[1,2]
- The prevalence and impact of AMR varies globally, and not all resistant organisms have a potential zoonotic component. Specific organisms of concern which have a demonstrated or potential involvement of companion animals in their transmission include methicillin-resistant *S. aureus* (MRSA), methicillin-resistant *S. pseudintermedius* (MRSP), extended-spectrum beta-lactamase producing *Enterobacterales* (ESBL-E), carbapenemase-producing *Enterobacterales* (CPE), and *Clostridioides difficile*. Antimicrobial resistance in *Campylobacter* and *Salmonella* are also a potential zoonotic concern and are discussed in the relevant sections on pages 24 and 71 respectively.

ACAZAP RECOMMENDATIONS

MINIMISING SELECTION FOR RESISTANCE IN ANIMALS

• Prescribing veterinarians should follow established guidelines for the prudent use of antimicrobials. The Australian Antibacterial Importance Ratings, developed by the Australian Strategic Technical Advisory Group (ASTAG) on Antimicrobial Resistance, categorises antimicrobials as of high, medium or low importance. Veterinarians should avoid the use of antimicrobials of high importance in human medicine, such as third generation cephalosporins and fluoroquinolones, where possible. Lower-importance, narrow-spectrum antimicrobials should be used as first line treatment options when antimicrobial agents are deemed clinically necessary.

ASTAG ANTIBACTERIAL IMPORTANCE RATING		
Low Importance: There are a reasonable number of alternative antibacterials in different classes available to treat or prevent most human infections even if antibacterial resistance develops.	**Medium Importance:** There are some alternative antibacterials in different classes available to treat or prevent human infections, but less than for those rated as Low Importance.	**High Importance:** These are essential antibacterials for the treatment or prevention of infections in humans where there are few or no treatment alternatives. These have also been termed "last resort" or "last line" antibacterials.
– Amoxicillin/Ampicillin – Chloramphenicol (topical) – Doxycycline – Neomycin – Procaine penicillin	– Amoxicillin with clavulanic acid – Cephalexin/Cephazolin – Clindamycin – Gentamicin – Metronidazole	– Fluoroquinolones, e.g. enrofloxacin, marbofloxacin, pradofloxacin – Fusidic acid (topical) – Polymyxin B (topical) – Third generation cephalosporins, e.g. cefovecin, ceftiofur

Importance ratings for some antibacterials commonly used in dogs and cats

- Antimicrobial prescribing guidelines provide a useful framework to help inform treatment decisions. A range of prescribing guidelines and tools to support prudent antimicrobial use can be found through the AMR Vet Collective.
- Culture and susceptibility (C&S) results should be used to guide antimicrobial choice whenever possible. If broad-spectrum higher-importance antimicrobial therapy is implemented in critical patients, de-escalation of antimicrobial therapy should occur if indicated when C&S results are available. Clinicians need to reconsider duration of therapy to match the clinical needs of the patient.
- Veterinarians should discuss with owners the importance of antimicrobials to human and animal health and the need to preserve their efficacy through prudent use. Veterinarians should reinforce to pet owners the importance of following the directions for use of any prescribed antimicrobial.

MINIMISING TRANSFER OF RESISTANT ORGANISMS BETWEEN PETS AND PEOPLE

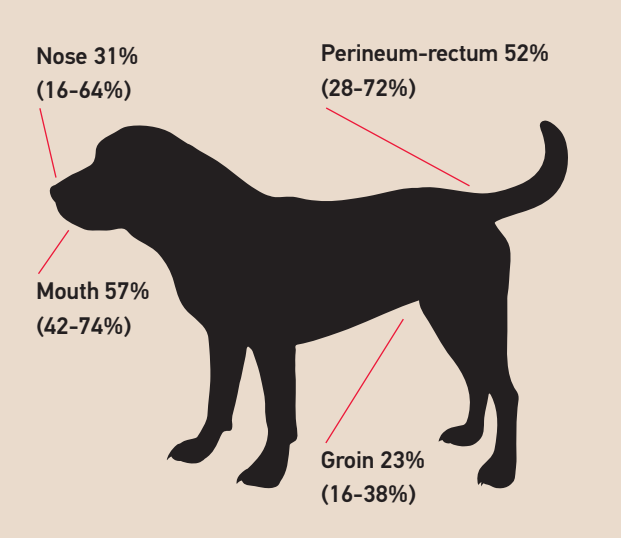
- Good infection control practices are essential to help prevent transmission of potentially zoonotic bacteria between pets and people, whether AMR or not. This encompasses not only hand hygiene but also regular cleaning of contaminated surfaces, as a failure to do either may contribute to transmission of resistant organisms.
- Animals should be bathed after visiting hospitals or aged care facilities to minimise the risk of acting as mechanical vectors. Animals with known AMR infections should not be used in animal assisted therapy programs. For additional information see *Animals in Care Facilities* on page 93.
- For animals with documented active AMR infections additional precautions are recommended:
  - Enhanced infection control should be practiced in the veterinary clinic setting including appropriate isolation and use of PPE (gowns and gloves). Consideration of the pathogen(s) involved and mode of transmission are important in determining the appropriate level of infection control practices (inclusive of PPE and isolation requirements) to be implemented. The AVA Guidelines for Veterinary Industry Personal Biosecurity are a useful resource in this regard.
  - Owners should be counselled to avoid contacting the infected area. Skin lesions or infections should be covered with impermeable dressings to avoid environmental contamination.
  - Thorough homecare instructions should be provided, specifically regarding wound management and environmental cleaning.
  - Contact should be minimised with other animals in the household.
  - Animal faeces should be promptly collected and disposed of.

Staphylococcus spp.

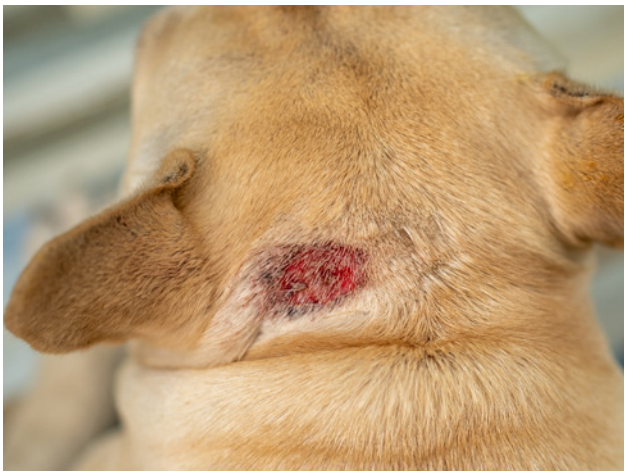
Staphylococci are gram-positive cocci frequently found as commensals on the skin and mucous membranes of mammals and birds. They may also act as opportunistic pathogens, particularly in animals with predisposing conditions, resulting in localised and invasive disease. More than 40 species are described, which are broadly divided into coagulase-positive and coagulase-negative organisms, with the former more commonly associated with infection than the latter.[3] Staphylococci display different host specificities, although cross-species transmission is common.

Staphylococcus pseudintermedius

- *Staphylococcus pseudintermedius* is a common organism colonising cutaneous and mucocutaneous surfaces in dogs and cats. Like other coagulase-positive staphylococci, colonisation is more commonly seen at mucocutaneous surfaces rather than skin. *Staphylococcus pseudintermedius* is more frequently isolated from dogs than cats, where *S. felis* is more common. *Staphylococcus pseudintermedius* colonisation or infection is uncommon in humans.
- Carriage rates of *S. pseudintermedius* in healthy dogs in Australia have been reported from 85.5% (rural Victoria)[4] to 46.2% (remote NSW)[5], and are similar to those from other countries. Carriage rates are highest in the nose, mouth and perineum.[6] In cats, carriage rates of 8.8% (remote NSW) have been reported.[5] Carriage is not typically associated with clinical signs, however opportunistic infections may occur, particularly cutaneous infections where it is the predominant pathogen in over 90% of cases of pyoderma.[7]
- Methicillin-resistance in *S. pseudintermedius* is a more recent phenomenon than in *S. aureus,* with the first reports in Australian dogs in 2014.[8] Methicillin-resistant *S. pseudintermedius* (MRSP) isolates have however been identified in archived samples in Australia dating back to 1999 (USYD archives, J. Norris, unpublished data). MRSP isolates are frequently resistant to a broad range of antimicrobials, including fluoroquinolones. As with *S. aureus,* resistance to beta-lactam antimicrobials in *S. pseudintermedius* is usually due to the presence of the mecA gene (which encodes penicillin-binding protein 2a [PBP2a]).
- Prevalence of MRSP varies depending on study population and methods. MRSP carriage in dogs in Queensland is reported as 8.7% versus 0% in cats.[9] A study from Sydney reported a similar finding, with MRSP carriage in 7% of client owned dogs, 8% of dogs owned by veterinary personnel, and 0% of cats.[10] Studies in remote Indigenous communities failed to identify MRSP in sampled dogs and cats, likely associated with limited access to veterinary care and use of antimicrobials in these communities.[5,11]
- Carriage of MRSP is not associated with clinical signs, however opportunistic infections may result in disease. In Australia, 11.8% of *S. pseudintermedius* submissions from clinical infections were MRSP, with resistant isolates most commonly associated with skin and soft-tissue infections and surgical site infections.[12] Overseas it is reported that up to 65% of *S. pseudintermedius* pyoderma cases are methicillin-resistant.[13]
- Transmission of MRSP from colonised or infected companion animals to humans has been reported but it is thought to be uncommon, with carriage in humans relatively short lived.[14]
- *Staphylococcus pseudintermedius* has been reported in up to 4% of owners of healthy dogs or cats based upon nasal swabbing, with carriage associated with rare or infrequent hand washing after handling pets.[15]
- Infection with *S. pseudintermedius* in humans is rare, most commonly involving local infection of bite wounds. More severe manifestations including bacteraemia, endocarditis, pneumonia, brain abscesses, and otitis have been rarely reported.[16]



Estimated carriage rates of *Staphylococcus pseudintermedius* at different body sites in dogs. Ranges are indicated in parentheses for each site
Adapted from Bannoehr et al (2012).[6]



***Staphylococcus pseudintermedius* is the most common pathogen associated with canine pyoderma**

Staphylococcus aureus

- *Staphylococcus aureus* is a cutaneous and mucocutaneous commensal in humans with approximately 30% of the human population thought to be asymptomatic carriers.[17] Three patterns of colonisation are recognised in humans: persistent colonisation, intermittent colonisation, and non-carriers.
- Methicillin-resistant *S. aureus* (MRSA) is a significant and growing public health concern. Up to 3% of the general population may carry MRSA, predominantly in the nasal passages. Higher rates of carriage are reported in veterinarians, with a 5-fold higher prevalence in veterinarians working with dogs and cats than those with minimal animal contact.[18]
- In humans, a range of presentations of MRSA infection may be seen. Localised infection is more common in people with underlying medical conditions – e.g. peripheral vascular disease or diabetes, and/or a history of hospitalisation. The strains causing this form of infection are usually hospital and long-term care facility associated strains (HA-MRSA). Sequence types ST22 and ST293 are the most prevalent in Australia. Invasive infection usually occurs when an MRSA colonised patient has an invasive procedure and sometimes follows cannulation and secondary line infection. Hence the focus of care is to reduce secondary complications of colonisation, using pre-operative decolonisation and prophylaxis and infection control management to prevent transmission in hospital. More recently, strains of MRSA causing recurrent localised and invasive infections in the community, have become more prevalent. These strains carry an associated virulence factor (PVL) which may enhance pyogenic potential. In Australia, ST93 is the most common. These strains may occur in patients without underlying diseases, including children, and are referred to as community-associated (CA-MRSA) strains. Decolonisation (e.g. using topical decolonisation with nasal mupirocin and chlorhexidine washes) is often used to prevent recurrent infection and intra-familial spread.
- Isolation of *S. aureus* in healthy dogs is considerably less common than *S. pseudintermedius*. One study from rural Victoria reported a prevalence of 14.5%,[4] with most of these animals having dual carriage with *S. pseudintermedius*. This study reported *S. aureus* isolation more commonly in female dogs. Another study in Australia reported a prevalence of 4.3% in dogs and 3.8% in cats in remote NSW.[5] Carriage of *S. aureus* in dogs may represent transient colonisation from cohabitating humans. In 50% of households where *S. aureus* was isolated from both dog and human, the strains were indistinguishable.[15] Interspecies transmission is evident, and although the direction of transfer is not certain, given the strains involved, this is likely to represent human-to-animal transmission. As with *S. pseudintermedius,* carriage of *S. aureus* is generally not associated with clinical signs, however opportunistic infections may occur.
- MRSA carried by dogs are generally human adapted lineages. Several studies have failed to detect MRSA carriage in healthy urban pet dogs, while two studies in dogs from remote communities in NSW and WA have shown a carriage rate of 2.6%, with the sequence types isolated in dogs reflecting the prominent types present in the local human population.[9-11,19] The increased prevalence of MRSA carriage in these dogs likely reflects the comparatively high rate of carriage of MRSA among their owners. A study in healthy pet cats in Brisbane failed to detect MRSA.[9]
- In other studies, being owned by human healthcare workers or being part of a hospital visitation program are risk factors for MRSA carriage in dogs, identifying that the carriage rate in pets reflects the prevalence in humans in their environment.
- Most animals that carry MRSA have no clinical signs, however opportunistic infections may occur. MRSA infection is reported with increasing frequency in companion animals, and is associated with a range of different infections including skin and soft tissue infection, pneumonia, urinary tract infections, and surgical wound infections. Sequence types isolated often correspond to locally prevalent human strains. Nosocomial outbreaks are also reported.[20]

ACAZAP RECOMMENDATIONS

- Resistant skin infections in companion animals are more likely to be MRSP than MRSA, and while MRSP can be transmitted to humans (particularly if there are any predisposing risk factors such as breaks in the skin etc.) it is unlikely. To reduce risk of transmission, owners should minimise contact with areas most likely to harbour *S. pseudintermedius* (e.g. nose, mouth, or perineum), cover open wounds and practice good hand hygiene.
- Dogs and cats are not primary reservoirs of *S. aureus* and colonisation is usually transient. The nose and perineum are high risk sites in pets. Colonisation will usually clear within a few weeks providing re-infection from a common source does not occur. Despite the generally transient nature of colonisation, dogs and cats may be a source of infection for humans. To reduce the risk of transmission, owners should minimise contact with areas most likely to harbour the organism, cover open wounds and practice good hand hygiene.
- There are no validated methods for decolonisation of pets, and therefore this approach is not recommended. In most cases MRSA in dogs and cats will be a result of human-to-animal transmission and colonisation or carriage is likely to be transient.
- Screening of dogs and cats for MRSA is generally not recommended, unless part of an overall strategy to manage recurrent MRSA in people. The clinical implications of carriage in pets may be low.

Enterobacterales

Enterobacterales is an order of gram-negative bacterial rods comprising seven recognised families, including the family *Enterobacteriaceae.*[21] *Enterobacteriaceae* includes the genera *Escherichia, Klebsiella,* and *Salmonella,* with many species normal inhabitants of the gastrointestinal tract of mammals. These organisms may however cause opportunistic infections in susceptible patients or when spread to locations outside the gastrointestinal tract. Some *Enterobacteriaceae* (e.g. *Salmonella* sp., *Shigella* sp.) are primary enteric pathogens.

Extended-spectrum beta-lactamase producing *Enterobacterales* (ESBL-E)

- By virtue of their location, commensal gastrointestinal organisms are exposed to selection pressure from orally administered antimicrobials and are a potential source of resistance genes. A growing concern in veterinary and human medicine are organisms producing extended-spectrum beta-lactamases (ESBLs), enzymes which hydrolyse and render inactive third generation cephalosporins. As ESBL resistance is carried on a plasmid, this can be easily transferable to other species of *Enterobacterales.* Presence on plasmids allows for the accumulation of other resistance factors. Hence ESBL resistance is frequently associated with co-resistance to other classes of antimicrobials, including fluoroquinolones and aminoglycosides.
- In humans the most common species carrying ESBL enzymes are *E. coli, Klebsiella pneumoniae* and *Enterobacter cloacae.* Presence of ESBL enzymes in *Salmonella* species is also a concern as this has considerable zoonotic potential.
- ESBL-E are a very uncommon cause of disease in dogs and cats in Australia.[22]
- Eating raw meat and recent antimicrobial treatment has been reported overseas as a risk factor for carriage of ESBL-E in dogs,[23,24] and a study of commercially available raw food diets for dogs in Sweden found *E. coli* in all tested samples (n=39), with ESBL isolated from 23%.[25] Owing to differences in antimicrobial prescribing and animal husbandry in Australia compared to other regions, the prevalence of ESBL-E in Australian production animals is low and therefore transmission to, and carriage in dogs and cats is likely to be very uncommon.[26,27]

Carbapenemase-producing *Enterobacterales* (CPE)

- Carbapenems are beta-lactam antimicrobials frequently used as a last-line treatment for severe infections in human medicine. Consequently, they are classified as highly important antimicrobials. Carbapenemase-producing *Enterobacterales* (CPE) are of increasing concern worldwide as the presence of these enzymes may render the organism virtually untreatable with currently available antibiotics. Because of concern for spread, CPE incidence in human infection in Australia is reportable.
- CPE have not been reported in dogs in Australia, and there is a single report of carbapenem resistant *Salmonella enterica* isolated from a systemically unwell cat and three cohabitating cats in the same facility.[28] The off-label use of carbapenems in dogs and cats is uncommon in Australia, with a review of over 4 million consultations, including almost 600,000 antimicrobial prescribing events failing to identify the use of this class of antimicrobial.[2] Despite this, CPE may develop in the absence of carbapenem use through co-selection of carbapenem-resistance associated with the use of other antimicrobials.
- Human CPE infections are mostly associated with prolonged hospitalisation and underlying diseases. Infections with enzymes such as KPC can result in up to 50% mortality. Enzymes such as NDM and OXA-48 have become increasingly prevalent, especially in parts of South and East Asia. All patients who have been recently hospitalised overseas are screened on admission in Australia, in order to avoid transmission and potential outbreaks.
- Occurrence in Australia is still relatively uncommon, with less than 1% prevalence in surveillance studies of blood-stream infections,[29] but some enzymes such as IMP-4 are locally endemic and have been found in environmental sources such as hospital drains and waste-water and in cats and wild birds in Australia.[28,30]

ACAZAP RECOMMENDATIONS

- Although the risk of ESBL-E in raw meat in Australia is very low, due to the risk of transmission of other potential pathogens (e.g. *Campylobacter, Salmonella),* it is recommended to avoid feeding raw meat diets to dogs and cats, or if fed, consider the potential for zoonotic infection through contact with the diet or the faeces of animals which have consumed the diet.
- Good hand hygiene is essential following contact with animals, animal food or treats, food bowls, animal bedding and animal faeces.
- Currently there is no known role of dogs and cats in transmission of CPE in Australia, however the possibility for human-to-animal transmission exists.

Clostridioides difficile

- *Clostridioides difficile* is a gram-positive anaerobe and the most common cause of hospital-acquired antimicrobial diarrhoea in people. *Clostridioides difficile* infection (CDI) is related to toxin production, not the mere isolation of the organism in culture or by molecular testing. Different strains are identified that vary in virulence due to differential production of toxins.
- *Clostridioides difficile* has been isolated from healthy dogs and cats, and those with diarrhoea. Globally, carriage of *C. difficile* in healthy adult dogs has been reported to be between 0-6%.[31] Carriage rates in healthy cats are thought to be similar to dogs.[31,32]
- Higher rates of carriage are reported in dogs that visit human hospitals, have contact with children, or reside with immunocompromised owners. Recent hospitalisation or out-patient veterinary care, and treatment with antimicrobials is also associated with increased carriage.[31]
- The role of *C. difficile* in infectious canine and feline gastrointestinal disease is unclear.
- There is low prevalence of *C. difficile* in healthy humans (except neonates, where *C. difficile* carriage is not uncommon), with increased risk of carriage of toxin positive strains and secondary *C. difficile* colitis associated with prolonged hospitalisation and prior antimicrobial therapy.
- Disease in humans may range from mild to fulminant and potentially fatal pseudomembranous colitis or toxic megacolon.
- Some strains are found in both humans and dogs suggesting interspecies transmission, however the direction of transmission is unclear (animal-to-human or vice versa).

ACAZAP RECOMMENDATIONS

- The zoonotic potential of *C. difficile* is unclear, and infection in cohabitating companion animals and humans may represent zoonotic transmission or a common source of exposure.
- All diarrhoeic animals should be considered potential sources of transmission and appropriate infection control procedures implemented.

KEY CONSIDERATIONS

1. A One Health approach is essential in tackling the issue of AMR as bi-directional cross-species transmission of organisms/genes from animal-to-human or vice versa may occur.
2. Good hand hygiene practices following contact with animals, animal food or treats, food bowls, animal bedding and animal faeces can minimise the zoonotic transmission of AMR. Additional precautions should be taken, both in the clinic and home environment, for animals with documented AMR infections.
3. The primary drivers of AMR are antimicrobial use and poor infection control practices.
   - Veterinarians should follow prudent use guidelines and avoid where possible the use of antimicrobials of high importance, such as fluroquinolones and third generation cephalosporins.
   - Clinics should have agreed and documented infection control practices that consider hand hygiene, environmental hygiene, and the appropriate use of PPE.

The primary drivers of AMR are antimicrobial use and poor infection control practices.

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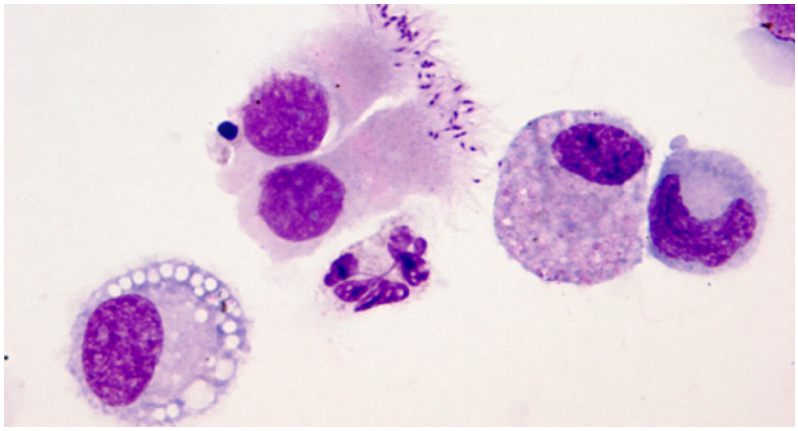
Bordetella bronchiseptica

- *Bordetella bronchiseptica* is a respiratory pathogen of a range of wild and domestic animals. It is a common primary pathogen of canine infectious respiratory disease complex (CIRDC) and a causative agent in feline upper respiratory tract disease.
- *Bordetella bronchiseptica* is closely related to the host-specific human pathogens *B. pertussis* (the cause of whooping cough) and *B. parapertussis.*

ACAZAP RECOMMENDATIONS

- Good hand hygiene following animal contact or work in animal facilities is essential.
- Advise owners on the potential zoonotic risk of kissing animals or allowing them to lick faces.
- Acquisition of animals with a lower likelihood of *B. bronchiseptica* carriage (older, from low population density environments) should be considered for at risk individuals.
- Good ventilation and air exchange are essential in animal care facilities (e.g. kennels and shelters) to minimise expose of staff and animals in the facility to infectious aerosols produced by infected (clinical or asymptomatic) dogs.
- Vaccination of dogs using mucosal (oral or intranasal) vaccines to reduce likelihood of shedding is recommended.
- Although confirmed disease from modified live canine *B. bronchiseptica* vaccines has not been reported in humans, prudent practice would ensure immunocompromised individuals are not present at the time of vaccination. Oral vaccination is likely to result in reduced aerosolisation compared to intranasal administration.

IN ANIMALS



Bronchoalveolar lavage cytology (Diff-Quik stained) from a dog infected with *B. bronchiseptica* showing numerous coccobacilli adhered to the cilia of columnar epithelial cells
(Courtesy of Prof. Michael Scott, Michigan State University)

AETIOLOGY AND EPIDEMIOLOGY

- *Bordetella bronchiseptica* is a gram-negative aerobic coccobacillus found in a range of animals where it is associated primarily with upper respiratory tract infections. In severe cases *B. bronchiseptica* may be involved in lower respiratory tract infections, albeit rarely.
- Although frequently isolated from healthy animals, *B. bronchiseptica* is not part of the normal flora. Prolonged carrier status is common in dogs and cats following clinical or subclinical infection. The organism colonises the ciliated respiratory epithelium, inducing paralysis of the mucociliary apparatus (ciliostasis) rendering the respiratory tract susceptible to secondary bacterial colonisation and subsequent inflammation.[1]
- Dogs and cats are infected through oronasal exposure (direct or indirect) to infectious respiratory secretions from shedding animals.

Kennels and other situations of high population density are a risk factor for *Bordetella bronchiseptica* transmission in dogs

- *Bordetella bronchiseptica* has been demonstrated to survive and even proliferate in the environment under the right conditions.[2] A role for environmental amoeba in the maintenance of *B. bronchiseptica* has been proposed, however the epidemiological or clinical significance of this is not known.[3]

PREVALENCE AND RISK FACTORS

- No peer-reviewed Australian-specific prevalence data is available for *B. bronchiseptica* infection in dogs, however prevalence in clinical submissions to a commercial reference laboratory in Australia was reported as 12.3% in dogs (n=122).[4] The same laboratory reported a prevalence in clinical submissions of 6.9% (n=521) for cats,[4] while a more recent publication reported a prevalence of approximately 10% in submitted feline samples.[5]
- Risk factors for infection and clinical disease in dogs include young age and increased population density.
- One of the most significant risk factors for feline upper respiratory tract disease (including *B. bronchiseptica*) is time spent in shelters. Other environments where cats are housed at high densities and multi-cat households have demonstrated higher prevalence of *B. bronchiseptica* infection.[6,7]
- Data suggests contact with dogs with respiratory disease is a risk factor for *B. bronchiseptica* infection in cats.[6]

CLINICAL DISEASE

- Asymptomatic carriage in dogs and cats is common, however multiple studies have demonstrated higher prevalence in animals with acute upper respiratory tract signs.
- Dogs with clinical signs typically present with acute tracheobronchitis, manifesting with a dry hacking cough. Bronchopneumonia has been reported, with puppies and young dogs particularly at risk.[8]
- Affected cats usually present with acute upper respiratory tract signs of variable severity, including sneezing, ocular discharge, and coughing. Coughing is less common in affected cats compared to dogs. Bronchopneumonia may be seen in young kittens.[6]
- Prolonged shedding post-recovery is common in both dogs (14 weeks)[9] and cats (19 weeks).[10]

DIAGNOSIS

- Diagnosis of *B. bronchiseptica* was traditionally reliant upon bacterial culture, however PCR testing, frequently as part of a multiplex panel of respiratory pathogens, has become more common.
- Due to the high rate of carriage of *B. bronchiseptica* in healthy dogs, PCR results should be interpreted in the context of relevant historical and clinical findings. Dogs may also test positive for several weeks following vaccination with modified live mucosal vaccines.

PREVENTION

- Vaccination against *B. bronchiseptica* is available for dogs in Australia, however no feline vaccine is available. Both modified live mucosal vaccines (intranasal or oral administration) and inactivated cell antigen extract parenteral (injectable) vaccines are available for dogs.

IN ANIMALS *continued*

- Both mucosal and parenteral vaccines aid in the prevention of disease and the reduction in clinical signs, however mucosal vaccines are recommended by the WSAVA Vaccination Guidelines Group due to their ability to stimulate local mucosal immunity.[11] Neither vaccine type provides sterilising immunity, however, mucosally vaccinated dogs have been demonstrated to have reduced shedding of virulent organisms post-challenge compared with parenterally vaccinated dogs.[12]

TRANSMISSION

- Transmission to humans is via oronasal exposure to infectious respiratory secretions (direct or indirect).
- Airborne nosocomial transmission has been documented.[13]

IN HUMANS

Oronasal exposure to respiratory secretions should be avoided

PREVALENCE AND RISK FACTORS

- Despite frequent human exposure to *B. bronchiseptica,* zoonotic infections are rare and are typically associated with either pre-existing localised lower airway disease (e.g. patients with bronchiectasis) or underlying immunodeficiency syndromes.
- Predisposing conditions include cystic fibrosis, bronchiectasis, HIV infection, and solid organ transplantation.[14] Rarely, cases have been reported in patients with no identified risk factors.[15,16]

CLINICAL DISEASE

- Clinical disease is primarily respiratory in nature and varies in severity from acute sinusitis and bronchitis with mild tracheobronchitis to acute fulminant bronchopneumonia.[14]
- Other reported systemic disease manifestations include septicaemia, endocarditis, meningitis, and peritonitis.[16-18]
- Although infection with *B. bronchiseptica* from modified live canine vaccines is considered a theoretical possibility, no laboratory confirmed cases of human infection have been reported despite more than 30 years of vaccine use in veterinary patients. One case report described a temporal association between exposure and development of a cough of undetermined aetiological cause.[19] Another case report described a solid organ transplant recipient with laboratory confirmed *B. bronchiseptica* pneumonia after contact with a recently vaccinated dog.[20] In this case the dog had recently been in a high risk environment for virulent *B. bronchiseptica* exposure (boarding), and no attempt was made to determine if the isolate from the patient was a vaccine strain or field strain.

KEY CONSIDERATIONS

1. Despite frequent human exposure to *B. bronchiseptica,* zoonotic infections are rare, and typically associated with significant immunocompromise.
2. Vaccination of dogs using mucosal (oral or intranasal) vaccines to reduce likelihood of virulent *B. bronchiseptica* shedding is recommended.
3. Although laboratory confirmed cases of human infection associated with modified live canine *B. bronchiseptica* vaccine strains have not been documented, it would be prudent to ensure immunocompromised individuals are not present at the time of vaccination.

Owners should be advised as to the importance of good hand hygiene and educated on the potential zoonotic risk of kissing animals or allowing them to lick faces.

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BRUCELLOSIS *(Brucella suis)*

- *Brucella* are gram-negative, facultative intracellular coccobacilli found in a number of different wild and domestic host species.
- In humans, cases of brucellosis acquired in Australia are due to *B. suis* and result from occupational or recreational exposure to feral pigs through hunting. Cases of brucellosis diagnosed in Australia due to other *Brucella* species (e.g. *B. melitensis*) are always acquired overseas.

ACAZAP RECOMMENDATIONS

- Consider brucellosis as a differential in dogs with back pain, joint pain, discospondylitis, orchitis/epididymitis or a history of abortion, particularly if there is a history of hunting or feeding raw feral pig meat.
- Good hygiene in conjunction with personal protective equipment (e.g. gloves, covering cuts and abrasions with waterproof dressings, protective eyewear, disposable face masks and gowns) is essential for veterinary staff performing procedures on pig hunting dogs. Particular care should be taken when collecting blood, neutering or assisting with whelping or caesarean sections for breeding bitches.
- Laboratory staff should be alerted about potentially infectious *B. suis* containing samples. Clearly mark lab specimens from pig hunting dogs to protect laboratory staff so they can take adequate precautions when handling specimens, inoculating media and agar plates.
- Veterinary staff should counsel the owner on the zoonotic risk posed by infected dogs. Risk assessment, family screening, and contact tracing should be performed as required.

Feral pigs in northern Australia

- Given the potential for zoonotic transmission, euthanasia of affected dogs should be considered, but this is not mandatory.[1] If euthanasia is declined, treated dogs should be neutered and undergo regular blood and urine culture. Serology may be monitored (using the complement fixation test) for a rising titre as an indicator of recrudescence.
- Test all dogs in contact with an affected dog and consider them infectious until negative.
- Consider testing dogs with a history of hunting or feeding raw feral pig meat prior to performing invasive procedures (even if asymptomatic).
- The use of dogs in the recreational activity of feral pig hunting is common in parts of Australia. Proactive advice should be provided to pig hunters on risk management with particular emphasis on cautious handling of feral pig carcasses. Other recommendations for pig hunters include:
  - Pregnant women and children should not participate in pig hunting activities and should avoid contact with pig hunting dogs as they are at greater risk of severe disease.
  - Routine hand hygiene, preferably with soap and running water, is important. Alcohol-based hand sanitiser may be used when hand washing facilities are not available.
  - Use clean, sharp knives to minimise the risk of self-injury. If cut or scratched, immediately clean the wound and protect it with waterproof dressings or gloves.
  - Tools, boots and surfaces should be thoroughly cleaned with a disinfectant. Vehicles used to transport carcasses should be cleaned with soapy water. High pressure hosing should be avoided to minimise aerosolisation.
  - Avoid opening reproductive tissues or swollen joints of pigs. Personal protective equipment should be worn when handling or disposing of reproductive organs or tissues of feral pigs and where possible carcasses should be burned or buried.

Care of pig hunting dogs

- Do not breed from dogs suspected or known to be infected with *B. suis.*
- Wash dogs and associated protective devices after hunting, preferably prior to leaving the hunting site. This should be performed away from other people and while wearing PPE.
- If dogs are wounded during a hunt, use personal protective equipment when cleaning or dressing wounds and seek veterinary advice. Untreated traumatic wounds can result in serious welfare implications and poor health outcomes including severe pain, sepsis and potential fatalities.
- Do not feed dogs raw feral pig meat, bones, offal, foetuses, or reproductive tissues. Pig meat can be rendered safe for consumption by thorough cooking. Note that freezing, smoking, drying and pickling of meat is inadequate to inactivate *Brucella*.

Bull Arab pig hunting dog (Courtesy of Arthur Zambellakis)

IN ANIMALS

AETIOLOGY AND EPIDEMIOLOGY

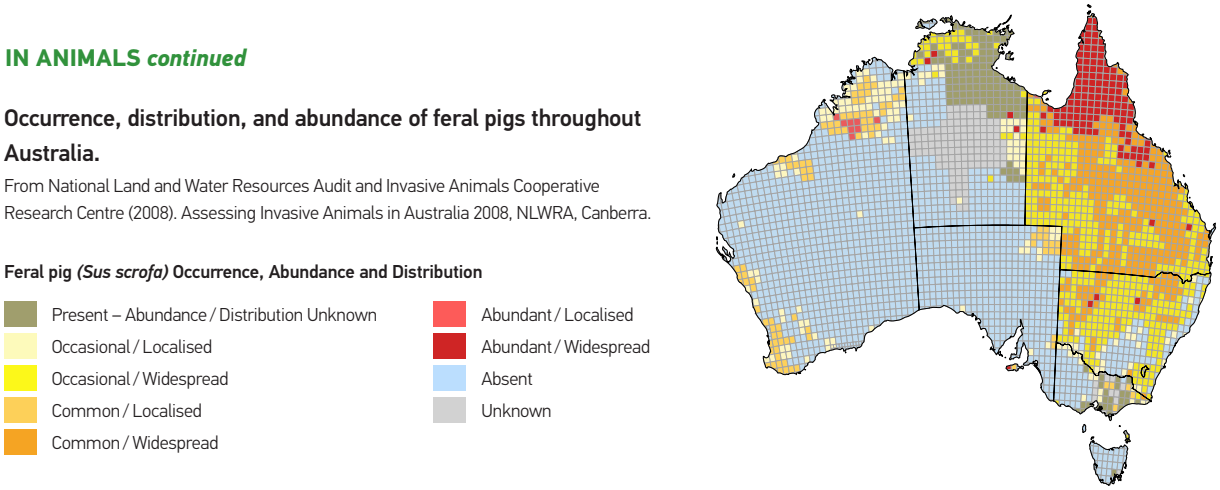
- There are currently twelve recognised species of *Brucella* identified, that can be divided into classical *Brucellae* species *(B. abortus, B. melitensis, B. canis, B. ovis, B. neotomae* and *B. suis)*, marine mammal species *(B. ceti* and *B. pinnipedialis)* and recently identified species considered 'atypical' *(B. inopinata, B. microti, B. paponis* and *B. vulpis).* There are additional *Brucella* strains awaiting genus affiliation.[2,3]
- The six classical *Brucella* species are highly genetically related to each other.[3] Of these species, *B. suis* is enzootic in Australia, whilst *B. melitensis, B. canis* and *B. abortus* are exotic, the latter having been eradicated in 1989.[4] *Brucella ovis*, which is present in Australia, is not considered zoonotic. *Brucella neotomae* is a rarely identified zoonotic disease in the literature.[5]

Six classical *Brucella* species

SPECIES	PRIMARY ANIMAL RESERVOIR	DISEASE STATUS IN AUSTRALIA	ZOONOTIC RISK
B. suis	Pigs	Enzootic in Australia. Present in feral pigs, dogs fed raw feral pig meat or involved in feral pig hunting can become infected	Feral pig hunters and their families, veterinarians, dog breeders, laboratory workers and abattoir staff
B. melitensis	Sheep, goats, camels	Exotic, not known to occur in sheep and goats in Australia	Exotic zoonotic disease. Travellers and migrants from countries where *B. melitensis* occurs in animals are at risk
B. abortus	Cattle	Eradicated from Australia in 1989[4]	Exotic zoonotic disease. Travellers and migrants from countries where *B. abortus* occurs in animals are at risk
B. canis	Dogs	Exotic, not known to occur in dogs in Australia	Significant zoonotic pathogen globally, not identified in Australia
B. ovis	Sheep	Enzootic in Australia, all breeds of sheep are susceptible	Not considered zoonotic[4]
B. neotomae	Rodents	Exotic	Rare cases reported[5]

Occurrence, distribution, and abundance of feral pigs throughout Australia.

From National Land and Water Resources Audit and Invasive Animals Cooperative Research Centre (2008). Assessing Invasive Animals in Australia 2008, NLWRA, Canberra.



- *Brucella suis* usually infects pigs and is enzootic in the feral pig population in Queensland and northern NSW. The seroprevalence of *B. suis* in feral pig populations in NSW is estimated at 3%, with cases found only in northern regions.[6] In Queensland seroprevalence in pigs has been reported as 4%.[7]
- *Brucella suis* results in widely disseminated infection in pigs. The organism is found in blood, tissues and urine, with particularly high levels in reproductive tissues/placentae. The most common signs in pigs are reproductive losses (abortion, stillbirths, weak live-born piglets), however these are unlikely to be noticed with the unmanaged husbandry of feral pigs. Orchitis and epididymitis may be seen in boars. Non-pregnant pigs are frequently asymptomatic, although some animals may develop arthritis, discospondylitis, or complications from abscess formation in other tissues and organs.[8]
- Organisms may remain viable in moist environments protected from direct light for months. Epidemiological significance of this for transmission to dogs is unclear. *Brucellae* may survive for years in frozen meat.
- Dogs are infected via ingestion, inhalation or exposure to mucous membranes, conjunctiva, or abraded skin following exposure to infected tissue.
- Dog-to-dog sexual transmission is considered possible.

PREVALENCE AND RISK FACTORS

- For dogs, exposure to feral pigs is the biggest risk factor. This may either be through hunting feral pigs, through being fed raw meat/offal from feral pigs or through indirect exposure to materials contaminated with bodily fluids or tissues from feral pigs.
- Seroprevalence of *B. suis* in pig hunting dogs has been investigated in a number of studies. The survey adjusted true seroprevalence for *B. suis* in dogs from pig hunting households across NSW and southern Queensland is 9.4%. In NSW, seropositive dogs were found mainly in the north and central west of the state. (Pers. comm. Cathy Kneipp, PhD scholar). In another study in north Queensland, 1% (1/97) of clinically healthy pig hunting dogs were found to be seropositive on both compliment fixation and rose bengal testing.[9]
- *Brucella suis* has not been reported in cats.

CLINICAL DISEASE

- Clinical signs in dogs are referable to the reproductive tract (orchitis, prostatitis, abortion), axial skeleton (discospondylitis) or appendicular skeleton (lameness), however infection may be subclinical. In one study, 40% of dogs testing positive (rose bengal test) were subclinical, while of the clinical cases 33% dogs exhibited reproductive signs, 13% had back pain and 10% had lameness.[10]
- High numbers of organisms are found in reproductive organs and placental tissue and fluids. It is likely that some pig-hunting dogs experience reproductive issues caused by *B. suis* infection.[11]
- Data on the duration of bacteraemia is limited, however as many cases are culture negative, it may be short. *Brucella suis* has been cultured from the semen of affected dogs.
- Affected dogs should be neutered. They may be treated with combination antimicrobial therapy (doxycycline and rifampicin) however the organism may persist in treated dogs and recrudesce, with resultant zoonotic implications. Euthanasia should therefore be considered.[1,10]

DIAGNOSIS

- In dogs, serology, based on cross reactivity against *B. abortus* (exotic to Australia), can be used to determine exposure to *B. suis*. Dogs may be seronegative early in the course of infection, and clinically suspicious cases should be retested 6 weeks later.
- Culture from tissue or blood can be used to confirm diagnosis, however many cases are culture negative.
- Diagnostic testing is restricted to state government laboratories who are able to perform serology using the rose bengal test, with confirmatory complement fixation testing. Practitioners are advised to contact their respective state government bodies for the latest information on testing suspected cases.
- Veterinary staff should wear appropriate PPE when collecting samples from dogs with suspected infections, and laboratory staff should be notified that specimens are potentially infected with *Brucella*.

TRANSMISSION

- *Brucella* spp. infect humans as incidental hosts, with a low infectious dose (estimated at 10-100 organisms) required for transmission.[4]
- Human infection occurs through exposure or direct contact with tissues or blood from infected animals, including placental tissues or fluids.
- Aerosol transmission is also possible, particularly in laboratory environments.
- Faeces from dogs fed meat/offal from feral pigs may be a potential source of infection as ingested organisms may remain viable during transit through the gastrointestinal tract.

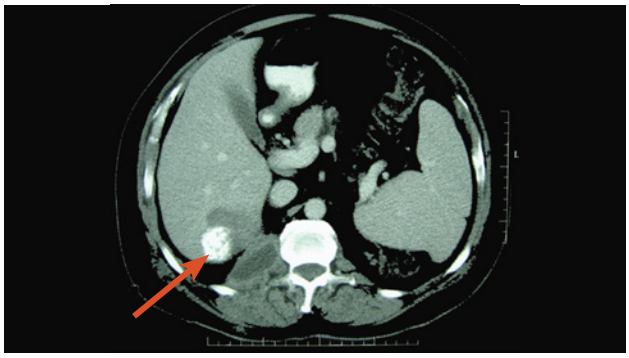
IN HUMANS

PREVALENCE AND RISK FACTORS

- In Australia between 1991 and 2019 an average of 30 human *B. suis* cases were reported annually, with most cases occurring in Queensland (80%).[4]
- Most reported cases are due to occupational or recreational exposure to feral pigs through hunting and pig hunting dogs. Based on conservative estimates, there are more than 156,000 adult pig hunting dogs in Australia, at an average of three adult dogs per hunter.[11]
- The main patient risk groups for *Brucella suis* infection are:[4]
  - Feral pig hunters and their families – direct or indirect exposure to feral pigs or their tissue products via skin abrasions and mucous membranes through the slaughter process and exposure to infected dogs.
  - Veterinarians and veterinary staff – exposure to infected dogs, especially during reproductive or obstetric surgery.
  - Dog breeders – exposure to reproductive tissues and fluids from whelping bitches.
  - Microbiology/laboratory staff – aerosol transmission, individuals working in microbiological facilities who handle *Brucella* cultures.

CLINICAL DISEASE

- *Brucella* infection (brucellosis) may be asymptomatic or symptomatic. Disease in humans can be multi-system but most typically presents with non-specific flu-like symptoms (fever, fatigue, myalgias, arthralgia) which may be relapsing or protracted.[12]
- Cardiovascular complications may include endocarditis, myocarditis, pericarditis and infected aortic aneurysms.[13]
- Osteoarticular involvement, usually seen as sacroiliitis in younger patients or vertebral infection (spondylitis, discitis and osteomyelitis) in older patients, is the most frequent complication of brucellosis (40% of cases).[13]
- Neurological involvement (neurobrucellosis) can occur at any stage of the disease with meningitis the most frequent central nervous system complication reported, estimated to occur in 5% of clinical cases.[13]



Chronic hepatic brucellosis - calcified granuloma (arrow) with surrounding abscess, perforation into psoas and psoas abscess

- Genitourinary involvement (orchitis and epididymitis), and granulomatous hepatitis can also occur.
- Human mortality is low (case fatality rate of 1-2%) and often related to cardiovascular complications.[4]
- *Brucellae* can infect human chorioamniotic tissue at any stage of pregnancy, leading to obstetric complications including foetal death and abortion.[14]
- Recurrent infections and relapses may occur in up to 10% of patients.[4] Some cases can manifest decades after primary exposure.
- There is no evidence that disease is more likely or more severe in the young or old.
- If human *Brucella* infection is suspected, serology and blood cultures are recommended. As *Brucella* is a laboratory hazard, identification must be performed in laboratories with appropriate facilities, and thus diagnosis may be delayed in regional or remote areas serviced by smaller laboratories. MALDI-TOF and PCR may be used for microbial identification.
- Human-to-human transmission thought to be rare with casual contact.

KEY CONSIDERATIONS

1. Zoonotic infection occurs primarily through occupational or recreational exposure to feral pigs or their products.
2. Infection of pig hunting dogs in enzootic regions is reported, with seroprevalence as high as 9%.
3. Veterinary clinic staff should take appropriate precautions when performing procedures, particularly reproductive procedures, on pig hunting dogs.

Proactive advice and education should be provided to pig hunters on risk management strategies.

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Campylobacteriosis (*Campylobacter* spp.)

- *Campylobacter* are spiral to curved gram-negative bacterial rods. There are numerous species in the genus, many of which are considered normal components of the microbiota of wild and domestic animals, and some of which are reported to cause disease in animals or humans.
- *Campylobacter* are the most common cause of notifiable diarrhoeal illness in humans in Australia, with approximately 30,000 cases reported annually. The true human disease burden is however estimated at 10 times this.[1]
- Although most human cases are foodborne (estimated 77% of cases)[2], transmission directly or indirectly from companion animals, particularly young animals, can occur.

ACAZAP RECOMMENDATIONS

- Hand hygiene immediately after contact with animals (including pet reptiles and amphibians), animal food or treats, animal bedding and animal faeces is essential.
- Avoid feeding raw meat diets to dogs and cats. If fed, consider the potential for human infection from either contact with the food or the faeces of pets consuming such diets.
- Animal faeces should be collected and disposed of immediately.
- Appropriate cleaning and disinfection of bowls and contact surfaces should be regularly performed.
- Maintain cats indoors to reduce risk of predation on and transmission from wildlife.
- *Campylobacter* infection during pregnancy can result in significant complications. Pregnant women should be advised of the risk and informed of the precautions that can be taken to prevent infection, such as avoiding contact with raw food diets or pets with diarrhoea. Disposal of pet faeces and litter tray cleaning should be undertaken by other members of the household. If not possible, adequate hand hygiene protocols should be followed.
- Antimicrobial treatment is generally not required for dogs and cats with *Campylobacter* infection, as infection is self-resolving and rarely serious.
- Screening healthy animals for *Campylobacter* is not recommended.
- In a veterinary clinic or animal facility setting (e.g. kennels, shelters) isolation of all animals with documented or potentially infectious diarrhoea is recommended.

IN ANIMALS

AETIOLOGY AND EPIDEMIOLOGY

- The most commonly isolated *Campylobacter* species in dogs and cats is *C. upsaliensis*, a canine adapted species, which may be considered part of the normal flora. Other species isolated include *C. jejuni, C. coli,* and *C. helveticus.* Animals may be co-infected with multiple *Campylobacter* species.
- *Campylobacter* is frequently isolated from meat (human grade) in Australia, with a recent study detecting *Campylobacter* in 90% of samples of chicken meat and 73% of chicken offal. Lower rates of detection were seen in lamb (38%), pork (31%) and beef (14%) offal.[3]
- Companion animals are infected via the faecal-oral route through ingestion of contaminated water, raw or undercooked food, or through direct or indirect contact with faeces from affected animals or people.

PREVALENCE AND RISK FACTORS

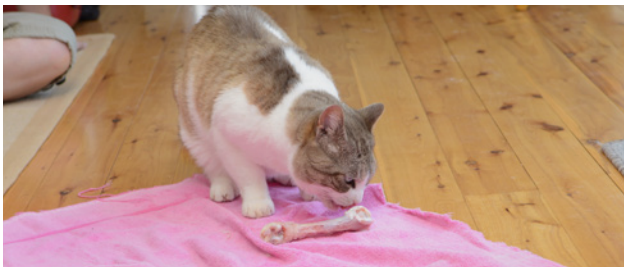
- *Campylobacter* are frequently identified in canine and feline faeces. A meta-analysis of 34 published studies reported a global weighted mean prevalence of approximately 25% in both dogs and cats.[4] A study in South Australia demonstrated *Campylobacter* carriage in 43% of dogs (34% *C. upsaliensis,* 7% *C. jejuni*, 2% *C. coli*) and 15% of cats (11% *C. upsaliensis*, 4% *C. jejuni*), however research using more modern molecular techniques is needed.[5] More recently a study in dogs and cats with diarrhoea reported *Campylobacter* spp. in 47.6% of cats and 36.3% of dogs.[6]

Computer-generated recreation of a cluster of *Campylobacter* bacteria based upon scanning electron microscopic imagery
(Public Health Image Library, CDC)

- Risk factors associated with increased prevalence and shedding:[7,8]
  - Age: young animals (less than 6 months of age) are more frequently infected than older animals.
  - Population density: dogs and cats housed in higher population density environments (shelters, catteries etc.) have higher prevalence of *Campylobacter*.
  - Diet: feeding of raw meat diets.
  - Outdoor access: in cats, outdoor access is associated with greater prevalence of shedding.
  - Presence of pre-existing or concurrent intestinal diseases, including the presence of other pathogens, protozoa and helminth parasites.

CLINICAL DISEASE

- The role of *Campylobacter* in gastroenteritis in dogs and cats is unclear, with conflicting results in the published literature. These discordant findings likely relate to differences in the infecting species and host factors such as age, stress, or co-infection. Mild, self-limiting gastroenteritis is the most frequently reported sign, however asymptomatic carriage of *Campylobacter* is common (43% in dogs).[5]
- Rarely, extraintestinal manifestations of disease are reported in dogs, including cholangiohepatitis/cholangitis and abortion.[8] A study from Melbourne University suggested a link between *Campylobacter* infection and acute polyradiculoneuritis (APN) in dogs, with affected dogs 9.4 times more likely to be shedding *Campylobacter* (as determined by faecal culture) than matched control dogs,[9] however the findings of this study have been contested.[10]
- Uncomplicated campylobacteriosis tends to be mild and self-limiting. Supportive treatment may be required in some cases, however antimicrobial treatment is generally not required in dogs and cats. Resistance to commonly used antimicrobials has been demonstrated in some *Campylobacter* isolates.[8]



Raw chicken is frequently contaminated with *Campylobacter*

DIAGNOSIS

- Species-specific assays to identify potentially pathogenic and zoonotic *C. jejuni* or other relevant *Campylobacter* spp. should be considered. Population level research of both healthy dogs and cats, and those with diarrhoea, is required to assess the range of species present and their role in clinical or zoonotic disease.
- Shedding may be intermittent and transient, however persistent infection and shedding has been reported. Shedding of host adapted *C. upsalensis* in dogs is more protracted and consistent than *C. jejuni*.

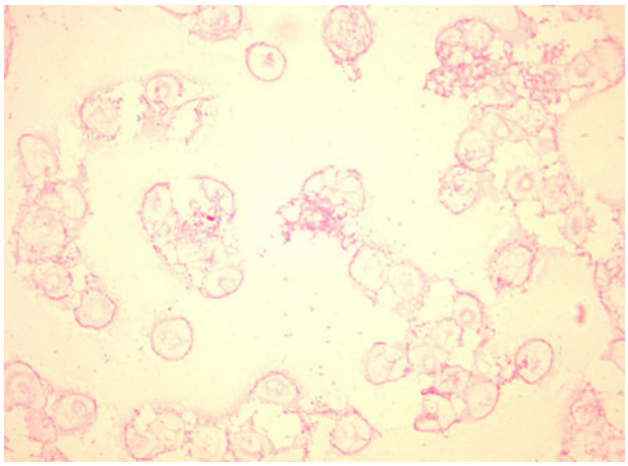
TRANSMISSION

- Humans can be infected via faecal-oral transmission through:
  - handling of human grade meat contaminated with *Campylobacter*.
  - handling and feeding raw pet food diets and treats, including handling of bowls and food preparation materials.
  - direct contact with pets or faeces or indirect contact with materials contaminated by faeces.

IN HUMANS

PREVALENCE AND RISK FACTORS

- Campylobacteriosis is mainly considered food-borne, with an estimated 77% of cases transmitted through food consumption in Australia.[2] Most outbreaks are linked to poultry as the primary source, with infection seasonal in temperate climates.[11]
- The two most common species causing human disease are *C. jejuni* and *C. coli*. Other *Campylobacter* species, including *C. lari, C. upsaliensis,* and *C. fetus*, may cause infection in humans, although these are more sporadic.
- PCR can detect a broader range of *Campylobacter* as some species are difficult to culture under routine conditions.[11] However this depends on the assay, as the targets included in PCR assays vary. Because of the limitations of culture diagnosis, PCR-positive, culture-negative results may be seen in 10-30% of cases.[12]

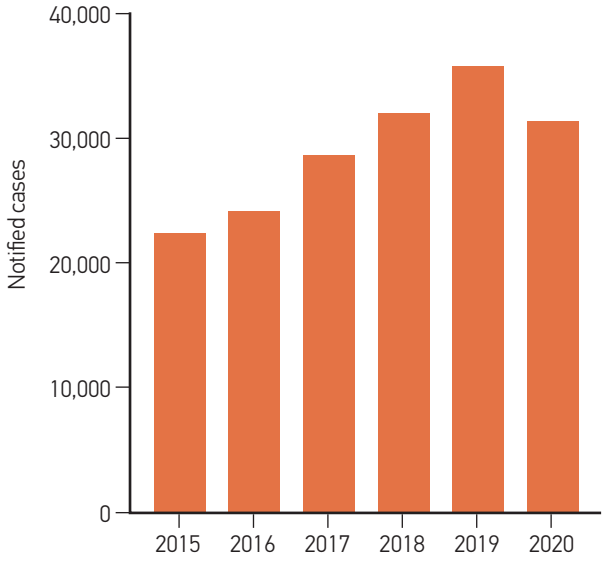


Gram stain of *Campylobacter* from blood culture

- In addition to direct contact with pets or indirect contact with a contaminated environment, other risk factors for human infection include consumption of contaminated meat, milk or water and international travel.
- The incidence of notified campylobacteriosis cases in Australia is 124.6/100,000 (2020 data – National Notifiable Diseases Surveillance System), with an estimated 10 cases for every notified case within the community.[1]

CLINICAL DISEASE

- The incubation period in humans is reported to be 2-5 days, with a longer incubation period in children, and a shorter incubation period associated with higher challenge doses.
- Gastrointestinal infection with *Campylobacter* can occur in any age group. In healthy immunocompetent individuals, infection may be asymptomatic or result in acute, self-limiting illness associated with diarrhoea, fever, and abdominal pain. More severe and persistent disease is seen in immunocompromised patients.



Campylobacteriosis notifications by year in Australia (2015 to 2020) from National Notifiable Diseases Surveillance System. Data accessed April 2021.

- Extraintestinal infection may occur through ascending infection (cholecystitis, pancreatitis) or bacteraemia (meningitis, pneumonia). Extraintestinal infection is more common in the very young or aged, or patients with primarily T-cell related immune deficiencies.[13]
- Post-infection campylobacteriosis complications may include irritable bowel syndrome, reactive arthritis and Guillain-Barre syndrome, a neurological disorder primarily affecting peripheral nerves. These are estimated to occur in 8.8%, 7%, and 0.03% of cases respectively.[14] Additionally, cardiovascular complications (myocarditis) and reproductive complications due to intrauterine infection have been reported.[15]
- *Campylobacter* infection in humans is notifiable in all states and territories of Australia.
- *Campylobacter* enteritis is usually a self-limiting condition and generally does not require antimicrobial therapy. Antibiotics may be indicated in severe or prolonged cases, in the third trimester of pregnancy, in infants, the immunocompromised and some elderly patients.
- Most infections acquired in Australia remain susceptible to macrolide and fluoroquinolone antibiotics, however quinolone resistance is frequently demonstrated in *Campylobacter* isolates acquired during travel.

KEY CONSIDERATIONS

1. *Campylobacter* infection not only impacts individual human health, it is also a societal issue regarding antimicrobial use and development of antimicrobial resistance (AMR).
2. Veterinary practitioners are well positioned to advise owners of the risks of pet-associated *Campylobacter* infections and the importance of routine hygiene measures when handling pets and their food.
3. Antimicrobial treatment of campylobacteriosis in dogs and cats is generally not required or recommended.

Most cases of campylobacteriosis in humans are food-borne (e.g. exposure to contaminated raw meat), however companion animals (and their food) may be a potential source of infection.

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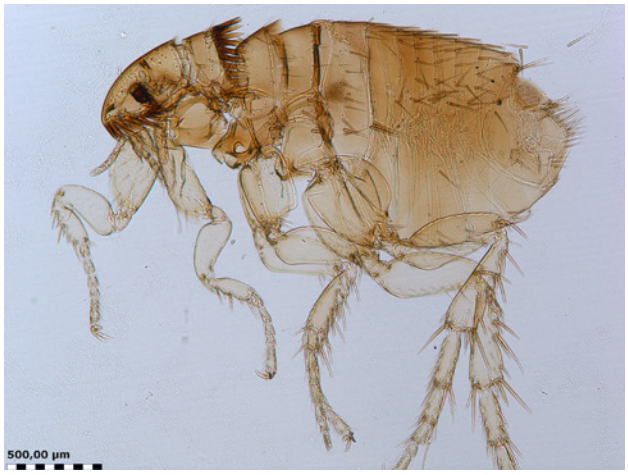
CAT-SCRATCH DISEASE *(Bartonella henselae)*

- *Bartonella* are gram-negative, vector-borne intracellular bacteria that infect a range of mammalian hosts. More than 30 different species of *Bartonella* have been identified, each adapted to a primary mammalian reservoir host species, in which the organism is maintained, typically without causing clinical disease.[1]
- The species most relevant to companion animal medicine are *B. clarridgeiae*, *B. elizabethae, B. henselae, B. koehlerae, B. quintana, B. rochalimae* and *B. vinsonii* subsp. *berkhoffii*, however the latter three species have not been identified in companion animals in Australia.
- *Bartonella henselae* is a feline-adapted species and is primarily associated with cat-scratch disease (CSD) in humans. *Bartonella henselae* bacteraemia has also been reported in a range of other species, including dogs. A second species, *B. clarridgeiae* is also commonly found in cats (estimated 10-30% of *Bartonella* infections) and may result in a cat-scratch like disease in humans.
- Dependent on the infecting species, *Bartonell*a infections in humans may be associated with a range of clinical manifestations, including asymptomatic infection, localised skin infection and lymphadenopathy (CSD), endocarditis, and neurological signs. More unusual and severe clinical manifestations may be seen in immunocompromised individuals.

ACAZAP RECOMMENDATIONS

- All cats and dogs should be administered effective flea control all year round. Environmental flea control may be required to deal with existing infestations.
- Guidance regarding safe animal handling for pet owners is an essential part of client education.
- Training and appropriate socialisation of pets is important to help avoid bites, scratches and licks.
- Avoiding bites and rough play with kittens is recommended, particularly for at risk groups. Open wounds should be covered to avoid potential contact with cat saliva and flea dirt.
- Good hygiene including washing hands thoroughly after handling pets and cleaning bite or scratch wounds immediately with soap and water is an important preventative measure. Scratches and open wounds should be covered with waterproof dressings.
- For individuals who are at greater risk of disease, including the immunocompromised, selecting an appropriate companion animal is essential. Young and/or flea-infested kittens from rescue facilities are more likely to be bacteraemic. Adopting adult cats (greater than a year of age) from flea-free environments is preferred.
- In a veterinary clinical setting care should be taken to avoid scratches and needle stick injuries. Appropriate handling and management of feline patients is essential to minimise the risk of bites or scratches that may transmit *Bartonella.*
- Routine testing of cats for *Bartonella* carriage is not indicated.

IN ANIMALS



The cat flea *(Ctenocephalides felis)* is the primary vector for *Bartonella henselae*

AETIOLOGY AND EPIDEMIOLOGY

- *Bartonella henselae* is a vector-borne pathogen, with the cat flea (*Ctenocephalides felis*) the primary vector. Cats are the primary reservoir species for *B. henselae,* however dogs may also be infected.
- The organism is transmitted between cats via flea faeces. After a blood meal from a *B. henselae*-infected cat, bacterial numbers rise within the flea's intestinal tract. Viable organisms are shed in flea faeces, with the bacteria remaining viable in flea faeces for at least 9 days.[1]
- Transmission occurs primarily through intradermal inoculation of *Bartonella*-containing flea faeces into skin wounds or bites. Such wounds may be self-inflicted in response to irritation caused by the vector. Less commonly, transmission may occur through direct inoculation onto the conjunctiva.
- It has been demonstrated that transmission does not occur between cohabitating cats in the absence of flea infestation.
- *Bartonella henselae* has been identified in ticks, but the contribution of this possible vector to the epidemiology of disease in cats is currently unknown.[2]
- Iatrogenic transmission through blood transfusion may occur.[3]

PREVALENCE AND RISK FACTORS

- Prevalence ranges widely dependent on the global location and study population. Prevalence is highest in warmer regions, owing to the more favourable conditions for the primary vector.
- In Australia, studies have demonstrated bacteraemia in 16-35% of cats based on blood culture or PCR.[4,5] In line with other research globally, infection is highest in young animals and those with fleas. Higher prevalence has been reported in feral compared to owned animals, likely associated with greater risk of ectoparasitism. Seroprevalence is higher in older cats, with bacteraemia greater in younger cats.
- Seroprevalence is typically reported at twice the rate of bacteraemia in the same population, however, as there is a poor association with bacteraemia, serology is not recommended for diagnosis.

CLINICAL DISEASE

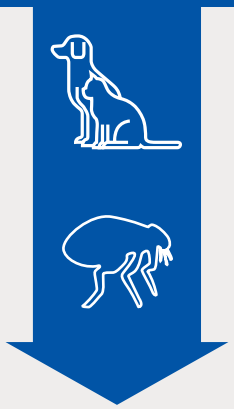
Infected cats are typically asymptomatic. A number of disease associations have been proposed for *B. henselae* in cats, including sporadic reports of myocardial, endocardial and ocular disease, however data are inconclusive.[6] Other *Bartonella* spp. (*B. clarridgeiae, B. quintana, B. koehlerae and B. bovis*) are less commonly isolated from cats than *B. henselae*, however challenges relating to isolation and identification make interpretation of the importance of these species challenging.

- Given that *Bartonell*a spp. can cause chronic intra-erythrocytic and endotheliotropic infections (a bacterial strategy for persistence of infection) in cats, dogs and other animal species, infections can potentially span weeks, months or years in duration.
- Chronic waxing-waning bacteraemia that persists for months or even years has been documented in young cats.[6] In a natural setting prolonged bacteraemia may be due to reinfection.
- Less commonly, *B. henselae* may infect dogs. Unlike cats, infection in dogs may result in disease, with endocarditis being the most commonly reported condition.

DIAGNOSIS

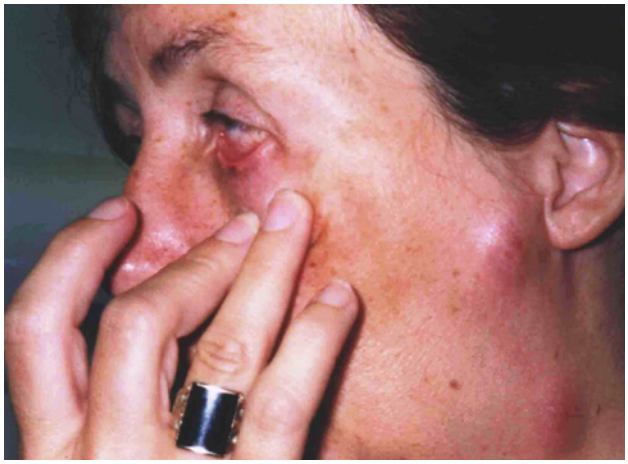
- Accurate diagnosis is challenging, as all of the available diagnostic tests have a low sensitivity, meaning a negative result cannot be trusted to rule-out infection. Culture or PCR of blood or tissues, and detection of antibodies in serum can all be used to aid diagnosis, however routine screening is generally not recommended. As *Bartonella* is relatively non-pathogenic in cats there are limited indications for feline testing.

TRANSMISSION

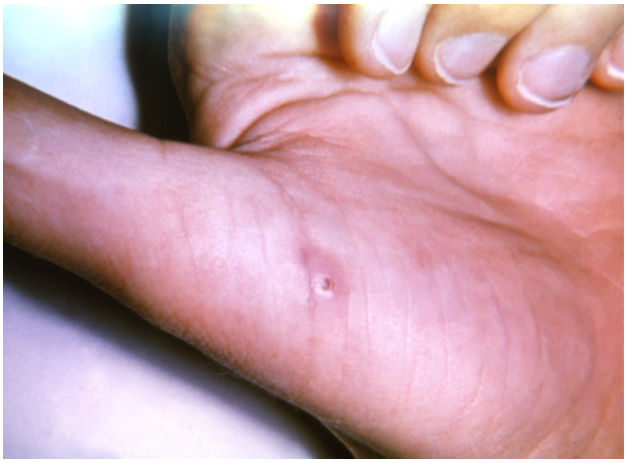


- Human exposure to infectious flea faeces is the typical route of infection, with the organism introduced subdermally through existing breaks of the skin or breaks created by scratches or bites from infected animals.
- *Bartonella* spp. may be present in the oral cavity and on the skin and claws of cats with active flea infestations.
- Most humans with *B. henselae*-associated clinical disease are believed to have been scratched.
- Conjunctival exposure is possible, and a single case of a veterinarian infected through needle stick puncture has been reported.[7]

IN HUMANS



Patient with Parinaud's oculoglandular syndrome (conjunctivitis and localised lymphadenopathy) caused by *B. henselae*



Small papule at the site of a cat-scratch in patient with cat-scratch disease
(Public Health Image Library, CDC)

AETIOLOGY AND EPIDEMIOLOGY

- Most human cases of *Bartonella* infection caused by *B. henselae* have a history of previous contact with cats, particularly kittens, and report being bitten, scratched and/or licked.
- *Bartonella henselae* infection is more commonly diagnosed in young children and teenagers in contact with young kittens and more frequently in children under ten years of age.[8,9] Veterinarians and veterinary practice staff are at increased risk of infection with *Bartonella* spp. by virtue of increased exposure over time to cats and fleas during physical examinations and procedures.

CLINICAL DISEASE

- *Bartonella henselae* can cause multiple clinical syndromes in humans, depending on the virulence of the strain, co-infection with other pathogens and an individual's immune status and co-morbidities.[10]
- Asymptomatic infections are common in humans.
- More severe disease and complications are seen in the immunocompromised (particularly associated with T-cell deficiency).[9]
- Classical cat-scratch disease is typically a self-limiting illness. The initial findings are a papule at the site of inoculation (3-10 days post infection), followed by solitary or regional lymphadenopathy 1 to 3 weeks later. The lymphadenopathy may persist for months. Fever, malaise, myalgia, arthralgia, and headache may be seen.
- Atypical presentations include:
  - Endocarditis (particularly in those with pre-existing valvular disease). *Bartonella* endocarditis and Q fever are the most common causes of culture-negative bacterial endocarditis in humans. As the diagnosis relies on serology, the diagnosis may be missed if serological testing is not requested.
  - Parinaud's oculoglandular syndrome – conjunctivitis and local lymphadenitis.

IN HUMANS *continued*

- Encephalitis may occur, without associated signs of classical cat-scratch disease.
- Neuroretinitis, presenting as painless vision loss.
- Splenic or hepatic granulomas.
- Osteomyelitis.
- Bacillary angiomatosis – vasculoproliferative tissue reaction that results in multiple nodular skin lesions. Most common in HIV patients with a low CD4+ count. Lesions may also involve internal organs.
- Peliosis hepatis is a rare condition characterised by vascular proliferation in the liver.

- Human-to-human transmission has not been documented.
- Diagnosis relies primarily on serology and on molecular testing (PCR) of tissue specimens. Unlike in bacteraemic cats, routine bacterial culture in humans is rarely positive.

KEY CONSIDERATIONS

1. Classical cat-scratch disease due to *Bartonella henselae* in humans is often associated with being bitten, scratched, or licked by cats and is more commonly diagnosed in young children and teenagers.
2. Training and appropriate socialisation of pets is important to help avoid bites, scratches and licks. This is particularly important for pet owners at greater risk of disease.
3. Any cat bite or scratch wound should be immediately cleaned with soap and running water. Open wounds should be covered prior to, and hands washed thoroughly after, handling pets.

Given exposure to infectious flea faeces is the typical route of human *B. henselae* infection, cats and dogs should be administered effective flea control all year round.

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CRYPTOSPORIDIOSIS *(Cryptosporidium spp.)*

- *Cryptosporidium* are apicomplexan gastrointestinal parasites that infect a range of animals. Numerous species are recognised, some with relatively restricted host ranges, while others are capable of infecting a broad range of hosts.
- *Cryptosporidium canis* and *C. felis* are host-adapted canine and feline species respectively, and are rarely found in humans or other animals. *Cryptosporidium hominis* is a human adapted species and is the most common cause of cryptosporidiosis in people.
- Most cases of cryptosporidiosis in humans are due to human-to-human transmission of *C. hominis*, and to a lesser extent *C. parvum* (typically livestock associated), however zoonotic infections with *C. canis* and *C. felis* have been reported, albeit rarely.

ACAZAP RECOMMENDATIONS

- Prevention of cryptosporidiosis is dependent on good hygiene. Alcohol-based hand sanitisers do not effectively inactivate *Cryptosporidium* oocysts, therefore thorough hand washing is essential.
- Prompt removal of animal faecal matter (at least daily) and thorough cleaning of litter trays and toileting areas is recommended.
- *Cryptosporidium* spp. are environmentally resilient and resistant to many common disinfectants when used at standard concentrations and contact times. High level disinfection (e.g. 50% ammonia, 3% hydrogen peroxide or 10% formalin) is necessary.[1] Steam and heat sterilisation may be required to inactivate oocysts.
- Infected animals should be isolated, particularly in group housed animal facilities. High density kennel situations should be avoided.
- Special recommendations for immunosuppressed individuals, children or those populations at risk:
  - Consider careful pet selection, choosing age-appropriate pets and avoiding adoption of young or stray animals.
  - Minimise exposure to potentially contaminated faeces, with other household occupants cleaning litter boxes/ disposing of faeces where possible. If required to dispose of faecal material, disposable gloves should be worn.

IN ANIMALS

AETIOLOGY AND EPIDEMIOLOGY

- Dogs and cats become infected through direct contact with infected hosts or indirectly through ingestion of oocysts from contaminated food, water, soil or through coprophagia.
- The primary species infecting dogs and cats are *C. canis* and *C. felis* respectively. *Cryptosporidium parvum* has also been detected in naturally infected dogs together with rare reports of *C. muris*.
- *Cryptosporidium* spp. complete their life cycle in a single host, alternating between asexual and sexual reproduction. After ingestion of sporulated oocysts, excystation occurs in the gastrointestinal tract followed by the release of sporozoites which infect the epithelial cells and undergo repeated merogony (asexual replication). The ability of *Cryptosporidium* to produce and release oocysts within the same host can lead to autoinfection. There are two types of oocyst: thin-walled and thick-walled. Thin-walled oocysts are responsible for autoinfection and thick-walled oocysts are shed into the environment.

PREVALENCE AND RISK FACTORS

- The pooled global prevalence of *Cryptosporidium* in dogs in a recent meta-analysis was 8%.[2] In Australia, prevalence of faecal oocyst shedding has been reported in several studies and ranges from 0.6% to 2%.[3,4] Dogs residing in rural areas and less than one year of age have a higher infection rate.
- Overall prevalence of *Cryptosporidium* in Australian cats has been reported as 2.2%, with infection rates higher in cats from shelters (3.5%) than owned pet cats (1.0%).[4] Prevalence was higher in cats under one year of age.

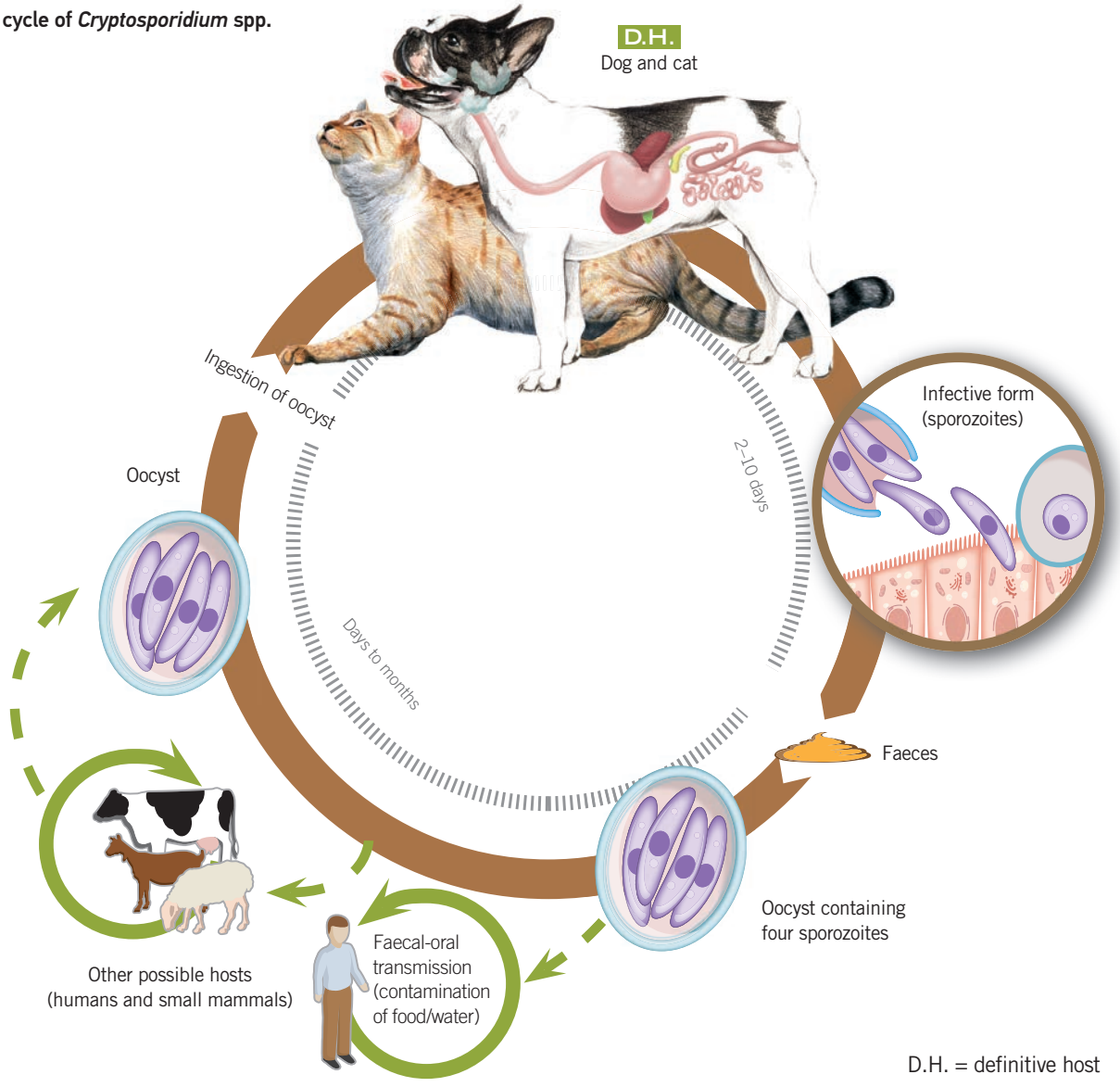
CLINICAL DISEASE

- A role for *Cryptosporidium* as a primary pathogen in dogs and cats is unclear, as clinical signs are rarely seen in healthy animals. Immunosuppression or concurrent gastrointestinal infection or parasitism may lead to clinical signs.
- If clinical signs are seen, animals typically present with self-limiting large-volume small-bowel diarrhoea, anorexia and weight loss. Systemic signs are rare.[5]
- Chronic, asymptomatic infection with relatively low intensity oocyst shedding can occur.

DIAGNOSIS

- Specialised stains such as the Ziehl-Neelsen or modified acid-fast staining of direct faecal smears are useful for visualisation of oocysts but lack sensitivity if oocyst shedding is low.
- Immunodiagnostic coproantigen ELISA assays and genus-based real-time PCR-based assays offered through commercial veterinary laboratories are the most reliable methods of diagnosis.

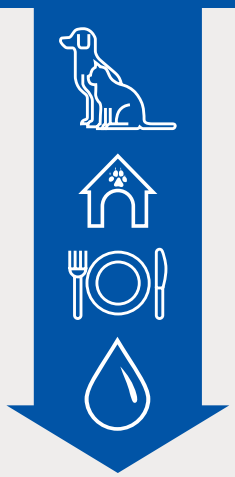
Life cycle of *Cryptosporidium* spp.



D.H. = definitive host

Life cycle from Beugnet, F., et al (2018) Textbook of Clinical Parasitology in Dogs and Cat. Grupo Asis Biomedica, S.L.; Adapted from Carithers, D., et al (2012) Pet Owner Educational Atlas.

TRANSMISSION

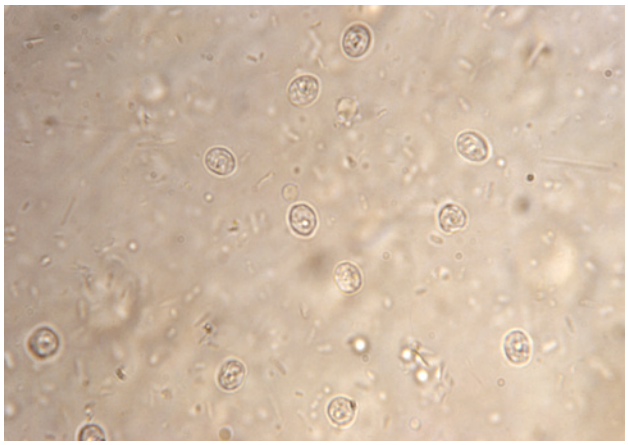


- *Cryptosporidium* is transmitted via the faecal-oral route. Zoonotic cryptosporidiosis may be transmitted through direct contact, and indirectly through ingestion of contaminated food or water. Aerosol transmission of oocysts has been reported.[6]
- *Cryptosporidium* oocysts are immediately infective after being passed in faeces.
- *Cryptosporidium* spp. can tolerate a range of environmental conditions, surviving in water and soil for months if the moisture and temperatures are suitable.[6,7]
- As few as 10 oocysts can cause disease in healthy individuals.[7,8]

IN HUMANS

PREVALENCE AND RISK FACTORS

- Cryptosporidiosis is an important waterborne protozoal disease globally. Australia has a higher rate of reported cryptosporidiosis than other similarly developed countries.[9]
- The majority of human cases in Australia (>85%) are caused by *C. hominis* (formerly known as *C. parvum* anthroponotic genotype) and to a lesser extent *C. parvum*.[10]
- The majority of cases of cryptosporidiosis in humans can be attributed to contaminated water – drinking from natural bodies of water or recreational water activities (e.g. camping, community swimming pools).[6,11] Occupational risk factors include working with young children (e.g. daycare centres) or animals (e.g. veterinarians, farmers).[6] In Australia, reports of cryptosporidiosis peak in summer, with an additional peak in spring in NSW and Queensland (thought to be associated with increased numbers of young livestock, in particular calves).[9]



***Cryptosporidium parvum* oocysts in a faecal sample from a human with cryptosporidiosis**
(Public Health Image Library, CDC)

Calves are the primary source of *C. parvum*. It is estimated that a single calf excretes approximately 6×10^{11} oocysts in the first month after birth.[12]

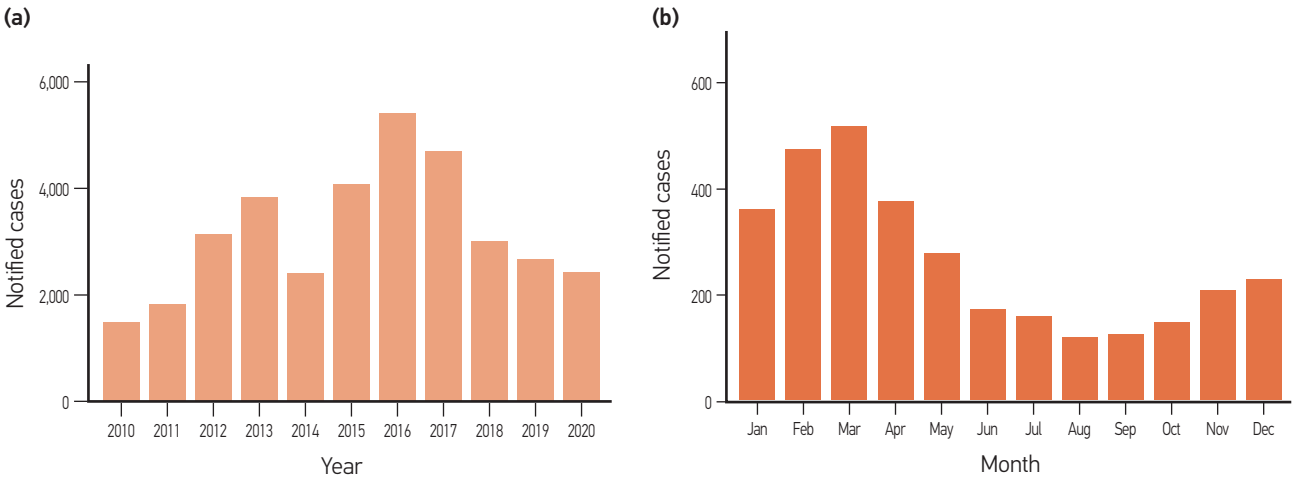
- The burden of cryptosporidiosis has been reported to be significantly higher in Aboriginal versus non-Aboriginal communities in Australia, with notification rates in Aboriginal people up to 50 times higher.[13] The proportion of infections with zoonotic *Cryptosporidium* species has been reported to be higher in non-Aboriginal individuals than Aboriginal individuals.[13]
- Human infections with both *C. canis* and *C. felis* have been reported in both immunocompetent and immunocompromised individuals, including children. A review of *Cryptosporidium* species isolated from more than 22,000 cases in 20 industrialised nations included only 59 cases (0.26%) of *C. felis* and 4 cases (0.02%) of *C. canis* infection.[14]

CLINICAL DISEASE

- Cryptosporidiosis can result in damage to the intestinal epithelium, disrupting absorption and barrier function and leading to mild to severe diarrhoea. A dose-dependent prepatent period of 3 to 12 days is reported in humans.[15]
- A significant proportion of cases are asymptomatic. Development of signs is related to infecting strain or species, host age (more common in young children, particularly under five years of age), immunocompromise or alterations in GIT microbiota.[15]
- Clinical signs include profuse watery diarrhoea, abdominal pain, vomiting and mild fever. Uncomplicated cases typically resolve within two weeks, however relapse is reported to occur in approximately a third of cases.[15] In children and infants, *Cryptosporidium* infections are sometimes associated with failure to thrive, stunted growth and malnutrition.[16]

IN HUMANS *continued*

Cryptosporidiosis in Australia: (a) cryptosporidiosis notifications by year in Australia from 2010 to 2020 and (b) average number of cases per month during this period. Data from National Notifiable Diseases Surveillance System, accessed April 2021.



- Chronic severe enteritis which is unresponsive to treatment may be seen in immunocompromised individuals. Patients can have chronic diarrhoea that lasts for greater than two months, with shedding of oocysts throughout this time. Specific conditions associated with chronic disease include advanced HIV infection, immunosuppressive chemotherapy affecting cell-mediated immunity (including corticosteroids), organ transplantation and primary T cell immunodeficiencies.[6,15]
- Extra-intestinal infection may be seen in immunocompromised patients, primarily from luminal extension to involve the biliary tree (resulting in biliary scarring) or pancreatic duct.[15] Disseminated infection is not common.
- Untreated cryptosporidiosis in pregnant women can result in severe dehydration and diarrhoea, with the potential to negatively impact the foetus.
- There is no specific treatment. Supportive therapy, including fluid and electrolyte replacement and antimotility drugs, may be indicated. In the immunocompromised patient, the most effective treatment approach is to aid recovery of the patient's immune status (e.g. anti-retroviral therapy) and/or reduction in immunosuppressive therapy (transplantation patients).
- Increasingly, human diagnostic laboratories are using combined *Giardia/Cryptosporidium* enzyme immunoassays and *Cryptosporidium* real-time PCRs for diagnosis. However, if microscopy is solely used for diagnosis, ensure that *Cryptosporidium* spp. is differentiated adequately from *Cyclospora cayetanensis*, as the latter responds to antimicrobials.

KEY CONSIDERATIONS

1. Most cases of cryptosporidiosis in humans are due to human host-adapted (*C. hominis*) or livestock adapted (*C. parvum*) species, however zoonotic infection with canine and feline adapted species (*C. canis* and *C. felis*) may rarely occur.
2. Cryptosporidiosis is more common in dogs and cats less than one year of age and is typically not associated with clinical signs.
3. Immunocompromised individuals at risk of significant disease associated with cryptosporidiosis should avoid adopting young or stray animals and minimise exposure to potentially contaminated faeces.

Prevention of cryptosporidiosis is dependent on good hygiene including hand washing (alcohol-based hand sanitisers do not effectively inactivate *Cryptosporidium* oocysts) and prompt removal and disposal of animal faecal matter.

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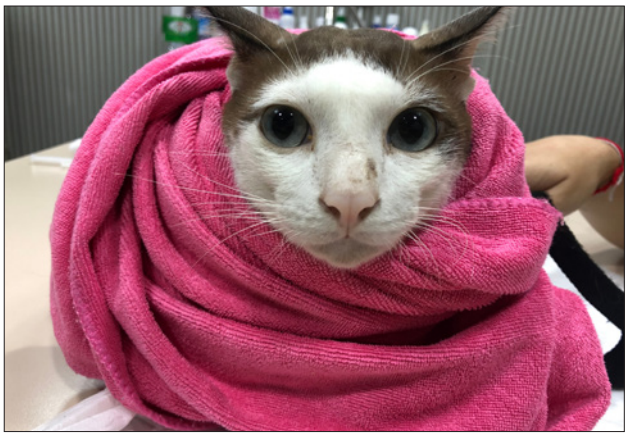
DOG AND CAT BITE WOUNDS

- Bite wounds from companion animals are common in Australia. Based on cases presented to hospital, the majority of animal bites in Australia are due to dogs (80%) and cats (7%).[1]
- In 1997 it was estimated that 2% of the Australian population were bitten annually by dogs, with 100,000 people requiring treatment.[2] More recently, a review of hospitalisation data from 2001 to 2013 reported an average of 2,061 individuals hospitalised each year in Australia for the treatment of dog bites.[3]
- The physical consequences of bite wounds result from tissue damage and local infection. Infection following a bite wound is typically polymicrobial and reflects the normal oral microflora of the biting species. Less commonly, infecting organisms may come from the patient's own skin or environment.
- In addition to physical trauma, psychological trauma (including fear, anxiety and post-traumatic stress disorder) has been reported and are likely under-appreciated in humans following animal bites.

ACAZAP RECOMMENDATIONS



Devices and tools to facilitate safe examination of patients should be used when appropriate

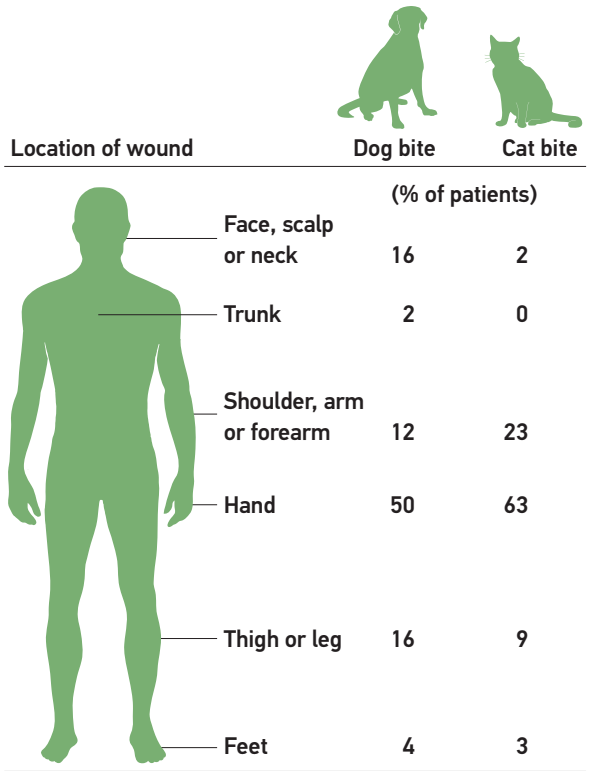
- Given bite wounds can be significantly contaminated, proper wound management is essential to reduce the risk of secondary infection. The affected skin surface should be thoroughly cleansed with soap and irrigated with water or normal saline.
- Prompt medical assessment of animal bites is recommended. Veterinarians should seek the advice of a health care professional for wound assessment and management and avoid self-treatment.
- Due to the significant risk of severe consequences of a post-bite infection in asplenic patients, any dog or cat bite or scratch in this population should be reviewed by a medical professional.
- While *Clostridium tetani* is an uncommon component of canine or feline oral microflora, contamination of wounds with environmental bacteria cannot be excluded. Although a rare sequela to dog or cat bites, it is recommended that bite victims are up-to-date with tetanus vaccination (as per the *Australian Immunisation Handbook*).
- Although terrestrial rabies is exotic to Australia, the potential for infection with rabies should be considered in travellers returning from rabies enzootic locations with a history of a dog or cat bite.
- Veterinary staff and animal health workers are at increased risk of dog and cat bites through occupational exposure.

ACAZAP RECOMMENDATIONS *continued*

- The following are recommended to minimise the risk of bite incidents for veterinary staff and attendant owners in the clinic setting:
  - The clinical records of animals who have previously displayed aggressive behaviour should be clearly flagged. Similarly, the cages of aggressive animals in hospital should be clearly marked.
  - Animals should be handled in a calm, stress-free manner to minimise excitement and agitation. For cats, refer to the *ISFM Feline-Friendly Handling Guidelines.*
  - Premedication of fearful or aggressive dogs and cats with an anxiolytic medication prior to a clinic visit may help facilitate a calmer visit. Trazadone (dogs and cats) and gabapentin (cats only) have been reported effective for this purpose.[4]
  - Be alert for behaviour changes that may indicate fear, agitation, or aggression.
  - Animals should be restrained only by trained staff members. Owners should not restrain animals under any circumstances.
  - Physical restraint and protection tools, such as muzzles, towels, blankets, cat-sacks etc. should be used where appropriate.
  - Chemical restraint (sedation or anaesthesia) can be used to enable examination of fractious or aggressive animals. Be aware that some behaviour modifying drugs may have a disinhibiting effect (e.g. benzodiazepines), resulting in a paradoxical increase in aggression.
- Veterinarians and general practitioners play a key role in the prevention of companion animal bites through education of pet owners on safe interaction with pets (particularly for children) and on the selection of appropriate pets for the household. The topic should be incorporated into puppy preschool/training.
- Children should be supervised at all times when around pets.
- Consideration should be given to the nature and breed of animal when acquiring a new pet.
- Puppies should be appropriately socialised to minimise the development of behavioural problems as adults that may predispose them to aggressive interactions with humans (e.g. fear associated biting). Similarly kittens should be taught appropriate play interactions with their owners using appropriate toys to redirect play aggression.
- Neutering of pets is recommended to reduce the risk of aggressive behaviour.

ANIMAL FACTORS RELATING TO DOG AND CAT BITE INCIDENTS IN HUMANS

- Identified animal risk factors for canine bite incidents in humans include intact reproductive status and male dogs.[5,6] Breed related data must be interpreted in the context of general breed prevalence. A study in South Australia reported German Shepherds, Pit Bull Terriers, Dobermans, Blue and Red Heelers and Rottweilers were over-represented compared to their prevalence as pets.[2] A more recent study also from South Australia reported dog bites in children were most frequently associated with the Bull Terrier group and Jack Russel Terriers.[7] Owned dogs are most frequently involved in dog bite incidents, with the dog commonly known to the victim.[5]
- Dog bites inflicted on any species, including humans, are typically associated with crushing, shearing, and tearing forces, and can result in significant tissue injury. Dog bite related infections are commonly polymicrobial. Common species identified in canine bites include *Pasteurella* spp. (particularly *P. canis*), *Streptococcus* spp., *Staphylococcus* spp. *(S. aureus, S. pseudintermedius),* and mixed anaerobes.[8]
- In contrast to dogs, cat bite wounds are typically puncture wounds associated with canine teeth, and are frequently located on the patient's extremities. Due to the nature of the wounds, cat bites are more likely to become infected than dog bites, with infection reported in 28-80% of cases.[9] Osteomyelitis of an extremity bone is a more common sequela of cat bites



Location of wound	Dog bite	Cat bite
	(% of patients)	
Face, scalp or neck	16	2
Trunk	2	0
Shoulder, arm or forearm	12	23
Hand	50	63
Thigh or leg	16	9
Feet	4	3

Anatomical location of dog and cat bite wounds. Adapted from Talan et al (1999).[9] Note that distribution of bites may vary with the age of the victim, with dog bites in children more commonly reported on the head and neck.

than dog bites due to their deep penetrating nature. Common species identified from cat bites include: *Pasteurella* spp. (particularly *P. multocida*), *Streptococcus* spp., *Staphylococcus* spp. *(S. epidermidis, S. warneri), Moraxella* spp., *Neisseria* spp., *Corynebacterium* spp., and mixed anaerobes.[8] *Bartonella henselae,* the causative agent of cat-scratch disease, may be found in the oral cavity of cats and be transmitted by biting.

- Although not frequently isolated from bite wounds (perhaps due to fastidious culture requirements) *Capnocytophaga canimorsus* is an important zoonotic pathogen that can cause severe and potentially fatal septicaemia in patients without a functional spleen. The bacterium is a normal inhabitant of the canine and feline oral cavity, with carriage rates of up to 74% in dogs and 57% in cats.[10]

HUMAN FACTORS RELATING TO DOG AND CAT BITE INCIDENTS IN HUMANS

- Reported human risk factors for dog and cat bites:[5,11,12]
  - Humans bitten by dogs are more likely to be male and living in a household with dogs. Overall, two-thirds of dog bite victims are bitten by their own dog, or a dog known to them. Young children are reported to be a greatest risk of dog bites, and the highest rate of serious injury from dog bites is in children under 5 years of age. It is reported that approximately half of all bite wounds in children involve the face and scalp.
  - Humans bitten by cats are more likely to be female, with two-thirds aged between 20 and 35.
- Dog and cat bites are a frequently reported occupational injury for veterinarians and veterinary nurses, and those working in other pet related occupations (e.g. grooming facilities, shelters, kennels). In one Australian study, 48% of veterinarians reported dog bites resulting in skin penetration and 67% reported a cat bite or scratch with skin penetration in the previous 12 months.[13] Male veterinarians were more likely to have experienced a dog or cat bite injury.[13]
- A study from the United Kingdom reported an occupational risk for dog bites for individuals involved in delivery services.[14]

CLINICAL DISEASE AND MANAGEMENT

- It is estimated that 10–20% of bite wounds become infected, with infection more common following a cat bite than a dog bite.[12] In addition to physical injury present at the site, clinical signs relating to infection may include cellulitis, abscess formation, or enlarged local lymph nodes.
- Particular risks for post-bite infection include:
  - Patient characteristics: immunosuppression (AIDS, cirrhosis, asplenism, cancer, neutropenia, diabetes, and treatment with corticosteroids or immunosuppressants).[12]
  - Wound characteristics: puncture wounds; tissue devitalisation from crush wounds; bone, joint or tendon involvement; location on the extremities; or delayed treatment.
  - Species characteristics: cat bite wounds have a higher risk of infection than dog bites. Cat bite injuries are often less overt (potentially resulting in delayed diagnosis) but can be more penetrating, resulting in septic arthritis and osteomyelitis.
- Infection with *Pasteurella* spp. is common following an animal bite, and is typically associated with a shorter latency period (time from bite to onset of signs of infection) and an abrupt onset of severe, localised pain compared with other bacterial infections (e.g. due to *Staphylococcus aureus*).[8] Presence of *Pasteurella* spp. on culture of human wounds should prompt further investigation into a history of an animal bite that may not have been elucidated on initial clinical history. In contrast to dog and cat bite wounds, *Pasteurella* spp. are not typically associated with human bite wounds. The bacterium *Eikenella corrodens* is commonly associated with infected human bite wounds, and its presence in an infected bite wound may indicate a human rather than animal source.
- *Capnocytophaga canimorsus* bacteraemia is a rare but significant sequela to dog, and to a lesser extent, cat bites. Risk factors for severe systemic disease include individuals without a functional spleen and those with a history of alcoholism, although cases have been reported in patients without identifiable risk factors.[10] *Capnocytophaga* cases present with septic shock, meningitis, peripheral gangrene, endocarditis or eye infections. A case fatality rate of 26% has been reported.[10]
- Psychological trauma, including fear, anxiety and post-traumatic stress disorder are potential sequelae of animal bite incidents, and should be managed appropriately.

KEY CONSIDERATIONS

1. Bite wounds from companion animals are common in Australia. In addition to physical trauma (tissue damage and infection), they may cause psychological trauma.
2. Veterinarians and general practitioners play a key role in prevention of companion animal bites through education of pet owners on safe interaction with pets (particularly for children).
3. The avoidance of human injury is paramount. In the clinic setting, if in doubt as to the temperament of a patient, chemical or physical restraint aids should be used.

Prompt medical assessment of animal bites is recommended and veterinarians should avoid self-treatment.

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FLEA-BORNE SPOTTED FEVER

(Rickettsia felis)

- *Rickettsia felis* is an emerging vector-borne bacterial pathogen and the causative agent of the human disease flea-borne spotted fever (FBSF).
- The primary vector of *R. felis* is the cat flea *(Ctenocephalides felis).*
- Dogs are considered the primary mammalian reservoir host for *R. felis,* however cats may also be infected. Infection of companion animals is not associated with clinical disease.

ACAZAP RECOMMENDATIONS

- Year-round flea control is recommended for all dogs and cats to reduce animal exposure to potentially infected fleas. Treatment of all dogs and cats in the household is recommended.
- Veterinarians play a key role in advocating flea control in domestic pets and educating pet owners, not only on the impact of fleas on the health and wellbeing of their pets, but also on the risk of flea-borne zoonotic disease.
- Exposure to flea-infested companion animals is a potential occupational hazard for Australian veterinarians; consider wearing long sleeve protective clothing when undertaking activities where increased contact with flea-infested animals is required.

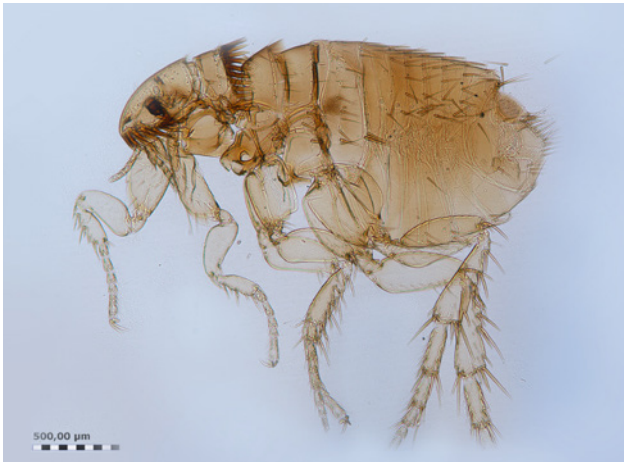
IN ANIMALS

AETIOLOGY AND EPIDEMIOLOGY

- Like many rickettsial species, *R. felis* can sustain infection in arthropod vectors by transovarial (parent flea to offspring) and/or transstadial (one life stage to the next) transmission.[1] The cat flea *(C. felis),* the only confirmed biological vector of *R. felis,* is capable of vertically transmitting *R. felis* for up to 12 generations.[2,3]
- The cat flea is ubiquitous in both tropical and temperate regions and is the primary flea infesting dogs and cats in Australia.[4]

PREVALENCE AND RISK FACTORS

- Australian data suggests environmental temperature impacts prevalence of *R. felis* in fleas, with the organism more prevalent in cooler, temperate climates. *Rickettsia felis* was detected in 6.7%, 13.2% and 15.5% of fleas sourced from tropical, subtropical and temperate regions of Australia's eastern seaboard respectively.[5]
- In Australia, studies have identified *R. felis* infection rates in cat fleas ranging from 19.8% in Brisbane, Sydney and Melbourne and up to 36% in regional centres in Western Australia.[6,7]



The cat flea *(Ctenocephalides felis)* is the only confirmed biological vector of *Rickettsia felis*

- Dogs are considered the primary mammalian reservoir.[2] Research in south-east Queensland identified *R. felis* DNA in the blood of 9% of healthy pound dogs, with another study in Indigenous community dogs in the Northern Territory reporting a prevalence of 2.3%.[8,9]

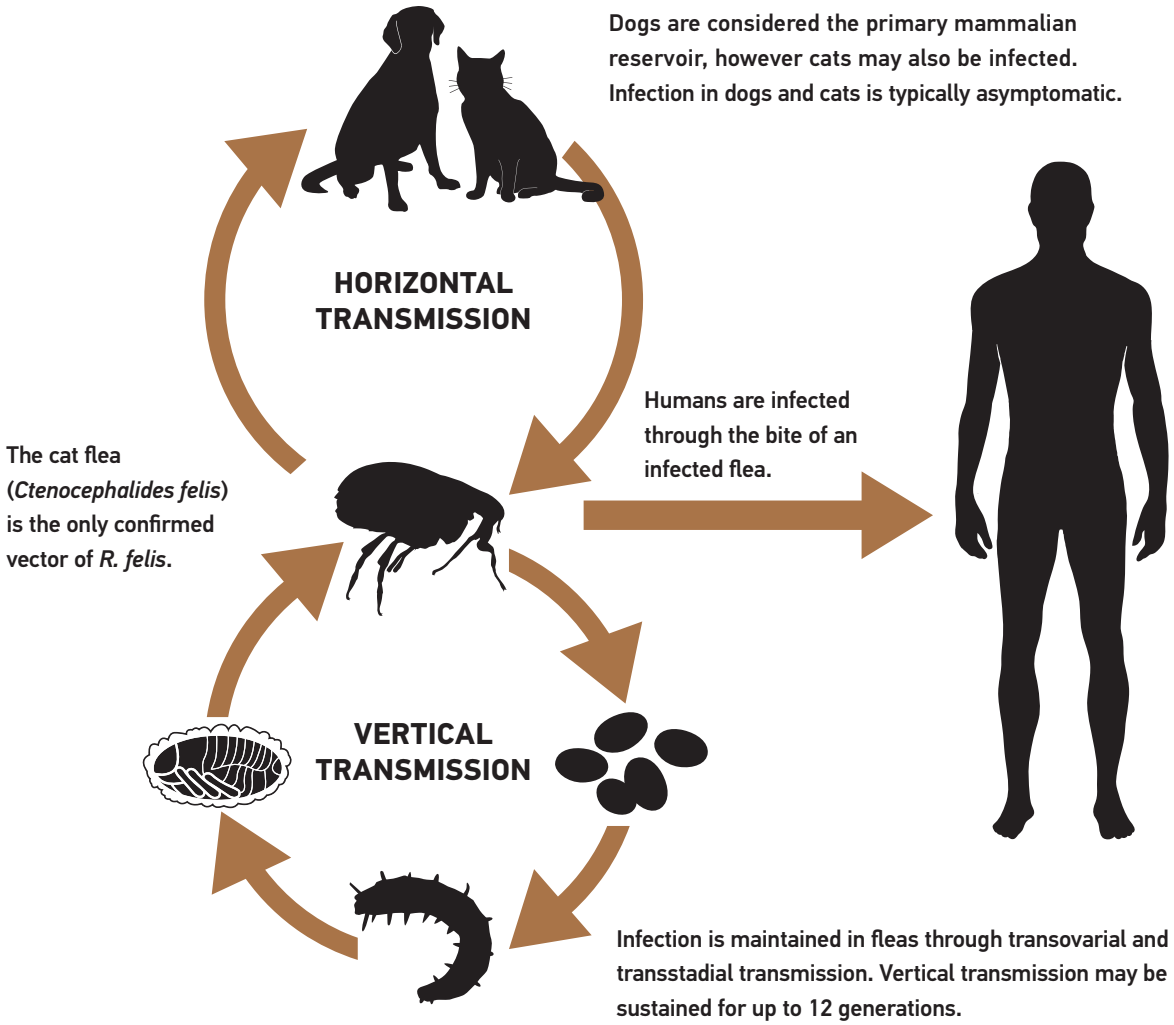
CLINICAL DISEASE

- Cats and dogs are susceptible to *R. felis* infection but are typically asymptomatic.
- Infected cats that seroconvert after exposure to infected fleas may have a short-term rickettsaemia but its clinical significance as a feline pathogen is currently unknown.
- Dogs may be bacteraemic but have normal haematological parameters and remain asymptomatic.[2]

DIAGNOSIS

- A test specifically for *R. felis* in companion animals is not commercially available, however PCR panels using pan-*Rickettsia* primers can be used in a research setting.

Life cycle of *Rickettsia felis*



TRANSMISSION

- The most common route of exposure to *Rickettsia felis* in humans is via flea saliva through the bite of an infected flea.
- A possible mechanism for indirect transmission is through cutaneous inoculation via contamination of broken skin or wounds with infectious vector faeces, as has been demonstrated with other rickettsial species.[10]

IN HUMANS

PREVALENCE AND RISK FACTORS

- Human infection with *R. felis,* known as flea-borne spotted fever (FBSF), is considered an emerging arthropod-borne zoonosis, however there is limited data on its epidemiology globally.
- Many cases are likely undiagnosed due to the non-specific nature of clinical signs (e.g. fever, lethargy). Severe disease is considered rare.
- Exposure to rickettsial species is significant in veterinary practice. A 2017 serological screening study of veterinarians in Australia demonstrated 16% of participants were seropositive to *R. felis* and 4.6% seropositive to *R. typhi*. A further 35.1% of tested veterinarians were seropositive for rickettsial exposure but unable to be differentiated to a particular species.[11]
- The first reported cases of human *R. felis* infection in Australia were documented in Victoria in 2009. Two adults and three children contracted a rickettsial disease, with all patients having extensive close contact with recently acquired *R. felis*-positive, flea-infested kittens.[12]
- A recent study has retrospectively identified fourteen probable Australian cases of *R. felis* infection in patients between August 2010 and December 2013, with the authors noting rickettsial disease is likely to have been misdiagnosed as murine typhus due to the previous unavailability of specific *R. felis* serological assays.[13]

CLINICAL DISEASE

- Clinical disease associated with *R. felis* infection is similar to other rickettsial infections. Symptoms may include pyrexia, myalgia and headaches. Cutaneous manifestations may include a maculopapular rash, and rarely a localised eschar at the flea bite site. Most human infection is self-limiting with mild to moderate illness observed.[3,14]
- Respiratory and gastrointestinal symptoms including cough, pulmonary oedema, pneumonia, nausea, vomiting and diarrhoea have been reported. Severe clinical manifestations such as neurological signs have also been documented.[14]
- Human infection can be diagnosed by PCR or serology. The presence of circulating IgG antibodies most likely indicates previous infection with *R. felis,* while rising paired acute and convalescent titres may indicate recent infection. Seroconversion may appear a month or more after rickettsial infection.[14]

KEY CONSIDERATIONS

1. *Rickettsia felis* is an emerging flea-borne bacterial pathogen, with data indicating it is more prevalent in cooler, temperate climates.
2. Infection of companion animals is common in Australia, however infection is not associated with disease in these species.
3. Veterinary professionals and animal care workers may be at increased risk of infection with *R. felis* due to more frequent opportunities for exposure to infected fleas.

Year-round flea control is recommended for all dogs and cats to minimise exposure to potentially infected fleas.

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GIARDIASIS *(Giardia* spp.)

- *Giardia* are gastrointestinal protozoan parasites that are found in most vertebrate species. The *Giardia duodenalis* 'species complex' comprises multiple assemblages (genetic groupings) A-H, with different host specificities. These assemblages have recently been 'reclassified' as separate species.[1]
- Humans and a range of animals, including dogs and cats, are susceptible to infection with *Giardia duodenalis* (Assemblage A) and *Giardia enterica* (Assemblage B) making these species potential zoonoses.
- Dogs (and other canids) and cats may also be infected with host-specific species, *Giardia canis* (Assemblages C and D) and *Giardia cati* (Assemblage F) respectively.
- Most cases of giardiasis in humans result from human-to-human transmission as opposed to being from a zoonotic source. Zoonotic assemblages A and B identified in wildlife and domestic animals could contribute to transmission between humans and animals.[2]

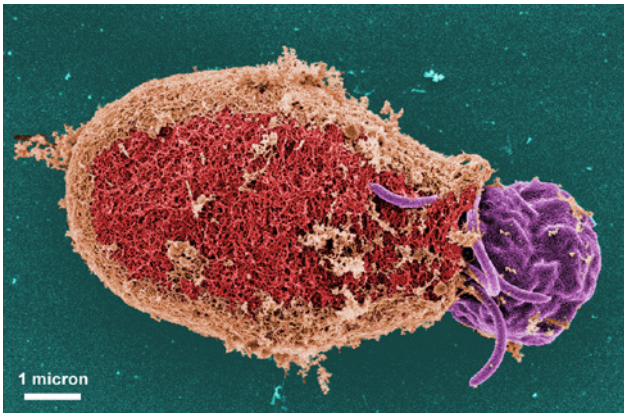
ACAZAP RECOMMENDATIONS

- Prompt removal of dog and cat faeces is recommended.
- Ensure good hand hygiene practices for all family members, especially children, after handling pets, playing outside and prior to eating.
- Cover sandpits and protect playgrounds and garden areas. Do not allow children to play in areas contaminated with animal faeces.
- Avoid drinking untreated water from rivers, lakes and other sites where the water supply is not considered safe.
- Although the risk is low, it is widely accepted that *Giardia* of animal origin may infect humans. The cysts of *Giardia* which infect dogs, cats and humans are morphologically identical. As such, all *Giardia* positive animals should be considered as carrying potentially zoonotic species and owners must be advised as such. Recommended precautions for managing animals with documented *Giardia* infection include:
  - Owners should wear gloves when disposing of animal faecal matter and reduce environmental contamination through cleaning and disinfection of in-contact surfaces where possible. Environmental areas (such as soil and grass) are challenging to decontaminate but all hard surfaces should be sanitised.
  - Infected animals should be quarantined and thoroughly washed prior to reintroduction to clean areas.
  - Pregnant animals should be tested and treated and then bathed prior to whelping with chlorhexidine shampoos to remove cysts on the coat.
  - In kennel or shelter environments management should include keeping cages clean and dry (inclusive of prompt removal of faecal matter) and disinfection of surfaces (cysts are sensitive to the majority of commercial disinfectants).
- Human-to-animal transmission is possible but thought to be uncommon. In the household setting of immunocompromised patients or relapsing human infection, treatment of household pets could be considered. A collaborative approach to case management between human healthcare professionals and veterinarians in these instances is recommended.

IN ANIMALS

AETIOLOGY AND EPIDEMIOLOGY

- Dogs are primarily infected with *Giardia canis* (Assemblages C and D) which are canine host adapted. Cats are primarily infected with *Giardia cati* (Assemblage F), a feline adapted species. These species are not considered zoonotic. In addition to their host adapted species, both dogs and cats are susceptible to infection with *G. duodenalis* and *G. enterica*, which have a wide host range, including humans.[1,3]
- *Giardia* has a simple two stage life cycle.[4] Trophozoites are the actively replicating stage that attach to the surface of the small intestine causing functional changes and damage to the intestinal villi. Trophozoites become encysted and are shed in faeces. Cysts are environmentally resistant and immediately infective after shedding.[4]
- Ingestion of cysts leading to giardiasis and subsequent clinical signs is dependent on host factors (e.g. co-infections, age, sex, genetic predisposition, immune competence) and also agent factors (e.g. assemblage, production of proteolytic enzymes).[4,5]
- The prepatent period ranges from 3-14 days in dogs and 5-16 days in cats.[6,7] Patency can persist for several weeks or months. Prolonged shedding is common and may be intermittent and inconsistent.

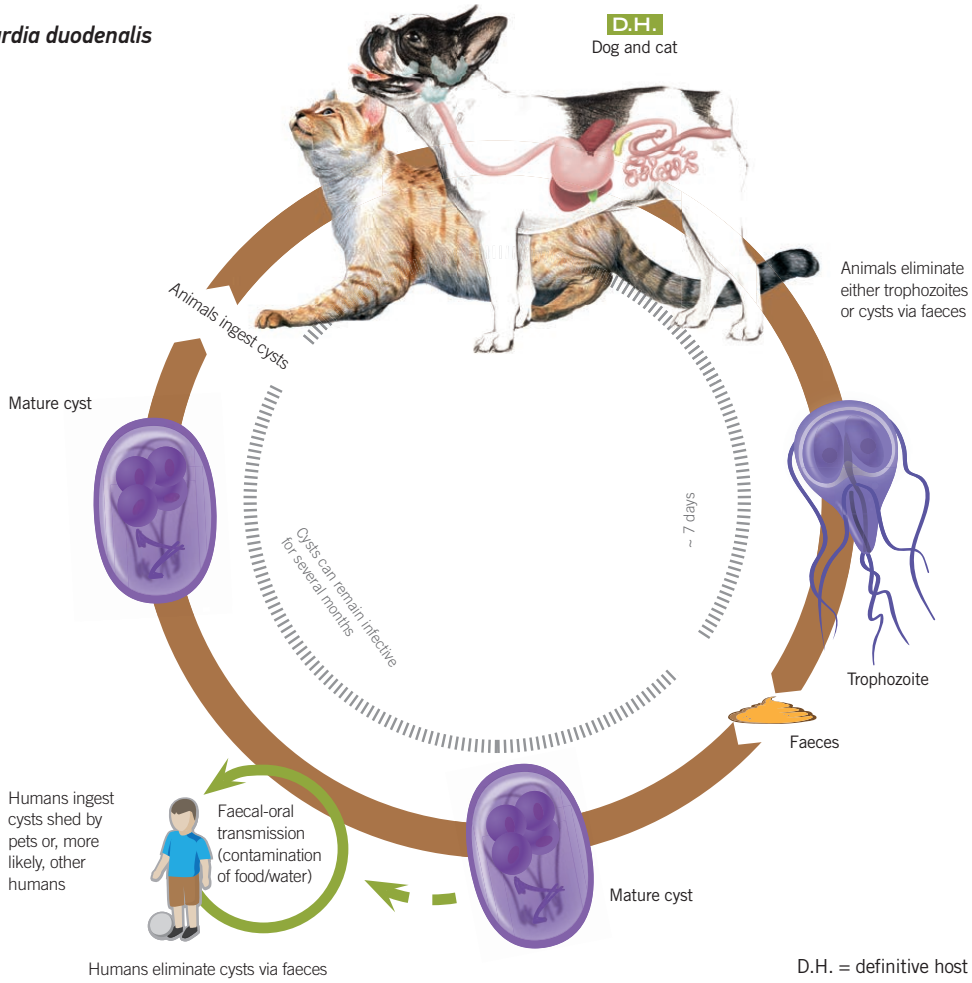


Scanning electron microscopic (SEM) image of *Giardia* spp. cyst undergoing "excystation", with a flagellated trophozoite emerging from the right side of the cyst
(Public Health Image Library, CDC)

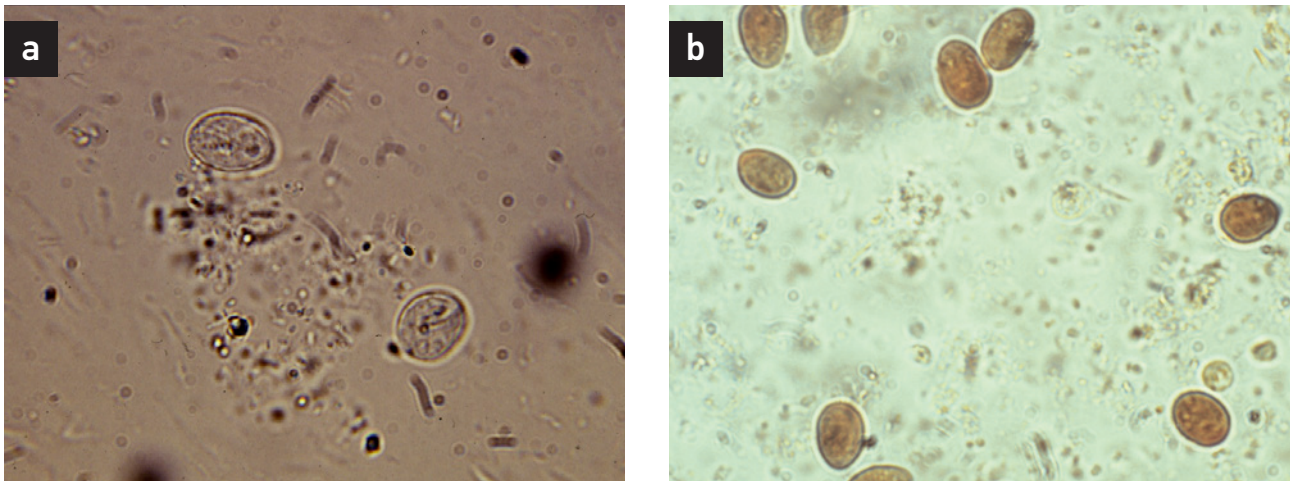
PREVALENCE AND RISK FACTORS

- Pooled global prevalence data for *Giardia* infection in dogs and cats from a recent meta-analysis was 15.2% and 12.0% respectively.[8] Studies conducted in Australia reported a prevalence of cyst shedding ranging from 3-50% in dogs[9-14] and 0-16% in cats.[9,11,13,14] High rates of faecal shedding

Life cycle of *Giardia duodenalis*



Life cycle from Beugnet, F., et al (2018) Textbook of Clinical Parasitology in Dogs and Cat. Grupo Asis Biomedica, S.L.; Adapted from Carithers, D., et al (2012) Pet Owner Educational Atlas.



Coproscopy can be used to diagnose *Giardia* infections: (a) unstained and (b) stained (Lugol's iodine) preparations

of *Giardia* by dogs in Indigenous communities have been found in many studies. The canine and feline adapted assemblages predominate in Australian studies, although the potentially zoonotic *G. duodenalis* (Assemblage A) has been identified in dogs.[15]

- Prevalence and cyst shedding intensities are higher in younger animals, those from high population density environments (shelters, kennels, catteries) and in those with compatible clinical signs (see below).[4,5]

CLINICAL DISEASE

- Infection is usually subclinical.[4,5] Clinical signs are more likely to occur in young or immunosuppressed animals, or those with concurrent gastrointestinal pathogens or parasites. Chronic mucoid diarrhoea and weight loss are the most common findings. Systemic signs of illness are uncommon.

DIAGNOSIS

- Several diagnostic assays are available for *Giardia*, including coproscopic identification of cysts using zinc sulfate centrifugal flotation, point-of-care coproantigen tests, and molecular diagnosis using real-time PCR. In a comparative study, real-time PCR has been demonstrated to have the highest sensitivity (97.0%), followed by coproantigen detection (71.9%), with standard coproscopy having the lowest sensitivity (48.2%).[16]
- Determination of assemblage/species, and therefore zoonotic potential, is not routinely performed by diagnostic laboratories. In the absence of this information all animals testing positive should be considered as carrying potentially zoonotic species and appropriate precautions taken.
- It is not recommended to treat clinically well animals that test positive for *Giardia*.

TRANSMISSION

- *Giardia* is transmitted via the faecal-oral route through the ingestion of cysts, either directly through close contact, or through contaminated food and water.
- Contaminated or untreated water is a risk for infection, such as at camping sites, rivers and lakes.
- Cysts can survive in the environment for prolonged periods under moist, cool conditions, but are susceptible to desiccation in hotter, dryer environments.
- Giardiasis has a low infectious dose with ingestion of as little as ten cysts capable of causing infection.[17]

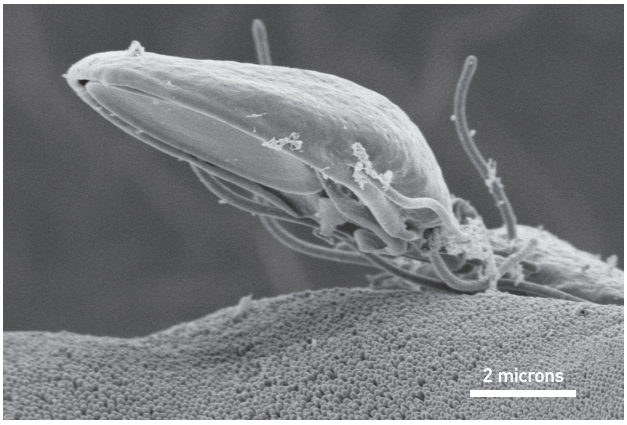
PREVALENCE AND RISK FACTORS

• Waterborne transmission (recreational exposure or drinking water) may account for up to 75% of human cases, followed by foodborne transmission.[18] Direct human-to-human faecal-oral exposure may also occur and explains the comparatively higher probability of *Giardia* in young children and in adults that work with or care for them (e.g. in childcare).[19,20]

• There is limited published data on zoonotic infection from dogs and cats, but the risk is considered to be low. Studies have demonstrated an association between owning a dog and Assemblage A giardiasis, and dogs have been shown to harbour zoonotic genotypes, including Assemblage A.[15,21-23]

CLINICAL DISEASE

• A median prepatent period of 2 weeks is reported in humans.[24] In *Giardia*-endemic communities, the majority of human infections are asymptomatic and do not require treatment. Clinical cases may present with bloating, flatulence and acute, intermittent or chronic diarrhoea. Infections are typically self-limiting, however prolonged infection may occur in both immunocompetent and immunocompromised individuals. Sequelae of chronic infection include malabsorption, weight loss and, in children, failure to thrive. Some studies have noted possible associations between chronic infection and irritable bowel syndrome, food allergies, arthritis and chronic fatigue syndrome.[25]

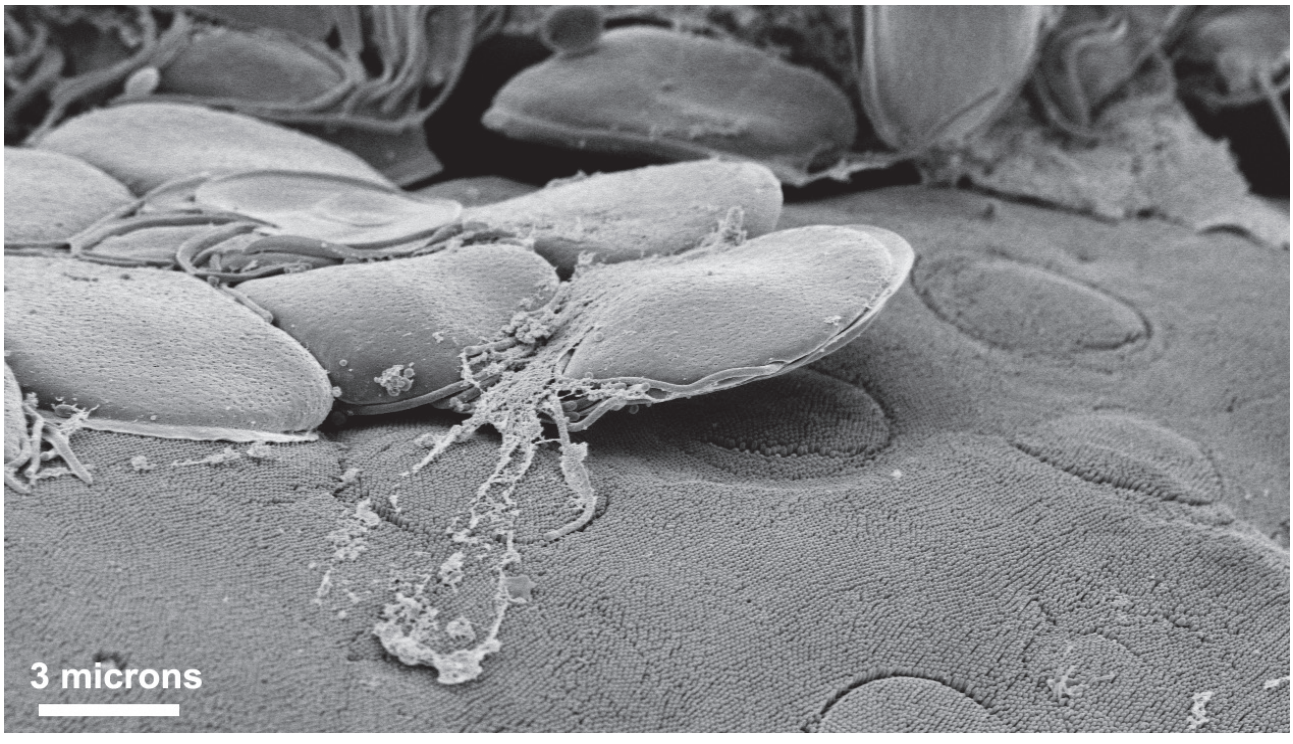


Scanning electron microscopic (SEM) image of *Giardia* spp. protozoan on the microvillous border composed of intestinal epithelial cells. The ventral adhesive disk, which facilitates adherence to the intestinal surface, can be seen on the underside of the organism
(Public Health Image Library, CDC)

• Temporary disaccharide intolerance can occur post infection and may last several weeks after clearance of the organism.

• Immunocompromised individuals, including those with congenital disease, hypogammaglobulinaemia, secretory IgA deficiencies and human immunodeficiency virus infection, have difficulty clearing intestinal *Giardia* infections.[26]

• Progressive immunocompromise and low CD4+ counts also increases the risk of symptomatic *Giardia* infection.[27]



Scanning electron microscopic (SEM) image showing group of *Giardia* spp. trophozoites clustered on the intestinal mucosal surface. Immediately adjacent to the organisms are a number of the characteristic circulars lesions that can be left on the surface as a result of the tight adhesion of the organism's ventral adhesive disk (Public Health Image Library, CDC)

IN HUMANS *continued*

- Faecal coproantigen assays and faecal real-time PCR for *Giardia* spp. are increasingly supplanting wet preparation and light microscopy for the diagnosis of *Giardia* infection.
- Human-to-animal transmission of *Giardia* is thought to be uncommon, however in the household setting of immunocompromised patients or relapsing human infection, treatment of household pets could be considered. A collaborative approach to case management between human healthcare professionals and veterinarians in these instances is recommended.

KEY CONSIDERATIONS

1. Dogs and cats are frequently infected with host-specific *Giardia* species, however they may also be infected with potentially zoonotic species.
2. Most human cases are thought to be acquired from direct or indirect human-to-human transmission.
3. Human-to-animal transmission is possible and should be considered in cases of relapsing human infections in households containing pet dogs or cats.

Although thought to be relatively low risk, *Giardia* positive dogs and cats should be considered as carrying potentially zoonotic species and owners must be advised as such. Appropriate precautions should be taken with such animals.

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HOOKWORM *(Ancylostoma* spp.*, Uncinaria stenocephala)*

- Hookworms are haematophagous nematodes that parasitise the gastrointestinal tract of a range of mammalian species, including domestic and wild animals, and humans.
- Hookworms are a common cause of morbidity (iron deficiency anaemia and malnutrition) in low- and middle-income countries, affecting an estimated 500 million people globally.[1] Human host-adapted hookworms *(Necator americanus* and *Ancylostoma duodenale)* are not zoonotic, however a third common hookworm species in humans, *Ancylostoma ceylanicum,* is a major zoonosis and highly endemic in dogs and cats across the Asia Pacific.
- Other hookworm species infesting dogs and cats are also known to cause zoonotic disease, most notably eosinophilic enteritis and cutaneous larva migrans.

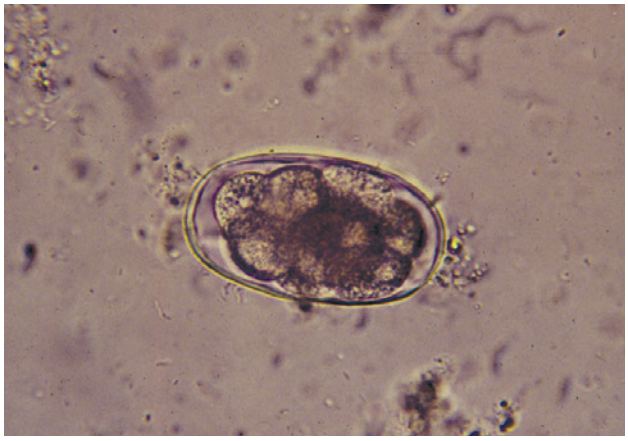
ACAZAP RECOMMENDATIONS

- Observing good hygiene measures and avoiding skin contact with contaminated soil and sand is recommended.
- Given hookworm-associated zoonotic infections are more frequently diagnosed in tropical and subtropical areas, adults and children in these locations should avoid walking barefoot to avoid larval penetration where dogs and cats are known to roam.
- Individuals with occupations that require consistent contact with moist soil for extended periods of time should consider wearing shoes and gloves.
- Children's sand pits should be covered when not in use.
- Prompt daily removal of cat and dog faecal matter from backyards will help prevent hookworm larval contamination of the environment.
- Puppies and kittens should be dewormed fortnightly from two weeks of age to eight weeks of age (two weeks after weaning). This is particularly important for puppies due to the risk of vertical and lactogenic transmission of *A. caninum.*
- Given the prepatent period of hookworm species may be as short as two weeks, at least monthly deworming of dogs and cats older than eight weeks is recommended to reduce environmental contamination and minimise zoonotic risk.
- Depending on health and lifestyle factors, adult dogs should have a faecal flotation performed yearly, with puppies tested more frequently.
- Do not feed raw meat or allow dogs to hunt, as many animals and birds act as paratenic hosts for some hookworm species.
- Veterinarians and public health workers should educate dog owners regarding the potential risks of improper parasite control in dogs.

IN ANIMALS

AETIOLOGY AND EPIDEMIOLOGY

- Five hookworm species have been identified in dogs and cats in Australia: *Ancylostoma caninum, A. tubaeforme* (cats only), *A. braziliense, A. ceylanicum* and *Uncinaria stenocephala*.[2]
- Hookworms are found throughout Australia, however there are geographical differences in the species distribution related to the climatic conditions required for larval development in the environment.[2]
  - *Ancylostoma caninum* is by far the most widely distributed owing to the ability of larvae to undergo arrested development during seasons unfavourable for its survival.
  - The other species of *Ancylostoma* are mainly found in warmer tropical and subtropical regions, with *A. braziliense* restricted to the wet tropics.
  - *Uncinaria stenocephala* is more adapted to temperate and cold regions and found in the southern parts of Australia.

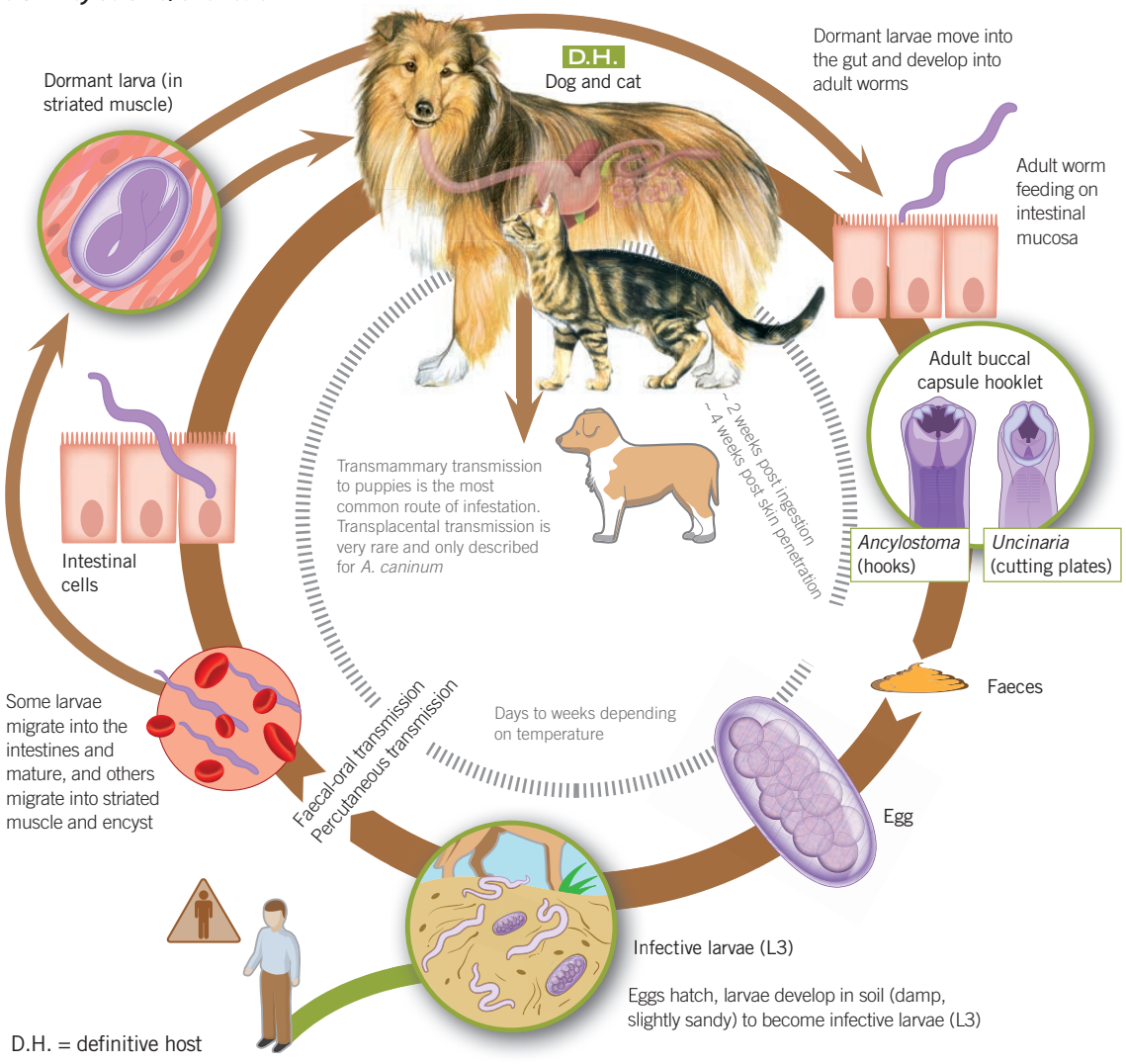


***Ancylostoma* spp. egg**

Buccal capsule of *Ancylostoma caninum* showing three pairs of teeth

- Dogs and cats become infested through ingestion of larvae in contaminated environments, through larval penetration of the skin or buccal mucosa, or through the ingestion of paratenic hosts (except for *A. ceylanicum*). For *A. caninum*, and to a lesser extent *U. stenocephala*, transmammary transmission can occur.
- Transmammary transmission of larvae from bitch to pup is an important route of infestation for *A. caninum*. In female dogs, larvae can undergo somatic migration and become disseminated in various tissues and organs. Larvae become hypobiotic and remain quiescent for months or years and can reactivate during parturition and lactation.

Life cycle of *Ancylostoma/Uncinaria*



Life cycle from Beugnet, F., et al (2018) Textbook of Clinical Parasitology in Dogs and Cat. Grupo Asis Biomedica, S.L.; Adapted from Carithers, D., et al (2012) Pet Owner Educational Atlas.

- Adult hookworms live in the small intestine. Eggs are excreted in the faeces where they embryonate and after two moults develop into infective third stage larvae. Larvae entering through the skin undergo pulmonary migration, are swallowed, and develop to adults in the gastrointestinal tract. Infections acquired orally undergo direct development to adults in the gastrointestinal tract.
- The prepatent period depends on the route of infection and species of hookworm, varying from two to four weeks.

PREVALENCE AND RISK FACTORS

- In an Australia-wide prevalence study, hookworm was identified in 6.7% of dogs and 1.4% of cats, with a significantly higher prevalence in the Northern Territory and Queensland.[3] A 2017 study confirmed 25% of domestic dogs in central Queensland to be infected with hookworms.[4]
- In Indigenous communities of the wet tropics of northern Queensland, *A. caninum* is the most common hookworm of domestic dogs and wild dingoes, with a prevalence of 92% and 100% respectively.[5] *Ancylostoma ceylanicum* is the second most prevalence hookworm species in this area, reported in 22% of domestic dogs and 11% of dingoes.[5,6]
- More recently, a 2020 study of dogs living in remote communities of the Northern Territory detected a prevalence of *A. caninum* of 31%.[7]
- Hookworms affect both dogs and cats, with stray animals, dogs in animal shelters and hunting animals more commonly infested. Puppies and younger dogs are more susceptible to heavy worm burdens owing to lowered age- and exposure-related immunity.

CLINICAL DISEASE

- The clinical features of infestation in puppies (as early as ten days old for *A. caninum*) may include acute haemorrhagic diarrhoea (melena or haematochezia), pallor, hypoproteinaemia and death.[2] Other signs include general loss of condition with an ongoing parasite burden (e.g. failure to thrive, anaemia), abdominal distension, chronic diarrhoea and more rarely, skin or respiratory conditions.[2]
- In adult dogs, acute infections may also result in haemorrhagic enteritis, however age and exposure-related immunity to trickle infections, which is not absolute, usually results in a chronic subclinical non-regenerative iron deficiency anaemia.[2]
- Clinical signs in cats are similar to those in dogs, but generally less severe as cats typically have lower hookworm burdens and *A. tubaeforme* is a less voracious blood feeder than hookworm species infesting dogs.

DIAGNOSIS

- Ancylostomiasis should be considered as a differential diagnosis in dogs and cats with intestinal disorders and weight loss. A definitive diagnosis can only be made by the detection of strongyle eggs via standard faecal flotation.
- In acute disease, especially in puppies, faecal antigen testing for intestinal parasites in combination with faecal flotation has been shown to be of benefit, given immature worms may still cause clinical signs prior to eggs being shed in the faeces.
- Alternatively, a presumptive diagnosis can be made if a rapid resolution of clinical signs is observed in response to treatment with an efficacious anthelmintic, accompanied by supportive care.

TRANSMISSION

- Infection in humans is either via direct skin penetration by infective larvae (typically by walking or lying barefoot on sandy beaches, contaminated soil or sandy areas) or via ingestion of larvae on contaminated surfaces and food.
- Shed eggs are not immediately infectious. The hatched non-infective larvae develop in faeces or soil to the infective third stage in 5-10 days.[8]
- Hookworm larvae can survive and remain infective for several months in warm and humid environments if protected from direct sunlight and desiccation.[9]

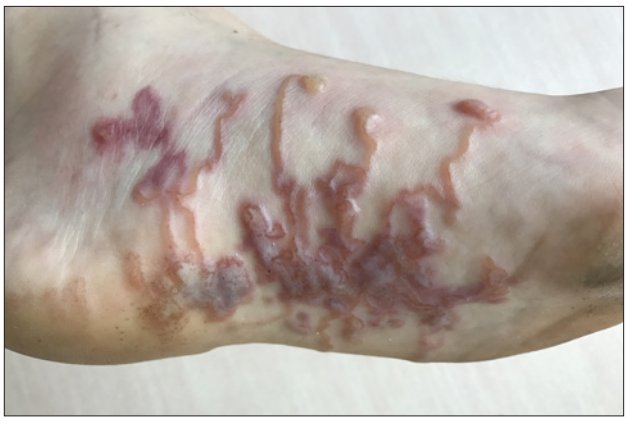
IN HUMANS

PREVALENCE AND RISK FACTORS

- Hookworms are the most common cause of cutaneous larva migrans (CLM) in humans, characterised by percutaneous penetration and migration of hookworm larvae in the skin.
- There is no human prevalence data in Australia for zoonotic hookworm infections, however reports typically emanate from wet tropical areas.
- There is no difference in probability of infection between veterinary staff, the general public or children that play in parks. A key factor in transmission is contact with moist sand and soil, particularly beach environments.

CLINICAL DISEASE

- All animal hookworms are zoonotic and capable of producing CLM. "Ground itch", consisting of a self-limiting pruritic papular rash, is the most common presentation at the site of percutaneous penetration of larvae.
- "Creeping eruptions", the typical highly pruritic and chronic migrating linear serpiginous lesions, are produced by *A. braziliense* and usually require medical intervention.[10] In Australia this is usually considered a travel-associated disease, with most cases diagnosed in travellers returning from the wet tropics where *A. braziliense* is endemic.[10,11]
- Whilst most hookworm species cannot complete their life cycle in people, *A. ceylanicum* can produce patent infections in humans and may result in diarrhoea (sometimes haemorrhagic), severe abdominal pain, fever, peripheral eosinophilia and anaemia.[12] Locally acquired cases of *A. ceylanicum* have been reported in people with gastrointestinal disturbances in Western Australia.[13] *Ancylostoma ceylanicum* is considered an emerging public health risk in northern tropical Australia, being found in areas frequented by tourists.[6]
- The global disease burden of hookworm is high, with an estimated 500 million people affected.[1] Recent studies in Australia's pacific neighbour, the Solomon Islands, demonstrated zoonotic ancylostomiasis caused by patent *A. ceylanicum* to be as high as 18.2%, while molecular-based surveys in Asia report between 6% and 23% of total patent hookworm infections are due to *A. ceylanicum*.[14,15]
- Hookworm infection is also a known cause of maternal anaemia in humans. Intestinal hookworm infection can result in iron deficiency anaemia and malnutrition in pregnant women and children. During pregnancy, hookworm infection has been associated with low birthweight and poor neonatal outcomes, including negative consequences for the cognitive and motor development of infants.[16,17] The public health impact of zoonotic infection with *A. ceylanicum* on a population scale are largely unexplored.
- *A. caninum* is a well-recognised agent of eosinophilic enteritis and aphthous ileitis in tropical Australia. Although most infections are asymptomatic, a single immature adult worm residing in the small intestine is capable of eliciting abdominal pain, intestinal bleeding, diarrhoea and weight loss.[18]
- More recently, rare patent infections of *A. caninum* have also been reported in humans.[19,20]



Cutaneous larva migrans caused by migration of larvae through human skin

KEY CONSIDERATIONS

1. Observing good hygiene measures and avoiding skin contact with contaminated soil and sand is recommended.
2. Individuals with occupations that require prolonged contact with moist soil should wear protective clothing, including shoes and gloves.
3. Veterinarians and public health workers should educate dog owners regarding the potential risks of improper parasite control in dogs.

At least monthly deworming of dogs and cats is recommended to reduce environmental contamination and minimise zoonotic risk.

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HYDATID DISEASE *(Echinococcus granulosus)*

- *Echinococcus granulosus sensu stricto (s.s.)* is a tapeworm endemic to parts of Australia.
- Echinococcosis is asymptomatic in the primary host (domestic and wild canids), however infestation of intermediate hosts can result in disease.
- Infection of humans results in hydatidosis or unilocular (cystic) echinococcosis, a cyst forming disease that can present as a clinically significant zoonosis.

ACAZAP RECOMMENDATIONS

- Feeding dogs cooked meat or commercial dog food is recommended. Provision of raw meats or meat by-products increases the risk of hydatid tapeworm infestation.
- Avoid feeding raw meat and offal to dogs, especially liver, lungs and other organs from on-farm slaughtering processes.
- Dogs, particularly in endemic or rural areas, should be supervised or restrained to prevent scavenging on dead livestock and wild animals (including macropods and feral pigs).
- If dogs have known exposure to *E. granulosus* or access to offal, deworming every six weeks with praziquantel is required.
- Praziquantel is not ovicidal, therefore tapeworm eggs within proglottids passed in the faeces are infective and could contaminate the environment following deworming. Faeces from 'at risk' dogs should be safely disposed of by deep burial or burning for 24 hours following deworming with praziquantel.
- Practice good hand hygiene following contact with dogs, playing outdoors, and prior to eating.

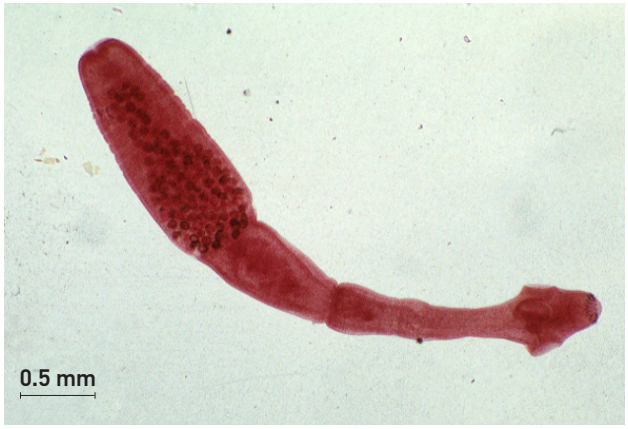
IN ANIMALS

AETIOLOGY AND EPIDEMIOLOGY

- The life cycle of *E. granulosus* is indirect involving predator-prey transmission between the definitive canid host and intermediate (mammalian) hosts. Dogs and other canids are infested after ingesting viscera, offal or meat containing fertile hydatids from an intermediate host.
- Domestic dogs, wild dogs, dingoes (and their hybrids), and less commonly foxes, are the known definitive hosts in Australia.[1]
- Cats are not definitive hosts for *E. granulosus* but may serve as an accidental intermediate host.[2,3]
- Intermediate hosts are herbivorous or omnivorous mammals. *Echinococcus granulosus s.s.* is widespread in mainland Australian native wildlife including macropods (e.g. eastern grey kangaroos, red-necked wallabies and swamp wallabies) and wombats, as well as domestic and feral introduced species (including sheep, cattle, goats and pigs).
- The larval (metacestode) stages commonly develop in the liver, lungs and various organs of the intermediate host. Clinical signs associated with infestation of livestock are rare, however production losses may be seen. Lung cysts leading to pulmonary dysfunction and fatalities in macropods have been reported.[4]

Scavenging on macropod carcasses is a risk factor for echinococcosis in dogs

- Two major transmission patterns are noted to occur: the sylvatic (or wildlife) pattern involving wild dogs and dingoes via predation of macropods, and the domestic pattern involving farm dogs and sheep.[5] Cattle and feral pigs are mostly infected with sterile cysts.[6] Sheep and certain species of macropods are the primary intermediate hosts in Australia, being the main source of fertile cysts.[5,6]
- When mature fertile cysts are ingested by canids, the cyst wall is digested away, freeing the protoscolices, which attach and develop into adult tapeworms in the dog's small intestine.



Echinococcus granulosus (note the small size)

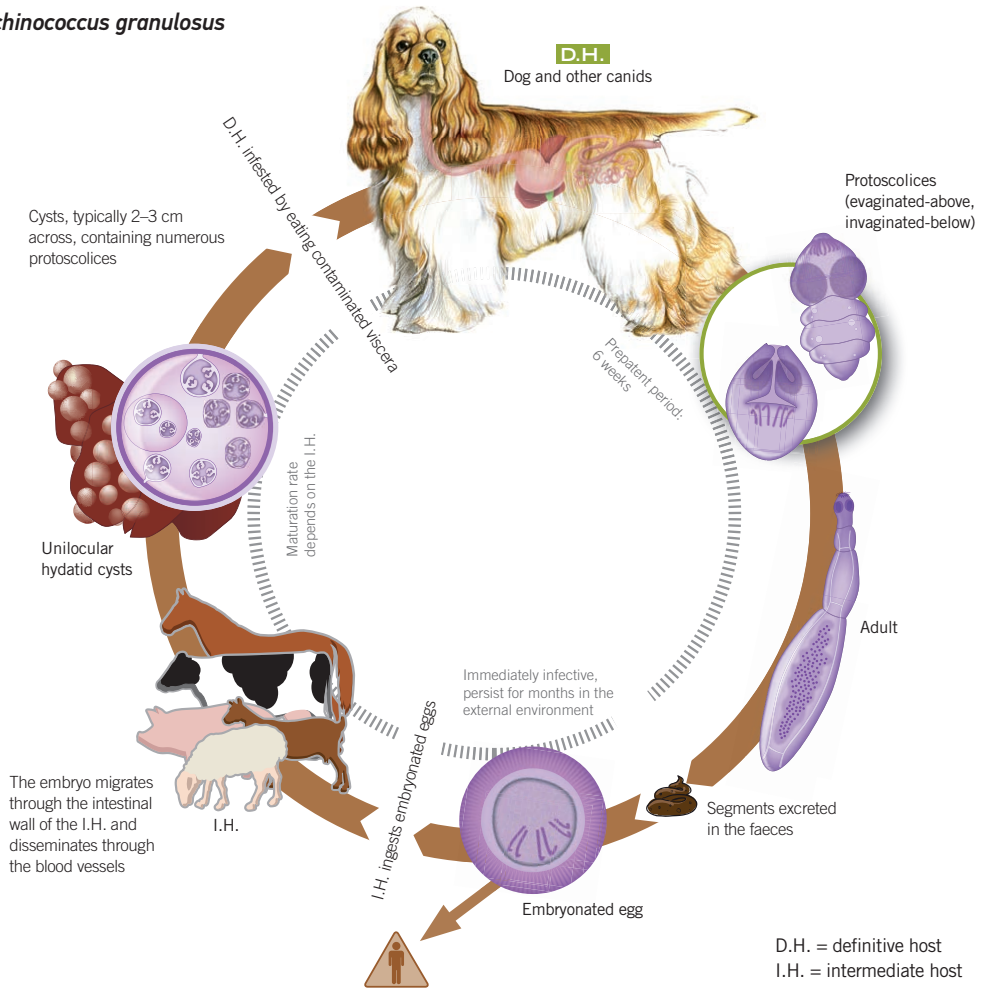
- Eggs, contained in proglottids, are shed in the faeces of the definitive host following a prepatent period of approximately 6-7 weeks and are immediately infective to intermediate hosts.[8]
- Susceptible intermediate hosts are infested through ingestion of eggs on pasture and in water. Following ingestion, the eggs hatch to release the oncosphere, which uses its hooks to burrow through the intestinal wall. They then enter the circulation and are transported to the organs, especially the liver and lungs. After localisation, the oncosphere develops into a metacestode (larval or hydatid cyst).[8] In sheep, the larvae are infective to canids within a year and may remain viable for a number of years.[9]

PREVALENCE AND RISK FACTORS

- A 2008 national study of gastrointestinal parasites in domestic dogs (owned and in shelters) did not find *E. granulosus* in any faecal samples, the authors noting infection is dependent on access to carcasses, an unlikely event for many pets in Australia.[10] A 2014 study of farm and rural

Hepatic echinococcosis in a sheep
(Public Health Image Library, CDC)

Life cycle of *Echinococcus granulosus*



Life cycle from Beugnet, F., et al (2018) Textbook of Clinical Parasitology in Dogs and Cat. Grupo Asis Biomedica, S.L.; Adapted from Carithers, D., et al (2012) Pet Owner Educational Atlas.

dogs in eastern Australia used coproantigen testing to detect *E. granulosus*.[11] The prevalence was 1.9% on the mainland (NSW, ACT, Qld, Vic) and 7.8% in Tasmania. The collection of faecal samples in Tasmania was more targeted than on the mainland, involving only rural dog owners living in the northern quarter of the state, where hydatid-infected cattle had previously been identified.

- A 2006 study of rural domestic dogs in farming areas detected *E. granulosus* coproantigens in 29% of dogs from farms in south-eastern NSW and 17.5% of dogs from farms in Victoria.[12] The majority of *E. granulosus* coproantigen-positive dogs occurred on farms with more than five dogs, where feeding commercial dry dog food was supplemented with wildlife carcasses. In the same study, 64% of owners in NSW and 95% of owners in Victoria admitted feeding raw meat of home-slaughtered animals or wildlife to their dogs.
- *Echinococcus granulosus* in wild dogs has been documented across eastern Australia, with prevalence between 50% in peri-urban wild dogs in south-east Queensland, and up to 100% in wild dogs in NSW.[13,14]

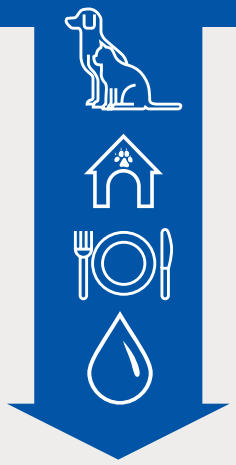
CLINICAL DISEASE

- Adult worms inhabit the small intestine of the definitive host and are not known to cause clinical disease, even in animals with significant worm burdens.[1,9]
- Case reports have documented rare instances of cystic echinococcosis in cats. These cases are hypothesised to be associated with immunosuppression.[2,3]

DIAGNOSIS

- Diagnosis of hydatid tapeworm infestation in a dog should be based on a history of access to raw offal. Clinical diagnosis via detection of eggs and proglottids on standard faecal flotations is unreliable and the eggs are morphologically identical to *Taenia* species.[1] Due to the zoonotic risk, direct examination of adult worms is not recommended.
- Diagnostic techniques include the detection of coproantigens (by coproantigen-ELISA) and/or copro-PCR, however these tests are not currently commercially available.[15,16]

TRANSMISSION



- Human infection occurs through accidental ingestion of eggs shed in dog faeces or close contact with an infected dog (*E. granulosus* eggs can adhere to dog hair and are immediately infective).
- Indirect transfer of *E. granulosus* eggs in contaminated food, water and soil can also cause infection.

IN HUMANS

PREVALENCE AND RISK FACTORS

- Hydatid disease in humans is uncommon in Australia, with the majority of cases believed to have been acquired overseas rather than from local exposure. Annually, 80–100 cases of echinococcosis are diagnosed in Australia (0.4 cases per 100,000 population). Higher rates have been documented in rural north-east and south-east New South Wales in one study, with an annual index of infection of 23.5 cases per 100,000 population in some communities.[17,18]
- Risk factors for human infection include previous episodes of echinococcosis, occupational and domestic exposure to dogs which consume raw offal, and travel to or from endemic areas. Children in endemic areas are likely to be at greater risk given their frequently close association with dogs and poor hand hygiene.
- Cystic echinococcosis in humans is not a Nationally Notifiable Disease in Australia.

CLINICAL DISEASE

- Cystic echinococcosis is characterised by the growth of hydatid cysts in internal organs. The incubation period in humans can vary from months to years.[19]
- After ingestion, *Echinococcus* eggs hatch and release oncospheres in the small intestine, migrating through the circulatory system to the liver and other anatomical sites, where cyst development begins.[20]

IN HUMANS *continued*

- The initial phase of the primary infection is typically asymptomatic. Small, well-encapsulated, non-progressive or calcified cysts may not induce clinical signs, and patients may remain asymptomatic for years or permanently.[9,21]
- Clinical presentation of hydatid disease is variable, with the nature and severity of signs dependent on the size, location and number of cysts. The onset of symptoms may be gradual once cysts become large enough to exert pressure on surrounding tissues and structures. Symptoms reflect impairment of the organ involved. Sudden onset of clinical signs is likely due to cyst rupture, which can lead to anaphylaxis or secondary bacterial infection.[20,22]
- Ruptured or leaking cysts can cause secondary echinococcosis, with leaking cysts releasing viable larval tissue stages (protoscolices). Surgical treatment can also cause trauma-induced rupture of primary cysts resulting in secondary hydatidosis.[19,22]
- The liver is the most frequently parasitised human organ, accounting for 50-70% of cases, followed by the lungs (20-30%) and less commonly the spleen, kidneys, heart, bones and central nervous system.[21]
- Diagnosis of cystic echinococcosis is based on clinical findings, imaging and serology. A standardised classification system is used for the analysis of cystic echinococcosis in the liver, which can also be applied to cysts located in other tissues. Cysts that are not accessible to ultrasound can be examined using other imaging modalities such as computed tomography (CT) or magnetic resonance imaging (MRI). Standard radiology is useful to diagnose thoracic and bone involvement. Microscopic examination of protoscolices post cyst fluid aspiration and histology can provide further evidence of the viability of cysts.[23]
- Treatment options for cystic echinococcosis vary depending on the number, size and stage of cysts, using criteria developed by the WHO, and range from observation alone, drug therapy alone, percutaneous drainage, and surgical excision.

KEY CONSIDERATIONS

1. An essential aspect of canine infestation is access to raw carcasses of livestock and wild animals. Dogs should not be fed raw meat or offal from on-farm slaughtering processes. Dogs, particularly in endemic or rural areas should be supervised or restrained to prevent scavenging on dead livestock and wild animals.
2. Infested dogs, even those with high worm burdens, do not show clinical disease.
3. If dogs have known or suspected exposure to *E. granulosus,* deworming every six weeks with praziquantel is recommended to minimise the public health risk from shedding of infectious eggs. Faeces from recently dewormed dogs should be disposed of by burning or deep burial.

Restricting access to raw carcasses (including offal) of livestock and wild animals can prevent *E. granulosus* infestation in dogs

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LEPTOSPIROSIS *(Leptospira* spp.*)*

- Leptospirosis, an acute bacterial infection caused by infection with pathogenic species in the genus *Leptospira,* is an important and emerging zoonotic disease globally.
- Although more than 300 different *Leptospira* serovars have been identified,[1] only a limited number have been demonstrated to infect dogs and cats.[2]
- Most cases in humans in Australia relate to occupational (agricultural) or recreational (e.g. water sports, kayaking) exposure. Infection via companion animals is thought to be uncommon but should be considered particularly in the context of a veterinary clinic with clinical cases of leptospirosis.

ACAZAP RECOMMENDATIONS

- Vets are at greater risk than the general population from companion animal transmitted leptospirosis due to frequent exposure to sick, potentially infected animals.
- Leptospirosis should be considered a differential diagnosis in any dog exhibiting signs of a non-specific illness or signs of haemorrhagic, renal or hepatic disease.
- In a veterinary practice setting where leptospirosis is suspected, consider restricting movement of the animal, disinfecting areas of contact, and placing the suspected cases into isolation facilities or restricted areas of the hospital. Consider an indwelling urinary catheter for urinary output. Catheter bags and materials contaminated with urine should be disposed of in clinical waste to minimise environmental contamination beyond the veterinary clinic.
- Preventative strategies should be considered for those at high occupational or recreational risk. These measures include the use of protective clothing and gloves, and covering cuts and wounds with waterproof dressings when in contact with potentially infectious material (urine or contaminated soil, mud or water). Hands should be thoroughly washed after potential exposure. Full personal protective equipment is recommended for managing cases in the hospital setting.
- Pressure washing of kennels and runs should be avoided as it may contribute to aerosolisation of urine. Regular hosing may also pose a risk of aerosolisation, and should be avoided where possible.
- Vaccination of dogs is possible, however immunity is serovar, or at best serogroup specific. Vaccination of dogs has been shown to reduce but not eliminate shedding.[3]
- In addition to the leptospirosis patient, any other dogs living in the same household should receive oral doxycycline therapy for two weeks.
- Whilst treated dogs represent a low risk to household members, until proper antimicrobial therapy is completed owners should avoid contact with their dog's urine, cover all cuts and abrasions with a waterproof dressing and wear gloves if cleaning up pet urine.

IN ANIMALS

AETIOLOGY AND EPIDEMIOLOGY

- Originally classified into two species (pathogenic *Leptospira interrogans* and non-pathogenic saprophytic *Leptospira bireflexa),* the taxonomy and nomenclature of *Leptospira* is complex and constantly evolving, with more modern classification based on genomic sequencing increasing the number of identified species. More than 300 serovars have been identified, although only a few have been shown to cause disease in companion animals.[1-3]
- Leptospires are maintained in different host-adapted species, dependent on the serovar, and thus an understanding of circulating serovars is important when considering risk

Introduced rats (wild and domestic) are the maintenance host of serovar Copenhageni

mitigation. Rodents are a common reservoir host for many serovars.

- Leptospires are primarily located in the proximal renal tubules of infected reservoir hosts, however other tissues and organs can also be a source of infection. Reservoir hosts (such as rats) typically do not demonstrate clinical signs but can harbour leptospires in renal tubules for extended periods of time, shedding into the environment via urine.
- Urinary shedding may be constant or intermittent, leading to contamination of soil, surface water, streams and rivers.
- If the bacteria come into contact with a susceptible animal or person, they can invade (via intact mucous membranes or breaks in the skin), spread through the body and cause generalised infections.

PREVALENCE AND RISK FACTORS

- An Australian seroprevalence study of shelter dogs in 2008 demonstrated 1.9% of tested dogs were currently, or had previously been infected with *Leptospira*.[4] State based variation was seen in terms of the prevalence and serovar involved. Seroprevalence was greatest in Victoria (2.8%), Queensland (2.5%) and New South Wales (2.3%). Serovar Copenhageni (in the Icterohaemorrhagiae serogroup) was the most prevalent serovar detected in this study, confirming findings of earlier studies.[5,6] More recently, clinical cases of leptospirosis associated with serovar Copenhageni have been reported in urban dogs in Sydney confirming the importance of this serovar in southern Australia.[7] Infection with serovar Hardjo has also been reported in a dog from Sydney who had visited rural NSW and was involved in herding animals. In north Queensland, serovar Australis is the predominant serovar infecting dogs.[8]
- For companion animals the risk of infection is related to exposure to the maintenance hosts (serovar dependent). For example, infection from the serogroup Icterohaemorrhagiae requires dogs to be exposed to rats or areas frequented by rats (e.g. dogs in kennel environments, ratters and pig hunting dogs). There is no published data on the prevalence of *Leptospira* in rats in Australia.
- There is limited data on *Leptospira* infection in cats in Australia. A small seroprevalence study showed 0% prevalence in feral cats in south-west Western Australia but as high as 42% in feral cats on Christmas Island, indicating current or prior exposure may be common in some geographic locations.[9] Overseas seroprevalence varies dependent on geography and study population but has been reported as high as 48%.
- Shedding occurs via urine, with one study reporting that 8.2% of dogs shed pathogenic leptospires irrespective of health status.[10] A recent study demonstrated cats can shed viable *Leptospira* spp. organisms.[11]

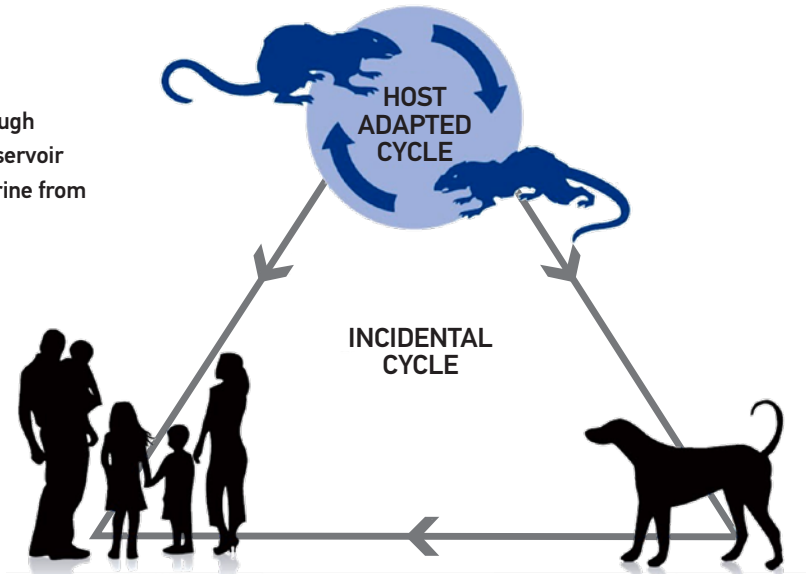
CLINICAL DISEASE

- In dogs, leptospirosis is classically associated with acute to sub-acute haemorrhagic, renal, or hepatic disease. Peracute disease has also been reported, associated with death with few preceding clinical signs. Based on seroprevalence data, subclinical infection with leptospires is common.
- Overt clinical disease in cats is rarely reported.
- Infected animals should be isolated (see recommendations) and treated with IV penicillin derivatives (ampicillin, amoxycillin) until able to accept oral doxycycline. Doxycycline should be given at 5 mg/kg PO q12h for two weeks. In contact household dogs should be tested and treated.

Dogs and humans may be infected from exposure to contaminated water sources

IN ANIMALS *continued*

Spillover infection of humans may occur through direct or indirect exposure to urine from a reservoir host, or less commonly due to exposure to urine from another infected incidental host



DIAGNOSIS

- Diagnosis may be based on identifying leptospires in body fluids, with PCR the primary modality. Serology may also be used for diagnosis. A single microscopic agglutination test (MAT) titre >1:800 is considered positive against non-vaccine serovars. Demonstration of seroconversion with a fourfold or greater increase in titre between samples taken 2 to 4 weeks apart is also considered diagnostic. Serology (MAT) is required to identify the infecting serovar.
- Infection results in an acute leptospiraemia lasting 7-10 days, followed by renal colonisation and leptospiruria, thus appropriate samples should be tested.[2] As the time of infection is typically unknown, simultaneous testing of blood and urine is recommended to increase diagnostic sensitivity.

PREVENTION

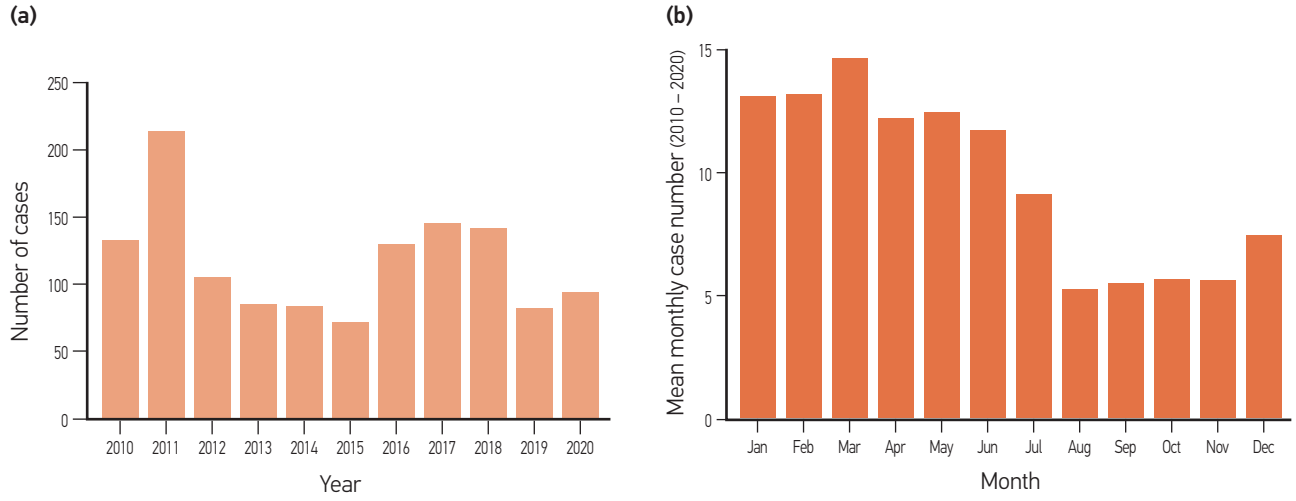
- Vaccination of dogs is possible, however immunity engendered by leptospirosis vaccines is serovar, or at best serogroup specific. In Australia, a registered vaccine is available against serovar Copenhageni and an unregistered vaccine against serovar Australis is available on conditional permit (available for dogs in Queensland and Northern Territory and for military/customs dogs in all states). *Leptospira* vaccines do not provide sterilising immunity but may reduce shedding.[3]

TRANSMISSION

- Leptospires are excreted constantly or intermittently in urine of infected carrier animals resulting in contamination of soil, surface water, streams and rivers.
- Organisms do not replicate outside the host, but may survive for prolonged periods. Environmental survival of leptospires is favoured by warm moist conditions, and may exceed 20 months in nutrient poor water.[12] Leptospires are susceptible to desiccation and UV light. Human and animal outbreaks have been linked to increased rainfall, flooding or contact with stagnant or slow-moving water.
- Leptospires gain entry to the body through skin abrasions (e.g. wounds and scratches) or across mucous membranes, including those of the gastrointestinal (via ingestion), respiratory (via inhalation) or genital tracts (via sexual transmission – very rarely in humans) and conjunctiva of the eyes.
- Therefore transmission is possible through direct contact with urine from infected animals or indirect contact with water, soil or food contaminated with urine containing leptospires.

IN HUMANS

Notified leptospirosis cases to National Notifiable Disease Surveillance System: (a) cases per year; (b) mean cases per month (2010-2020) – accessed 25 Jan 2021



PREVALENCE AND RISK FACTORS

- Occupational exposure or recreational activities that involve contact with contaminated water or soil is the most common source of human infection.[13]
- The risk of zoonotic infection is highest for people that work outdoors (banana plantation workers, sugar cane harvesters, sewage workers) or with animals (farmers, abattoir workers and veterinary staff).[14,15] Leptospirosis can also be a recreational hazard for campers, bushwalkers and those involved in a range of water sports.[14,15]
- Leptospirosis in humans is a nationally notifiable disease in Australia. Most cases are reported in young and middle-aged males. Increased incidence is associated with flooding events. Most cases occur in northern Australia.[14]

CLINICAL DISEASE

- Incubation period in humans is typically 5-14 days, but can be variable, with a range of 2-30 days reported.[14] In many cases, infection is subclinical or results in a non-specific flu-like illness (fever, headaches, muscle pain, nausea and vomiting).[15] Due to the mild and non-specific nature of infection, as is the case in dogs, human infections are also thought to be underdiagnosed. Aseptic meningitis may occur, more commonly in children and young adults.[15]
- Generalised conjunctival erythema – called conjunctival suffusion – is a useful clinical sign in leptospirosis, occurring in up to 50% of cases of clinical leptospirosis while being rare in other "flu-like" illnesses.
- Leptospirosis is a biphasic disease, with an initial infectious phase followed by an immune-mediated phase. Activation of host innate immunity and cytokine storm contribute to severe disease. Severe leptospirosis is characterised by dysfunction of multiple target organs and it is estimated that approximately 5 to 10% of human patients will develop severe signs of hepatic and renal disease (classically called Weil's disease), and up to 15% of these patients die as a result.[15]
- Some patients present with pulmonary involvement, a more common presentation of severe leptospirosis combined with Weil's disease. Pulmonary infection can progress from mild illness, to bilateral lung infiltrates, to an acute respiratory distress syndrome. Pulmonary haemorrhage can occur in some patients, also known as severe pulmonary haemorrhage syndrome (SPHS), with reported mortality rates from 50 to 70%.[16]
- Severity of disease is related to the infecting serovar (e.g. more severe with those of the Icterohaemorrhagiae serogroup) and host factors (age, immunocompetence etc.).
- Other than conjunctival suffusion that is frequently seen during the initial leptospiraemic phase, ocular inflammation, particularly in the form of uveitis, is well-described during the recovery phase of illness.
- Infection during pregnancy may result in abortion. Leptospires can be shed in breast milk.

KEY CONSIDERATIONS

1. Risk of infection in companion animals relates to direct or indirect exposure to reservoir hosts (e.g. rats).
2. Given the most common route of infection in humans is contact with urine from infected animals, in a veterinary clinic setting where leptospirosis is suspected or confirmed, consider preventative measures such as full personal protective equipment and appropriate management of animal waste, such as urine.
3. Vaccination is available for at risk dogs; however vaccination is serovar specific and does not provide sterilising immunity.

Leptospirosis should be considered a differential diagnosis in any dog exhibiting signs of a non-specific illness or signs of haemorrhagic, renal or hepatic disease.

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Q FEVER *(Coxiella burnetii)*

- *Coxiella burnetii*, an obligate intracellular gram-negative bacterium, is the causative agent of Q fever in humans and coxiellosis in animals.
- *Coxiella burnetii* is highly contagious and environmentally resilient, with only small numbers of organisms required to infect humans.
- Human infection is most commonly associated with exposure to animals at the time of parturition, particularly ruminants (primarily goats, cattle and sheep), however there is a growing awareness of the potential risk associated with periparturient companion animals.

ACAZAP RECOMMENDATIONS

- Exposure to periparturient animals is the biggest risk factor for pet-associated Q fever infection. Appropriate cleaning and disinfection of potentially contaminated areas is essential. *Coxiella burnetii* is a highly resilient organism and resistant to many disinfectants, heat and drying. Effective disinfectants include: 70% alcohol (30 min), hydrogen peroxide, and sodium hypochlorite (>5% solution).
- Given that inhalation is the primary route of transmission, extreme care should be taken to avoid aerosolisation during the birthing process (e.g. when reviving puppies and kittens) and when cleaning potentially infectious areas.
- Contaminated fabrics (e.g. drapes, towels, scrubs) should be autoclaved or disposed of via contaminated waste collection facilities. Standard washing and machine drying may result in aerosolisation.
- Personal protective equipment consisting of disposable water-resistant gowns, gloves, P2/N95 masks and eye protection should be used during all reproductive procedures.
- Vaccination is available for at risk individuals >15 years of age and is highly recommended for all people likely to be exposed through lifestyle or occupation (including veterinarians, veterinary nurses, veterinary students, animal refuge workers, wildlife rehabilitators and professional dog and cat breeders). Due to the requirement for pre-vaccination testing for existing immunity (serology and skin test), vaccination may not be available from all medical practitioners. A list of registered vaccinators can be found at www.qfever.org.
- Individuals at greater risk of severe consequences from infection (e.g. pregnant women, immunocompromised individuals and individuals with valvular disease) should avoid contact with periparturient and neonatal dogs and cats.
- Education for dog and cat breeders on the risks and their mitigation, such as the use of dedicated birthing areas away from household facilities and vaccination as per the *Australian Immunisation Handbook* is important.

IN ANIMALS

AETIOLOGY AND EPIDEMIOLOGY

- *Coxiella burnetii* is an obligate intracellular gram-negative bacterium with a life cycle that includes two distinct morphological and functional forms. The "large-cell variant" is the metabolically active intracellular form and the "small-cell variant" is the inactive extracellular pseudo-sporulated form that is highly environmentally resilient and transmits the infection between hosts.
- Dogs and cats are likely exposed through inhalation of organisms from close contact with parturient animals and birth products or other contaminated body secretions.
- Ingestion of contaminated meat from reservoir species is a potential route of infection in dogs and cats. *Coxiella* DNA has been detected in commercially available kangaroo meat, however whether this represents viable organisms, and if so what role it may play in the transmission to dogs and cats (and humans), is unclear.[1] Feeding raw kangaroo is a risk factor

Exposure to periparturient animals poses the greatest risk for zoonotic infection as high numbers of organisms can be found in placentae and reproductive fluids, including those from cats with normal parturition

for *Coxiella*-seropositivity in cats.[2] Feeding of raw meat is a common husbandry practice among Australian cat breeders, practiced by 89% in a published study.[3]

- *Coxiella burnetii* has been identified in a range of different arthropod vectors of which ticks are the most common, however the importance of vector-borne transmission is not clear.[4]

PREVALENCE AND RISK FACTORS

- In a large study of cats in eastern Australia, *Coxiella* seroprevalence ranged from 0-9.3%, depending on the lifestyle of the cats.[5] In this study no feral or shelter cats were seropositive. Owned pet cats had a seroprevalence of 1% while cattery-confined breeding cats had a prevalence of 9.3%.
- A large seroprevalence study in dogs reported *C. burnetii* exposure in 1.9-6.5% of dogs, with prevalence dependent on the study population. Prevalence in shelter dogs, breeding dogs, and pet owned dogs (1.9%, 2.3% and 3.0% respectively) were not significantly different, while free-roaming dogs in remote Aboriginal communities were 2.8 times more likely to be seropositive than dogs from other populations (prevalence 6.5%).[6] A study of pig hunting dogs in tropical north Queensland reported a seroprevalence of 23%.[7]
- A subsequent study in north-western NSW, in an area with a relatively high incidence of human Q fever, reported a seroprevalence in healthy dogs and cats of 26.1% and 13.1% respectively, suggesting that exposure of pets and people occurs through a shared common source.[2] In this study, *C. burnetii* DNA was not detected in blood or tissues (post desexing) from any dogs or cats, suggesting bacterial shedding is uncommon.[2]

CLINICAL DISEASE

- There is a lack of data supporting a role for naturally acquired *C. burnetii* in causing clinical disease in dogs and cats.
- There is no direct evidence of reproductive disorders in dogs or cats due to coxiellosis,[8] however outbreaks of Q fever in humans have been reported associated with exposure to parturient dogs and cats who have given birth to young which have died during the immediate perinatal period.[9,10]
- In experimentally infected cats, non-specific clinical signs of fever, lethargy and anorexia have been reported.[11]

DIAGNOSIS

- Laboratory diagnosis of coxiellosis in dogs and cats is not commonly performed outside of a research setting. Confirmation of coxiellosis in dogs and cats likely requires serial PCR and serological testing, however a validated method for these species has not been determined as it has for humans.

TRANSMISSION

- High numbers of organisms are found in placentae and reproductive fluids, therefore exposure to periparturient animals poses the greatest risk.
- Infection is primarily through inhalation of aerosolised bacteria or dust contaminated with birth products. Very low infectious dose is reported in humans via inhalation.[12]

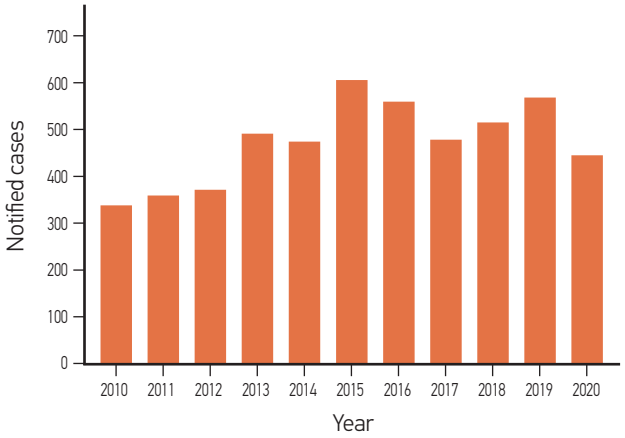
IN HUMANS

PREVALENCE AND RISK FACTORS

- Q fever in humans is uncommonly reported (approximately 500 notified cases per year in Australia) but there is likely a reasonable degree of under-reporting due to asymptomatic infection and difficulty in diagnosis. This is supported by seroprevalence rates of up to 7% in the general population.[13]
- There are regional differences in incidence across Australia, with New South Wales and Queensland accounting for 87% of total notifications.[14] Prevalence is highest in Queensland (6.3 cases annually per 100,000 population), followed by New South Wales (3.1 cases annually per 100,000 population) and South Australia (1.1 cases annually per 100,000 population).[15] Individuals living on farms in outer regional or remote areas are at greater risk of contracting Q fever.[13,16]
- Gender and age differences are seen, with notifications more common in older males.[14]
- Occupational exposure through the livestock industry (farmers, abattoir workers, shearers, livestock transport drivers, veterinarians, veterinary nurses etc.) or exposure to other animals, particularly parturient animals, is a risk factor for Q fever.[14,15] Almost two-thirds of notifications are from individuals with known occupational or environmental risk factors.[14] *Coxiella burnetii* is highly infectious and Q fever can occur in people with remote (e.g. living down-wind from abattoirs) or very transient exposure, hence clinical history and consideration of Q fever is very important in making a diagnosis. Human-to-human transmission is extremely rare.
- Seroprevalence in (unvaccinated) veterinary workers in Australia was 19%, with seropositivity associated with working in an outer regional/remote area and having spent >50% of total career working with ruminants.[17]
- Sporadic cases of disease in veterinary staff, animal carers, and breeders working with dogs and cats are reported.[3,18,19]

CLINICAL DISEASE

- Q fever can cause both acute and chronic infection, ranging in severity from asymptomatic to mild to severe. Differences in clinical manifestations are seen with geographic variation which may be the result of differences in regional strains of *Coxiella burnetii*.
- Acutely, Q fever can manifest as an influenza-like illness with fever, headache, malaise and myalgia. Biochemical hepatitis (elevation in liver function tests without clinical evidence of hepatitis) is common and clinically evident hepatosplenomegaly may be present. Non-productive cough along with a febrile systemic illness are typically present in Q fever pneumonia. Although characteristically described as an 'atypical' pneumonia, it can be severe and sometimes fatal.



Q fever notifications by year in Australia from 2010 to 2020. Data from National Notifiable Diseases Surveillance System, accessed April 2021.

- Chronic Q fever, also called persistent localised infection, can occur months or years after acute infection regardless of whether the acute infection was clinically apparent or asymptomatic. Patients with chronic Q fever typically have a febrile illness with non-specific symptoms, consistent with PUO (Pyrexia of Unknown Origin), with focal clinical manifestations that vary with the site of persisting infection. The cardiovascular system is the most common organ system implicated, with chronic Q fever endocarditis and vascular bed infections (e.g. mycotic aneurysm) both occurring. Persistent osteomyelitis can occur, with multi-focal osteomyelitis more characteristic in children. Less commonly a variety of focal organ system infections have been reported.
- Post-infectious chronic fatigue-like syndrome is well-described and is thought to result from the ongoing presence of non-viable *Coxiella* antigens causing persistent immune cytokine stimulation.
- Infection in pregnancy, particularly during the first trimester, is associated with an increased likelihood of obstetric complications including intrauterine growth restriction and foetal death.
- Q fever in humans is typically diagnosed with serology. For acute infection a single high phase 2 IgM titre is suggestive, however serology on paired acute and convalescent sera demonstrating a greater than fourfold increase in IgG titre is preferred. Patients with chronic Q fever will typically have raised phase 1 antibodies. Molecular diagnosis with PCR is increasingly being used, and may be performed on blood or tissues (e.g. infected valve tissue).

KEY CONSIDERATIONS

1. While Q fever has traditionally been associated with production animal species it is now apparent that companion species can pose a risk, albeit much lower.

2. Although seroprevalence is relatively high in some groups of animals, shedding and transmission is more likely during the periparturient period. Veterinarians have an important role to play in the education of dog and cat breeders on the management of risks associated with *C. burnetii*.

3. Implementation of risk mitigation strategies is important for high risk groups and situations. This should include PPE, appropriate cleaning and disinfection and most importantly, vaccination of at-risk groups.

Vaccination against Q fever in humans is effective and is strongly recommended for all staff working in a veterinary clinic.

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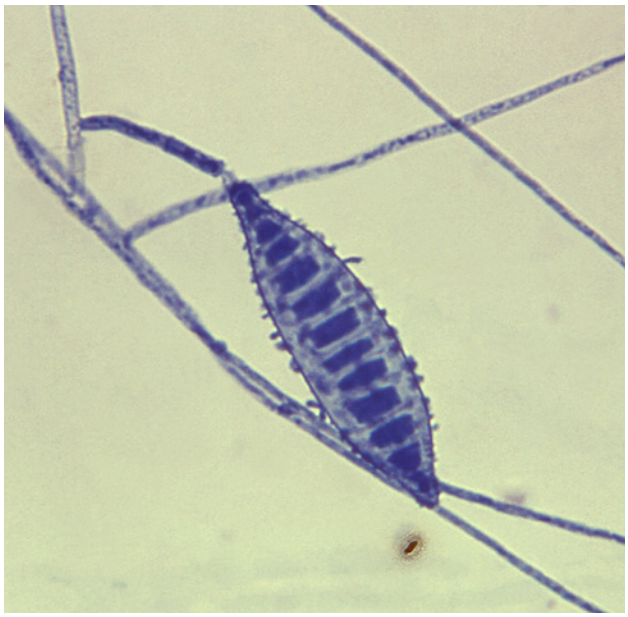
RINGWORM

- Dermatophytes are filamentous fungi with a high affinity for keratin, capable of causing superficial cutaneous infections in human and animal hosts.[1,2]
- Over forty species from three genera (*Microsporum, Trichophyton* and *Epidermophyton*) are identified, and more than twenty species have been isolated from companion animals.[3,4]
- Dermatophytosis (also known as ringworm) is a common superficial fungal infection of dogs and cats, most commonly involving *Microsporum* and *Trichophyton* species.[4] *Microsporum canis* is the species most commonly implicated in zoonotic dermatophyte disease of humans, however human dermatophytosis is primarily non-zoonotic, associated with anthropophilic dermatophytes (e.g. *Trichophyton rubrum, T. mentagrophytes*) and geophilic dermatophytes (e.g. *Nannizzia gypsea*; formerly *Microsporum gypseum*) acquired via human-to-human contact or contact with soil respectively.

ACAZAP RECOMMENDATIONS

- If a cat or dog is diagnosed with dermatophytosis, all in-contact animals and household human contacts should ideally be screened for dermatophytes using fungal culture. Both asymptomatic and symptomatic individuals and animals should be treated to prevent the cycle of transmission continuing.
- Topical therapy can decrease the zoonotic risk associated with dermatophytosis by disinfecting the hair coat and minimising environmental contamination. Twice weekly application of a fungicidal shampoo containing miconazole is recommended for the treatment of generalised dermatophytosis in dogs and cats.[5] Careful clipping of hair around localised lesions is recommended, however full coat clipping may contribute to further spread of skin lesions associated with skin microtrauma and increased environmental contamination if not carefully performed.[5]
- Topical therapy may be combined with systemic antifungal treatment, particularly for recurrent and/or generalised infections, in immunocompromised animals, or when managing outbreaks in large facilities. Although there are no registered systemic antifungal products for dogs and cats in Australia, a number have been reported effective in these species including itraconazole, terbinafine and griseofulvin, although the latter may be associated with a greater potential for adverse events.[5]
- Animals with chronic *M. canis* infections should be evaluated for underlying diseases.
- Vacuuming and mechanical cleaning of the environment is essential to reduce the presence of infective material. To minimise the potential for redistribution of infective material, vacuums incorporating a HEPA exhaust filter are recommended.
- In a veterinary hospital or shelter setting, additional environmental decontamination using a disinfectant with anti-fungal efficacy (e.g. 1:10 dilution of household bleach) is vital to minimise fomite carriage and potential re-infection. Surfaces should be thoroughly cleaned prior to disinfection.
- Potential fomites should be discarded where possible or appropriately cleaned. Bedding and blankets should be washed daily in water and bleach.
- All heating and cooling vents should be vacuumed and disinfected. All non-porous surfaces (floors, surfaces, counter tops) should be thoroughly cleaned.
- Education of veterinary and animal handling staff about the risks of zoonotic infection is essential. Gloves should be routinely worn when examining animals with skin lesions.
- In the veterinary clinic, infected animals should be isolated and gloves and protective clothing should be worn when handling infected animals and bedding.
- If a human patient is diagnosed with a dermatophyte infection, examination and testing of household pets is recommended to determine their role, if any, in the infection. This is particularly important in the case of recurrent human infections.

IN ANIMALS



Photomicrograph of *Microsporum canis* macroconidium. *Microsporum canis* macroconidia are spindle-shaped, have a thick wall and roughened surface, and are divided into six or more internal compartments
(Public Health Image Library, CDC)

Patchy alopecia in a kitten with ringworm
(Courtesy of Prof. Jacqui Norris)

AETIOLOGY AND EPIDEMIOLOGY

- Cats and dogs are recognised as the natural hosts of *M. canis*, the most frequently isolated dermatophyte from companion animals.[6] *Microsporum canis* also infects humans and other animal species including cattle, horses, pigs, goats, rabbits and guinea pigs.[3]
- *Microsporum canis* infections may be acquired through direct or indirect contact with an infected animal. Arthrospores shed from animal hair and scales can remain infective for 12-24 months.[4,5] Contaminated fomites, including bedding, collars, brushes and toys, may be a source of infection.
- Studies on the fungal flora of healthy cats and dogs have shown *M. canis* is not part of the normal skin microbiome.[5]

PREVALENCE AND RISK FACTORS

- Dogs and cats may show clinical signs or be asymptomatic (subclinical) carriers. Asymptomatic carrier states are common in cats, particularly long-haired breeds. Asymptomatic infection is rare in dogs but has been reported in Yorkshire Terriers. Persian cats and Yorkshire Terriers are noted in the literature as predisposed to dermatophytosis.[5-9]
- The highest incidence of dermatophytosis is reported in kittens, puppies, immunocompromised animals and long-haired cats. In immunocompromised animals, the outcome may be a multifocal or generalised skin disease.[5,8]
- Dermatophytosis is more common in warm, humid climates and may be seasonal in temperate areas.[4,8]
- Animals in group housing (kennel environments, catteries, animal shelters) and stray animals may be at greater risk of exposure to dermatophytes. Companion animals living with other cats and dogs are also at greater risk of acquiring dermatophytes.[4,8]

CLINICAL DISEASE

- Dermatophytosis is an infection of the keratinised layer of the epidermis, hair shafts and stratum corneum. Patchy alopecia is the most common clinical sign, with or without associated skin lesions. Skin involvement can be localised, multifocal or generalised, and skin lesions may include any combination of papules, crusts, scaling, erythema, seborrhoea, and alopecia (patchy alopecia is more common than circular alopecia). Affected animals are frequently non-pruritic, or if pruritus is present it is generally mild.[5,8]
- In some cats, dermatophytosis can present as a papulocrustous dermatitis ('miliary dermatitis') affecting mainly the dorsal trunk.[7,8]
- The incubation period of *M. canis* ranges from 1-4 weeks.[3,5] Lesions caused by dermatophytes can be mild to severe depending on several factors including the infecting species, infective dose, virulence factors, location of infection, presence of secondary infections, physiological stress and

environmental conditions.[3,5] In most immunocompetent hosts, dermatophytosis is a self-limiting disease.

DIAGNOSIS

- Diagnosis of dermatophytosis is based on clinical suspicion in conjunction with the results of Wood's lamp and direct examination to document active hair infection and/or fungal culture.[5] False-negative results with Wood's lamp examination may occur as fluorescence is only seen with dermatophytosis due to *M. canis*, and not all *M. canis* isolates will fluoresce.
- PCR detection of dermatophytes is difficult to interpret as a positive PCR does not necessarily indicate an active infection.[5]

TRANSMISSION

- The main mode of transmission of *M. canis* is through direct or indirect contact with the coat or skin lesions of infected animals.
- Contact with accumulated scale and hair in the environment and fomites (including furniture, linen, brushes etc.) are also potential sources. Contact with a contaminated environment in the absence of concurrent skin trauma is considered a rare source of infection for humans and animals.[5]

IN HUMANS

PREVALENCE AND RISK FACTORS

- Dermatophytes are grouped as either anthropophilic, zoophilic or geophilic depending on whether their primary source is human, animal or environmental, respectively. Whilst dermatophytosis is a common skin disease in people, the rate of transmission from companion animals to humans is unknown.
- *Microsporum canis* is a causative pathogen for the human dermatophyte skin infections tinea capitis (infection of the scalp, hair follicles and surrounding skin) and tinea corporis (infection of glabrous skin, with lesions that may involve the trunk, neck, arms and legs).
- It is estimated that approximately 50% of humans exposed to *M. canis* infected cats acquire the infection, and in 30-70% of households with an infected cat at least one cohabitating human will become infected.[10]
- Data on human skin infections with *M. canis* in Australia is limited. Melbourne data on 12,316 dermatophyte isolates collected during 1996–1998 found that *M. canis* was responsible for 75% of laboratory-diagnosed tinea capitis cases.[11] A review of superficial fungal cultures submitted to a commercial laboratory in 2013 identified 7.4% of the culture-positive samples as zoophilic dermatophytes, with equal numbers of *M. canis* and *Trichophyton interdigitale*.[12] Zoophilic fungal infections were more likely in younger patients.
- Tinea corporis can be caused by various dermatophyte species, however patients in close contact with companion animals are commonly infected with *M. canis.* The incubation period is 1-3 weeks.[13] It occurs most frequently in post-pubertal children and young adults, with children more likely to contract zoophilic infections through contact with pets.[13]

Ringworm lesions on the face and arms of a child
(Courtesy of Prof. Richard Malik)

CLINICAL DISEASE

- Tinea corporis typically presents as a well-demarcated, single or multiple, oval or circular, mildly erythematous lesion with a raised border (the characteristic 'ringworm' lesion). Varying levels of pruritus may be present. In immunocompromised individuals, tinea corporis can present as a disseminated skin infection or as a subcutaneous/deep abscess.[13]

IN HUMANS *continued*

- *Microsporum canis* is considered one of the most common causes of tinea capitis in children and can be divided clinically into inflammatory and non-inflammatory types.[14,15] The non-inflammatory type is characterised by areas of patchy circular alopecia, stubbled hair and mild scaling. The inflammatory type has lesions with diffuse, patchy alopecia, erythema, crusting scale, kerion formation and pustules. It may be associated with painful regional lymphadenopathy.[16] Tinea capitis occurs mainly in children between 3 and 14 years of age but can affect any age group.[17] It may also involve the eyelashes and eyebrows.[17]
- Immunocompromise can lead to impaired hair shaft strength and growth allowing dermatophyte colonisation. Predisposing factors for dermatophyte infection include underlying diseases such as diabetes mellitus, immunosuppressant medications, neoplasia and anaemia. In immunocompromised individuals the most common complication of *M. canis* infection is a protracted treatment time.[5,17]
- Human diagnosis traditionally relies on skin scrapings for detection of dermatophytes and fungal culture for confirmation and species differentiation. Culture for dermatophytes requires up to 4 weeks. Increasingly, multiplex dermatophyte PCRs are being introduced routinely, as these can confirm and differentiate dermatophyte species within 24 hours of testing, thus aiding clinical management.

KEY CONSIDERATIONS

1. Approximately 50% of humans exposed to *M. canis* infected cats acquire the infection, and in 30-70% of households with an infected cat, at least one cohabitating human will become infected.
2. Diagnosis in animals is based on a combination of clinical suspicion, the results of Wood's lamp and microscopic examination and/or fungal culture.
3. As shed arthrospores can remain infective for 12 to 24 months, contaminated fomites (such as bedding, collars, brushes and toys) may be a source of infection or ongoing re-infection. Thorough cleaning and disinfection is needed to minimise the risk.

Following diagnosis in a pet, all in-contact animals and household members should be screened for dermatophytes using fungal culture. Individuals and animals testing positive should be treated irrespective of whether they are symptomatic.

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SALMONELLOSIS *(Salmonella* spp.*)*

- *Salmonella* are gram-negative bacterial rods that are widely distributed in domestic and wild animals. More than 2,600 serovars of *Salmonella enterica* are recognised which vary in geographic distribution, host specificity and pathogenicity.[1] Serovars are divided into typhoidal and non-typhoidal *Salmonella* (NTS) serovars. The former are highly host adapted, with humans the exclusive reservoir, while the latter have a broad host range.
- The typhoidal serovars, Typhi and Paratyphi (A, B, and C), the causative agents of typhoid and paratyphoid fever respectively, are not zoonotic.
- Most human infections with NTS serovars are acquired from contaminated food (estimated at 71%), with direct transmission from animals (including companion animals) estimated to account for only 4% of cases.[2]

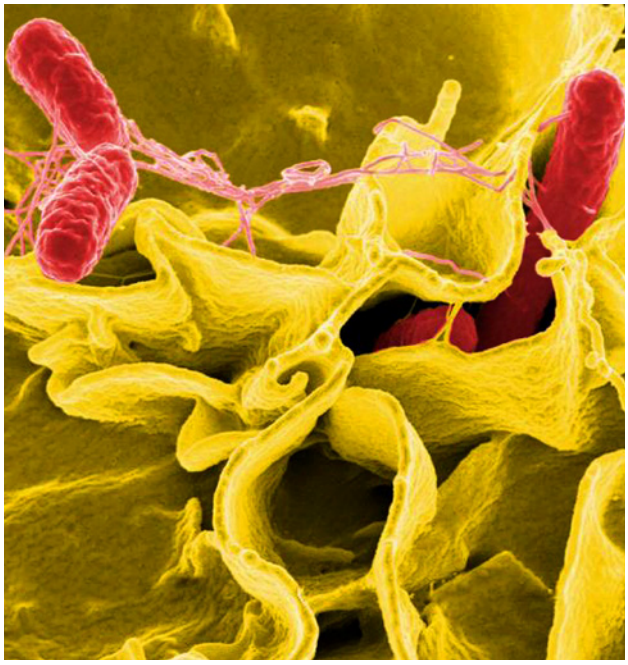
ACAZAP RECOMMENDATIONS

- Wash hands immediately after direct contact with any animal (especially pet reptiles and amphibians) and following contact with animal food or treats, food bowls, animal bedding and animal faeces.
- Avoid feeding raw meat diets to dogs and cats, or if fed, consider the potential for zoonotic infection through contact with the diet or the faeces of animals which have consumed the diet.
- Store pet food separately from food intended for human consumption.
- Maintain cats indoors to reduce risk of acquiring infection via predation of wildlife (especially reptiles).
- Appropriate cleaning and disinfection of bowls and contact surfaces is recommended.
- Animal faeces should be picked up and disposed of immediately.
- Due to the risk of intrauterine infection and abortion, pregnant women should be advised of precautions to take to prevent infection, including avoiding handling of raw diets and pets with diarrhoea. Where possible, disposal of pet faeces and litter tray management should be undertaken by other members of the household.
- It is not recommended to screen healthy animals for *Salmonella.*
- In a veterinary clinic or animal facility setting (e.g. kennels, shelters), isolation of all animals with documented or potentially infectious diarrhoea is recommended.
- Given the known source of *Salmonella* transmission to humans from reptiles and amphibians, limiting access to these pets for children under five years of age should be considered. Pet dogs and cats should be restricted from interacting with reptiles and amphibians to avoid infection.

IN ANIMALS

AETIOLOGY AND EPIDEMIOLOGY

- The most common source of infection for companion animals is ingestion of contaminated raw pet food or treats, including pig ears. There are rare reports of commercial dry pet food contaminated with *Salmonella*.[3] Animals that scavenge or predate wildlife, in particular cats, may be infected with wildlife associated strains.
- *Salmonella* can survive for a prolonged period in the environment (weeks to years), multiply rapidly in contaminated food sources and readily survive freezing for several weeks.[4,5]



Digitally colourised scanning electron microscopic (SEM) image depicting a number of *Salmonella* spp. bacteria (red) in the process of invading an immune cell (yellow)
(Public Health Image Library, CDC)

PREVALENCE AND RISK FACTORS

• Globally, the prevalence of *Salmonella* carriage in dogs is reported to be between 0-44% (median, 4%; mean, 7.7%) with factors such as the study location and study population (sick versus healthy dogs, husbandry conditions etc.) significantly impacting prevalence.[6] In Australia, a published study of healthy dogs in Brisbane in 1969 reported a prevalence of 6.9%.[7] More recently, PCR analysis of faecal samples from dogs with diarrhoea in Western Australia demonstrated a prevalence of 8.3%.[8]

• Reported global prevalence in cats varies from 0-13.6% (median, 2%; mean, 3.9%).[6] No published data is available on the prevalence of *Salmonella* in cats in Australia.

• Consumption of raw food or treatment with antimicrobials have been identified as risk factors for shedding *Salmonella* in faeces. A study in Canada reported the odds of shedding *Salmonella* to be 23 times greater for dogs fed a raw food diet compared to those on standard commercial rations.[9]

• Rural dogs are more likely to shed *Salmonella* than urban or suburban dogs,[6] and contact with livestock has been identified as a risk factor. Higher rates of infection have been identified in group-housed dogs, including greyhound breeding facilities.[10]

CLINICAL DISEASE

• Clinical salmonellosis is rare in dogs and cats, with most infections asymptomatic. Bacterial colonisation of dogs and cats is usually transient, with animals naturally clearing the infection over a period of weeks.

• Clinical disease and shedding is more common in immunosuppressed dogs and cats, young animals, pregnant animals, those in crowded conditions, and those with underlying disease (such as neoplasia, diabetes mellitus, retroviral infection and immune-mediated conditions).[4]

• Enteric manifestations of disease range from mild to severe and the character of the diarrhoea can vary (e.g. mucoid, watery, haemorrhagic) and is therefore not diagnostic. Septicaemia may occur with or without gastrointestinal signs and severely affected animals may develop septic shock. Dissemination of bacteria may rarely result in *Salmonella* seeding in distant organs with resultant organ dysfunction, even when enteric clinical signs are absent.

• Animals with enteric salmonellosis should be provided with supportive care. Antimicrobial treatment is not indicated and may prolong shedding. Indiscriminate antimicrobial treatment may result in a subclinical infection becoming clinical.

• Inappropriate use of antimicrobials to treat uncomplicated enteric salmonellosis is a societal risk as it leads to the development of resistance.

DIAGNOSIS

• Diagnostic testing was traditionally performed solely using faecal culture, however it has been mostly replaced by multiplex PCR screening, with culture performed on PCR-positive samples for antimicrobial susceptibility testing. The increased sensitivity of PCR may result in a diagnostic dilemma given the presence of asymptomatic carriers.

• The significance of a positive result must be interpreted in the clinical context and with a healthy dose of scepticism, even in sick animals. *Salmonella* may be present in the faeces of animals with diarrhoea associated with another cause as concurrent illness may increase shedding in a subclinical carrier.

• Regardless of its contribution to clinical disease in an individual animal, positive animals may be a source of infection for other animals and humans.

• Antimicrobial resistant strains are documented in multiple species. These are not inherently more pathogenic but may pose problems in circumstances where antimicrobial treatment is required.

TRANSMISSION

- Faecal-oral transmission through direct contact with pets or faeces, or indirect contact with material contaminated by faeces.
- Human and animal infection may occur due to exposure to a common source (e.g. pet food or treats).
- Reptiles and amphibians are a known source of salmonellosis in people, directly, or potentially indirectly, through intermediate infection of cohabitating dogs and cats.

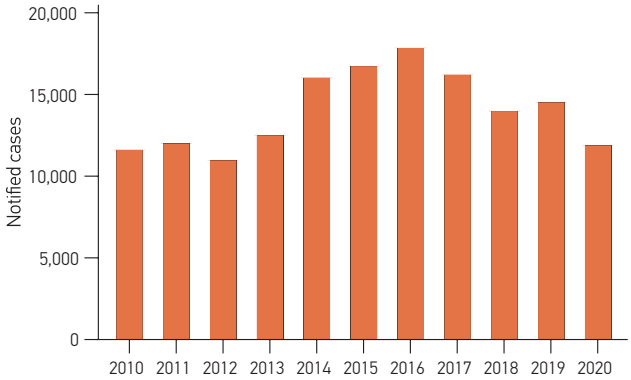
IN HUMANS

PREVALENCE AND RISK FACTORS

- Approximately 14,000 cases of non-typhoidal salmonellosis are reported annually in Australia, however it is estimated there are seven unreported cases for every one report.[11] The most common serovar in humans in Australia is *S. enterica* ser. Typhimurium. Most infections are foodborne in origin.[2]
- A number of host factors are associated with increased susceptibility to salmonellosis (lower initial inoculum required)[12], more severe infection, more protracted disease course or increased risk of complications:
  - Gastric acidity – patients on proton pump inhibitors require a lower inoculum.[13]
  - Age – neonates, children under the age of five and the elderly.[14]
  - Decreased cell mediated immunity – e.g. HIV, transplantation, immunosuppressive therapy.[14]
  - Altered intestinal flora (e.g. antimicrobial therapy).

CLINICAL DISEASE

- Uncomplicated cases present with gastroenteritis which begins 6-72 hours after exposure. Diarrhoea and abdominal pain are frequently observed. The condition is typically self-limiting, lasting 3-7 days. Exposure to large inocula can result in a shorter incubation period and more severe disease.[15] Typical shedding period is up to 6 weeks, although longer shedding has been reported. Shedding is longer in pregnant patients, in immune compromised patients and in patients given antimicrobial therapy.[16]
- Bacteraemia is reported in approximately 5% of immunocompetent patients, with a higher incidence in the very young, very old, and immunocompromised.[16] The consequences of *Salmonella* bacteraemia are more serious in adults due to the comorbidities of age, and may include:
  - Vascular complications – seeding of atherosclerotic plaques leading to infectious endarteritis, including seeding of prosthetic vascular grafts.
  - Focal infections including endocarditis, meningitis, septic arthritis and osteomyelitis, or pneumonia.
  - Intrauterine infection may result in abortion in pregnant patients.[17]
- Some serovars are more likely to result in invasive extraintestinal disease (e.g. Typhimurium, Dublin, Choleraesuis).[16] Some of these have been reported in dogs and cats, however good serovar prevalence data is lacking.
- The presence of multi-drug resistance, including to fluoroquinolones, third generation cephalosporins and more recently carbapenems, is of great concern internationally.



Salmonellosis notifications by year in Australia (2010 to 2020) from National Notifiable Diseases Surveillance System, accessed April 2021

KEY CONSIDERATIONS

1. Most non-typhoidal *Salmonella* infections in humans are foodborne in origin, however direct contact with dogs and cats (and their food) may be a source of infection.
2. Veterinary practitioners should advise owners of the risks of zoonotic pet-associated salmonellosis, including from pet food and from clinically well animals.
3. Avoid feeding raw meat diets to dogs and cats, or if fed, consider the potential for zoonotic infection through contact with the diet or the faeces of animals which have consumed the diet.

Antimicrobial treatment for animals with enteric salmonellosis is not indicated and may prolong shedding. Inappropriate use of antimicrobials to treat such cases is a societal risk as it leads to the development of resistance.

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SARCOPTIC MANGE *(Sarcoptes scabiei* var. *canis)*

- *Sarcoptes scabiei* has a broad host range, capable of infesting more than 100 mammalian species, including companion animals, livestock, wildlife and humans.[1] *Sarcoptes scabiei* is considered to be a single species divided into strains based on host specificity.
- Sarcoptic mange (or canine scabies) is a highly contagious, pruritic, ectoparasitic disease of dogs caused by the dog adapted variety, *S. scabiei* var. *canis.*
- Human scabies is mostly associated with the human host-adapted variety *S. scabiei* var. *hominis* after direct human-to-human transmission, however zoonotic infestation with *S. scabiei* var. *canis* from dogs may occur.

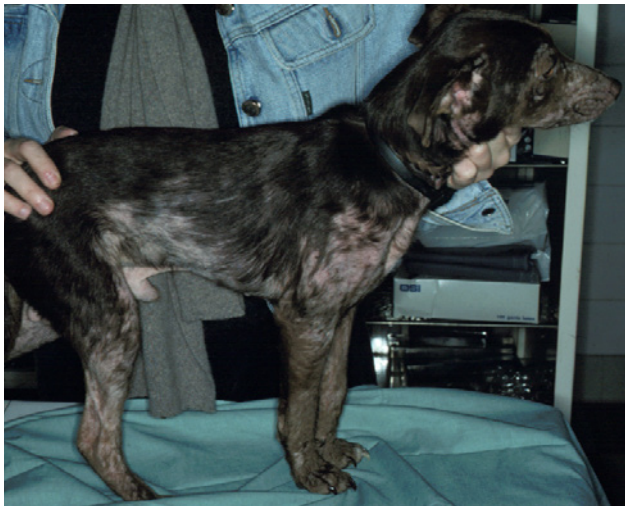
ACAZAP RECOMMENDATIONS

- An all year-round parasite control program in dogs with a parasiticide registered to treat and control mites and other ectoparasites is recommended.
- Thorough cleaning or disposal of bedding and grooming equipment from mite-infested dogs is essential. Whilst mite survival off the host is poor, fomites are a potential source of re-infestation. Bedding, towels and other materials should be washed (preferably on a hot cycle) or subjected to heat (from a clothes dryer).
- Environmental control products containing a pyrethroid may be effective against *S. scabiei,* however this is generally not required to manage infestations if animals are treated with an effective acaricide and appropriate cleaning is performed as detailed above.
- All dogs in the household should be treated concurrently. In multi-dog households, it is possible some dogs may harbour *Sarcoptes* mites without demonstrating clinical signs. Other pets in the household (such as cats, guinea pigs and rabbits) should also be checked for mites.
- Individuals exposed to infested pets should seek medical advice if they develop any cutaneous lesions. All members of the family and close contacts should seek medical advice and be treated if necessary.

IN ANIMALS

AETIOLOGY AND EPIDEMIOLOGY

- *Sarcoptes scabiei* var. *canis* has a host preference for dogs and other canids, including foxes. *Sarcoptes* infestations of cats have been reported but are rare.[2]
- Sarcoptic mange is non-seasonal and highly contagious, with transmission primarily occurring through direct contact. Cross infestation of *Sarcoptes scabiei* var. *canis* between dogs and foxes is not uncommon in dogs with a history of access to foxes.[2] Transfer of mites between hosts may also occur indirectly through contaminated bedding, cages or grooming equipment.[3]
- *Sarcoptes* mites are obligate parasites, with their entire life cycle taking place on host animals (on the skin surface and in tunnels burrowed into the epidermis). Mites dig out tunnels or burrows in the horny layer of the epidermis, with females laying 2-3 eggs per day. Tissue-feeding larvae moult two days later, either moving to the skin surface to dig new moulting pockets or remaining in the tunnels where they hatch. After 4-6 days, larvae moult into protonymphs, followed by tritonymphs which then develop into adults. Males live for about 3-4 weeks while females live for up to 3 months.[4] The prepatent period of *S. scabiei* var. *canis* is 14-21 days, with mites beginning to lay eggs within approximately three days of becoming adults.[4]
- *Sarcoptes* mites survive for a short time in the external environment (1-2 days) at room temperature (25ºC and 25-97% relative humidity).[5] High relative humidity and low temperature prolong the environmental survival of mites.[5]



Typical distribution of skin lesions in a dog with sarcoptic mange

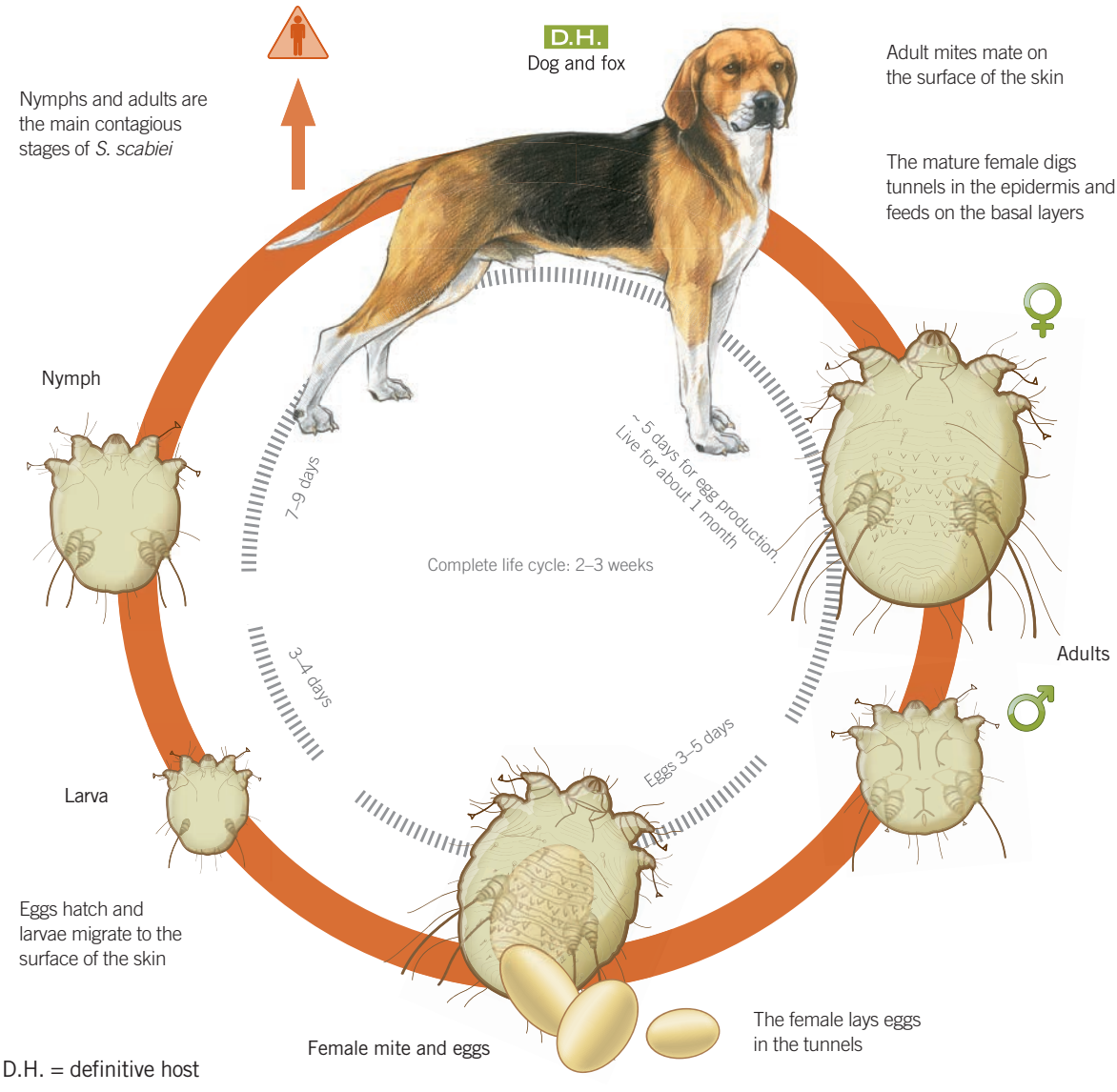
PREVALENCE AND RISK FACTORS

- There is no published prevalence data for sarcoptic mange in dogs in Australia.
- Sarcoptic mange can affect dogs of all ages and breeds, although younger dogs (less than two years of age) are more commonly affected.[6] Immunocompromised dogs are more susceptible to severe disease.[4]

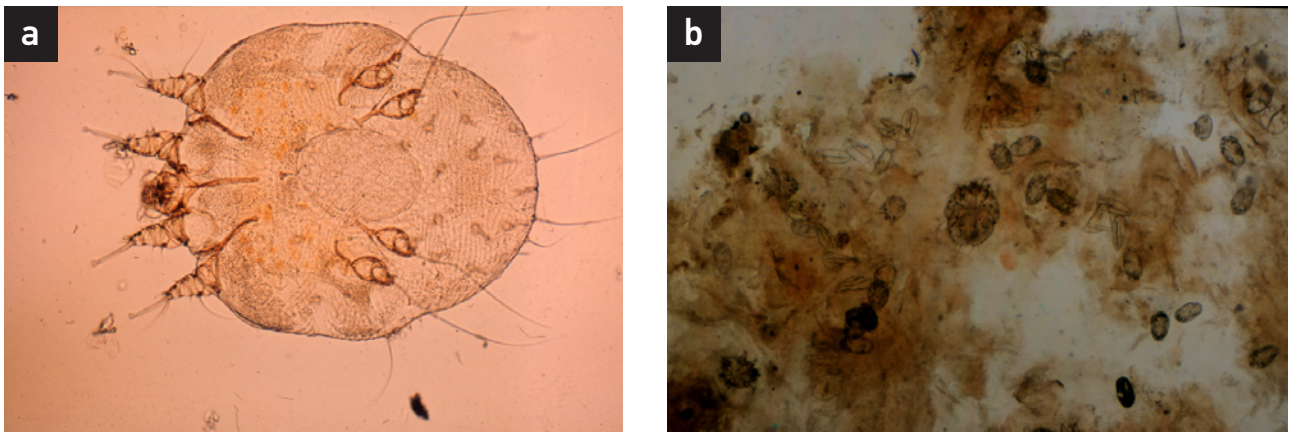
CLINICAL DISEASE

- *Sarcoptes* infestations in dogs are initially characterised by urticaria, progressing to intense pruritic lesions associated with localised dermatitis, papules, erythema, excoriation, and alopecia. Lesions are more common over the pinnae, face, limbs (particularly elbows and hocks) and the ventral abdomen.[4,7,8] Dogs may begin to be pruritic prior to the development of obvious skin lesions.

Life cycle of *Sarcoptes scabiei* var. *canis*



Life cycle from Beugnet, F., et al (2018) Textbook of Clinical Parasitology in Dogs and Cat. Grupo Asis Biomedica, S.L.; Adapted from Carithers, D., et al (2012) Pet Owner Educational Atlas.



Adult *Sarcoptes scabiei* mite (a) and skin scraping showing mites and eggs (b)

- Clinical signs in dogs may be due to physical irritation caused by the presence of mites and/or hypersensitivity reactions to salivary antigens (type I, III and IV hypersensitivity reactions have been reported).[4] Type III hypersensitivity reactions may be associated with immune mediated glomerulonephritis.
- Traumatic lesions due to self-mutilation can occur which vary in intensity from mild (uncommon) to severe. Secondary lesions including excoriations, erosions, crusts, lichenification and hyperpigmentation may follow the pruritus. Inappetence and weight loss can occur in severe and chronic infestations. Superficial secondary bacterial infections may develop, including pyoderma and *Malassezia* spp. dermatitis.[3,4]
- Norwegian (or crusted) scabies, where large mite populations are present causing thick crusts on the face, lateral elbows and other parts of the body, is a rare severe form of sarcoptic mange that can occur in animals with concurrent disease or immunosuppression.[4]

DIAGNOSIS

- Definitive diagnosis of canine sarcoptic mange is based on finding *Sarcoptes* mites in a skin scraping sample (sampling needs to be deep enough to examine the full thickness of the epidermis). A positive pinnal-pedal scratch reflex (rubbing of an ear margin triggers the ipsilateral hind leg to elicit a scratching reflex) is present in 75-90% of cases and is suggestive of sarcoptic mange in dogs with compatible clinical signs.[4,9] If no mites are visualised but lesions are strongly suggestive of sarcoptic mange, diagnosis can be based on a positive response to treatment with an effective acaricide.[3]

TRANSMISSION

- Human infestation with *Sarcoptes scabiei* var. *canis* is via direct contact with infested animals or indirectly via contact with contaminated environments or fomites such as infested bedding.
- Prolonged skin-to-skin contact between an infested animal and humans is a major source of transmission, with human lesions usually found in areas of direct contact. The transmission rate of *Sarcoptes scabiei* var. *canis* from dogs to humans is estimated at 10 to 50%.[10]

IN HUMANS

PREVALENCE AND RISK FACTORS

- Human scabies is mostly associated with the host adapted variety *S. scabiei* var. *hominis;* however, zoonotic disease caused by *S. scabiei* var. *canis* has been reported. A study in northern Australian communities where canine and human scabies are co-endemic demonstrated canine-derived and human-derived *S. scabiei* populations are genetically distinct, however this finding has subsequently been questioned.[11,12] The role of dogs in the transmission of scabies in these communities remains unresolved.
- Human scabies is more common in school-aged children, Indigenous communities and residential aged care facilities.

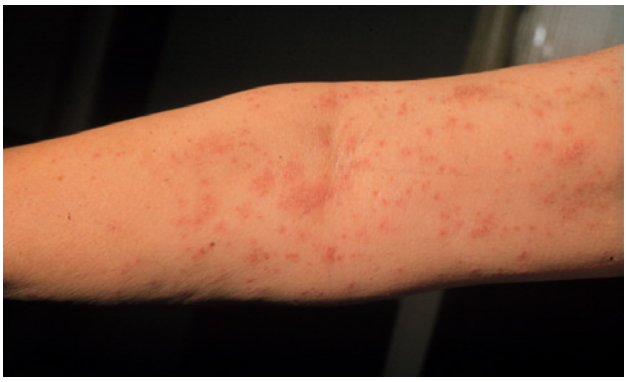
CLINICAL DISEASE

• Human infestations with *S. scabiei* var. *canis* are usually self-limiting. It generally manifests in areas of contact associated with the affected dog, such as the forearms, thighs, chest and abdomen.[13] Lesions associated with *S. scabiei* var. *canis* are more limited in extent and duration than that of *S. scabiei* var. *hominis*.

• The rash and irritation associated with scabies in humans due to canine associated *Sarcoptes* mites shows features of both type I (immediate) and type IV (delayed) hypersensitivity reactions.

• Human scabies induced by the canine mite can result in a highly pruritic papulovesicular rash that can last for several weeks, but which typically resolves spontaneously (whereas human scabies can last several years without treatment). Human infestation with the canine strain of *Sarcoptes* can be evident within 24-96 hours of contact with an affected pet.[13] In contrast, the incubation period of *S. scabiei* var *hominis* following initial exposure is 3-6 weeks, as clinical signs are in part due to a hypersensitivity reaction to the mite.[14] With subsequent exposure the incubation period may be as short as 1-3 days.

• In some cases, hyperinfestation of mites can occur due to an inadequate immune response (e.g. immunosuppression, including advanced HIV) and/or inadequate ability to react or seek treatment (e.g. in patients with dementia). Known as 'crusted scabies', this condition presents as hyperkeratotic dermatosis, often with deep skin fissures, and is highly contagious due to a significantly higher mite burden.[15] Crusted scabies requires longer courses of systemic therapy such as ivermectin, rather than topical preparations (permethrin cream). Crusted scabies due to *Sarcoptes scabiei* var. *canis* has been reported very rarely in the literature.[16] It is usual practice to treat all significant contacts.



Cutaneous lesions due to the transmission of *Sarcoptes scabiei* from a dog to its owner

• Diagnosis in humans can be made by microscopy of skin scrapings. When the mite is not detected on microscopy, response to treatment can be considered diagnostic.

KEY CONSIDERATIONS

1. Human scabies is mostly associated with the host adapted variety *S. scabiei* var. *hominis* after direct human-to-human transmission, however zoonotic infestation with *S. scabiei* var. *canis* from dogs may occur.

2. Human infestation with *S. scabiei* var. *canis* is typically self-limiting and the lesions of a more limited extent and duration compared to infestation with the human host-adapted variety.

3. Owners of infested pets should seek medical advice if they develop any cutaneous lesions.

An all year-round parasite control program in dogs with a parasiticide registered to treat and control mites and other ectoparasites is recommended, particularly in areas with a high prevalence of sarcoptic mange.

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STRONGYLOIDIASIS *(Strongyloides stercoralis)*

- The genus *Strongyloides* contains over fifty species of gastrointestinal parasites capable of infesting a range of animal hosts including dogs and cats, with two species known to infect humans.[1-3]
- *Strongyloides stercoralis,* the major causative agent of strongyloidiasis, is an intestinal threadlike nematode that is endemic to the northern two-thirds of Australia.[3] Globally it is estimated more than 600 million people are infected with *S. stercoralis*.[4]
- Although *S. stercoralis* is primarily a parasite of humans, dogs may be infested and be a source of zoonotic infection.

ACAZAP RECOMMENDATIONS

- In *S. stercoralis* endemic regions it is essential to minimise exposure by:
  - Wearing gloves if handling potentially contaminated soil.
  - Wearing shoes in areas known or likely to be contaminated.
  - Practicing good hand hygiene.
- Prompt removal of faeces is important to avoid contamination of the soil.
- Infection in animal shelters represents a zoonotic risk for staff and visitors. Thorough cleaning involving regular removal of faeces and mechanical cleaning of cages, floors and impervious surfaces with disinfection is likely to decrease the parasitic burden in shelters. Animals with diarrhoea should be isolated.
- In at-risk human and canine patient groups, it is recommended individuals are screened for *Strongyloides* infection prior to starting immunosuppressive medication (such as corticosteroids or chemotherapy) or in those with haematological malignancy.
- Owners of dogs diagnosed with *S. stercoralis*, particularly pet owners on immunosuppressive medication, should be screened for *Strongyloides* infection and seek advice from a human healthcare professional.

IN ANIMALS

AETIOLOGY AND EPIDEMIOLOGY

- *Strongyloides stercoralis* is primarily considered a parasite of humans, however natural patent infestations may occur in non-human primates and canids (domestic and wild).[1] Cats may be infested experimentally with *S. stercoralis* but it is not known to be a normal feline parasite.[1,2]
- Dogs carry genetically distinct haplotypic clades of *S. stercoralis*, one shared with humans, the other exclusively found in dogs, suggesting that dogs are a possible reservoir for zoonotic *Strongyloides* infection.[5]
- *Strongyloides* spp. have complex and unique developmental phases with two distinct life cycles; a free-living (heterogenic) cycle and a parasitic cycle completed within the host. Only the female worms are parasitic, reproducing asexually in the small intestine.
- Larvae passed in faeces undergo rapid development to moult to either filariform larvae or free-living adults. After mating, non-parasitic females reproduce in the environment to generate second-generation infective filariform (L3) larvae. Second generation filariform larvae more commonly penetrate the skin of a new host, however they may also infest the host via ingestion.[6] Transmammary transmission can also occur. The prepatent period following percutaneous penetration of *S. stercoralis* is 5-21 days.[1]
- *Strongyloides stercoralis* can complete its life cycle without leaving the host, leading to chronic, life-long infections if left untreated. Larvae undergo hepato-pulmonary and tracheal migration to develop into adults in the intestines and produce larvae that re-infect the host percutaneously via the skin or colonic mucosa (a process known as autoinfection). This can occur in neonatal and immunocompromised dogs, and lead to hyperinfection (excessive parasitic burden in the gastrointestinal tract and lungs) and more seriously, disseminated infection

(widespread larval migration outside of the gastrointestinal tract and lungs, e.g. to liver, brain, heart, and urinary tract).[6] Hyperinfection can occur in any host, whilst disseminated disease predominantly occurs in immunocompromised populations, often leading to an accompanying fatal septicaemia.[7,8]

PREVALENCE AND RISK FACTORS

- Infestation is more frequent in warm and humid areas, although infestation can occur in temperate climates. Poor sanitation and damp areas such as heavily soiled cages are highly favourable for harbouring *S. stercoralis* larvae and the parasite may become a chronic problem in kennel environments.[9,10]
- Young animals are more susceptible to *S. stercoralis* infestation, particularly puppies.[9]
- In a 2020 Australian study, the prevalence of *Strongyloides* spp. in environmental canine faecal samples (collected from communities across the Northern Territory, central Australia, northern areas of Western Australia and the north-west of South Australia) was 21.9%.[11]

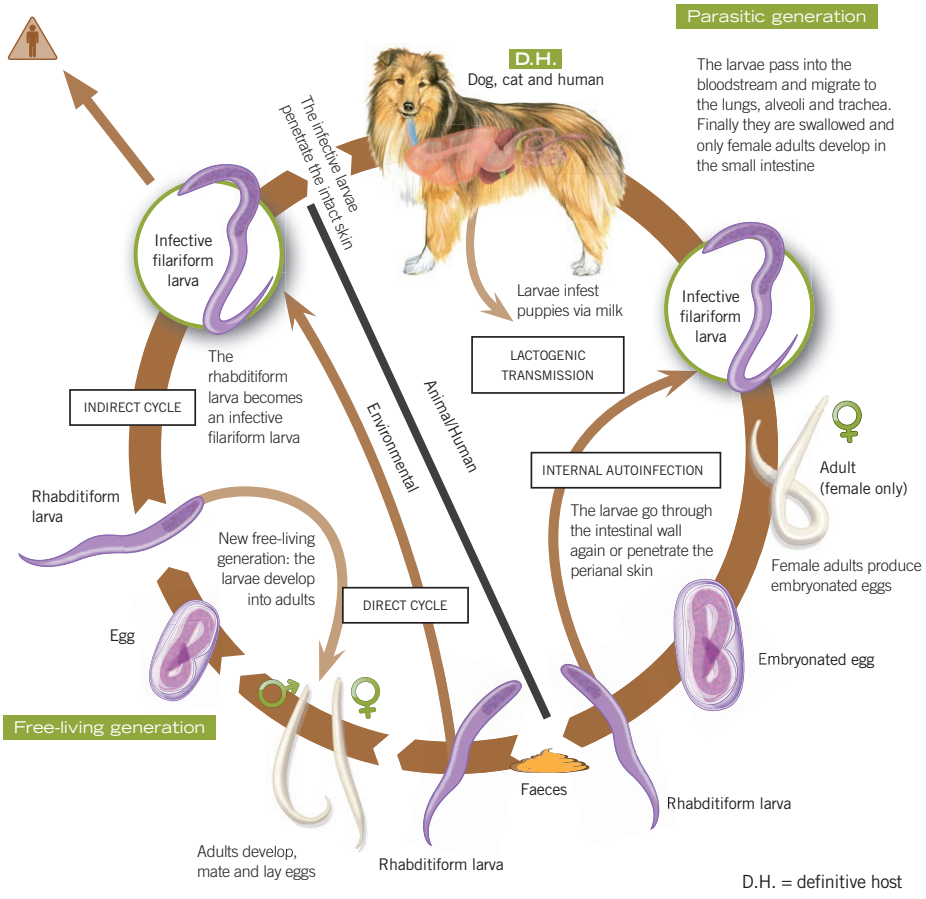
CLINICAL DISEASE

- In immunocompetent animals, the infestation is mostly asymptomatic. Immunity develops within the first 8-12 weeks of life resulting in a cessation of larval shedding.[12]
- *Strongyloides stercoralis* lives in the mucosa of the small intestine in dogs. With high worm burdens, severe enteritis accompanied by abdominal pain, diarrhoea, anaemia and signs of wasting can occur. Migrating larvae may result in damage to the lungs and other tissues with respiratory signs such as pneumonia.[6,12] Percutaneous penetration of larvae may cause pododermatitis.[13]

DIAGNOSIS

- Prevalence is likely underestimated as standard faecal flotations have low sensitivity for the detection of *S. stercoralis*. Generally, faecal examination for *Strongyloides* spp. can be difficult.[14] The Baermann method is recommended for the isolation and identification of *Strongyloides* larvae in fresh faeces.[13]
- For refrigerated, frozen or ethanol-fixed faecal samples, PCR is the test of choice, where available.
- In areas endemic for strongyloidiasis, it is advised that dogs be screened for *S. stercoralis* prior to commencement of corticosteroids or immunosuppressive drugs.
- No products are registered in Australia for the treatment of *Strongyloides* spp. in dogs and cats. Ivermectin at 200 µg/kg once daily for 2 days has been reported to be effective in the treatment of *S. stercoralis* in dogs.[15]

Life cycle of *Strongyloides stercoralis*



Life cycle from Beugnet, F., et al (2018) Textbook of Clinical Parasitology in Dogs and Cat. Grupo Asis Biomedica, S.L.; Adapted from Carithers, D., et al (2012) Pet Owner Educational Atlas.

TRANSMISSION

- Strongyloidiasis is transmitted by infective filariform larvae penetrating human skin, usually following contact with moist soil contaminated with faecal matter.[16]

IN HUMANS

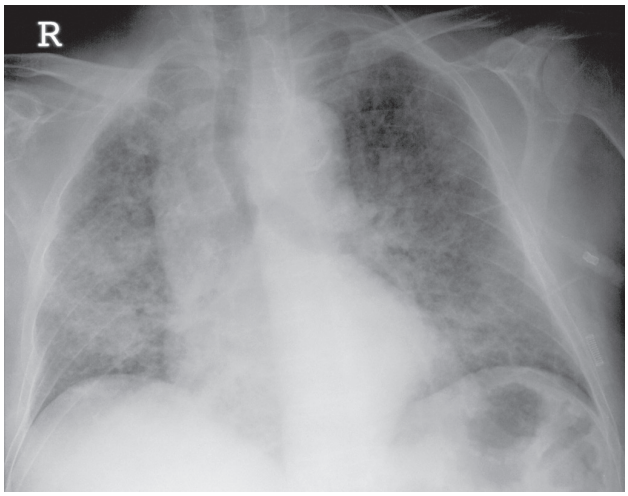
PREVALENCE AND RISK FACTORS

- Strongyloidiasis occurs after larval penetration of intact skin in contact with contaminated soil, and is considered primarily a disease of tropical and subtropical areas.[17] Infection may however occur in any location where poor sanitation or other risk factors are present that enable transmission through environmental faecal contamination.[17,18]
- It is estimated that 600 million people globally are infected with *Strongyloides*.[4] Estimates of strongyloidiasis prevalence within endemic areas in Australia vary widely dependent on diagnostic methodology, study population and seasonality, with reported prevalence rates based on faecal larval detection ranging from 1% (for the majority, living in temperate and urban settings) to 41% (in certain high risk groups).[3] *Strongyloides stercoralis* prevalence has been demonstrated as high as 60% in remote Aboriginal and Torres Strait Islander communities in northern Australia.[3] Children are documented to have a higher prevalence than adults.[3] In other parts of the world prevalence increases with age, as it is a cumulative life-long infection.
- In Australia, strongyloidiasis is most commonly identified in those living in or travelling to Aboriginal communities, immigrants from endemic settings (including tropical and Mediterranean countries), refugees, war veterans (World War II veterans serving in the Asia-Pacific and Vietnam War) and returning travellers from endemic areas.[19]

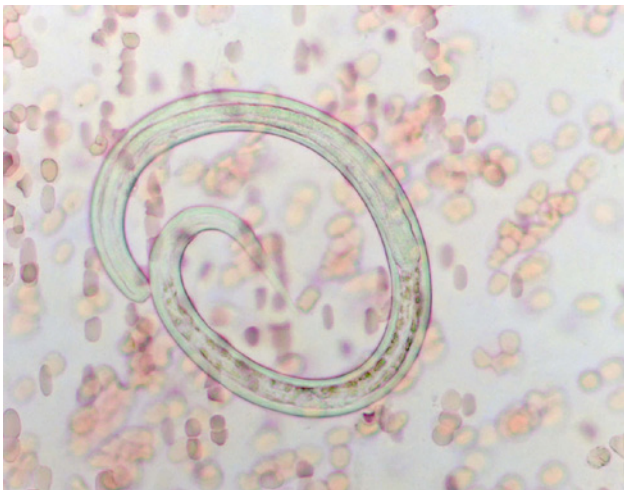
CLINICAL DISEASE

- *Strongyloides stercoralis* may persist indefinitely in the absence of exogenous infection via the process of autoinfection. Eggs laid by female worms in the small intestine hatch to produce rhabditiform larvae which are excreted in the faeces. Some larvae transform in the large intestine into infective filariform (L3) larvae which then penetrate the gut mucosa or perianal skin to undergo pulmonary and tracheal migration and re-develop as adults in the intestines.[18] Hence patients can retain replicating *Strongyloides* in the gastrointestinal tract for decades after initial exposure. Patients remain largely asymptomatic, unless hyperinfection or disseminated infection is induced, usually after initiation of immunosuppression as part of a disease process or treatment.
- Acute and chronic manifestations of strongyloidiasis are recognised:
  - In acute strongyloidiasis, a local reaction can occur almost immediately at the site of larval entry. Clinical presentation is related to the path of larval migration from the site of infection. Pulmonary symptoms (cough, tracheal irritation) may occur within a week, and gastrointestinal signs (diarrhoea, constipation, anorexia, abdominal pain) can occur as early as three weeks after infection.[17,18]

    Larval migration through the bowel wall can carry faecal flora into the bloodstream or peritoneal cavity. Hence the first recognition of strongyloidiasis is sometimes presentation with acute or recurrent gram-negative sepsis.
  - Chronic strongyloidiasis is often asymptomatic. Symptomatic patients may have intermittent gastrointestinal manifestations such as diarrhoea, constipation and vomiting. Dermatological conditions such as pruritus, urticaria, angioedema and larva currens (a cutaneous eruption that causes a pruritic, serpiginous or linear rash along the lower trunk, thighs and buttocks resulting from migrating larvae through the subcutaneous tissues) are described. Peripheral eosinophilia is frequently noted, even in asymptomatic patients.[18]
- The majority of zoonotic *Strongyloides* infections are asymptomatic and self-limiting, however immunosuppression may be associated with accelerated autoinfection and a subsequent hyperinfective (or disseminated) syndrome which is often fatal. Risk factors for infection include patients with HIV/AIDS, alcoholism, patients with diarrhoea and malignancy.[16] Children are noted as a higher risk group. Individuals with impaired cell mediated immunity (such as transplant patients, patients receiving corticosteroids or immunosuppressants) are also at risk.[8]
- Transmission of *S. stercoralis* infection has also been suggested by transplantation of organs where only the donors had a historical exposure and the recipient subsequently developed disease.[18]
- Faecal microscopy will rarely detect the presence of *Strongyloides* unless accompanied by culture concentration methods (traditionally 'Harada culture', Baermann's technique



Chest radiograph demonstrating typical findings of pulmonary strongyloidiasis. Note ground-glass opacity



Rhabditiform larva of *S. stercoralis*

or Koga Agar plate culture utilising a lawn of *E. coli*). PCR testing is also increasingly used, but serology is the mainstay of diagnosis.

- Immunocompetent patients with chronic strongyloidiasis may demonstrate persistent eosinophilia, however eosinophilia may disappear during hyperinfection, and hence can be an unreliable marker of severe disease. Because of the risk of disseminated infection, serological screening for *Strongyloides* is recommended prior to immunosuppressive treatment, particularly in patients who had lived in 'high risk' settings. Serology is also used to demonstrate falling titres, and ensure cure following ivermectin therapy.

KEY CONSIDERATIONS

1. Strongyloidiasis is considered a neglected tropical disease, endemic to parts of tropical and subtropical Australia.
2. Screening of patients (human and animal) in known endemic areas prior to initiating immunosuppressive therapy is recommended due to the risk of disseminated infection.
3. Due to the ability of *S. stercoralis* to cause autoinfection, human patients can have a life-long infection unless provided with effective treatment. Infected humans may be a source of infection for dogs.

Minimise contact with potentially contaminated soil through appropriate clothing, footwear, protective equipment and good hand hygiene.

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TOXOCARIASIS *(Toxocara canis, T. cati)*

- Roundworms infesting dogs and cats are an important and common helminth zoonosis globally.
- Human toxocariasis is a parasitic infection caused by the migrating larvae of *Toxocara* species and is associated with a range of clinical syndromes, with children noted to be more commonly affected.

ACAZAP RECOMMENDATIONS

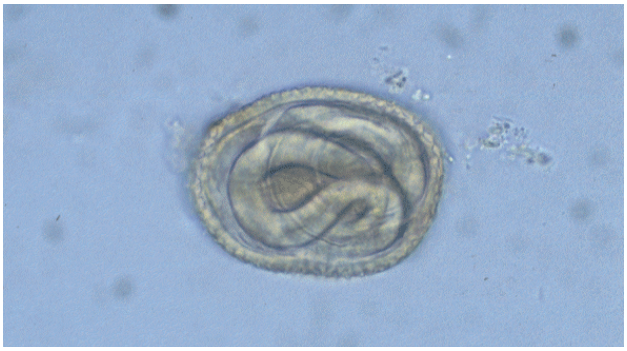
- In general, puppies and kittens should be dewormed fortnightly until eight weeks of age, preferably with a product with activity against adult and immature worms.[1]
- Given that the prepatent period of *Toxocara canis* is approximately five weeks (following ingestion of eggs) and *Toxocara cati* five to eight weeks, monthly deworming of dogs and cats is recommended to reduce environmental contamination and minimise zoonotic risk.
- Adult dogs and cats, depending on health and lifestyle factors, should have a faecal flotation performed yearly, with puppies and kittens tested more frequently than adult animals.
- Do not feed raw meat or allow dogs and cats to hunt as many animals, birds and molluscs act as paratenic hosts for *Toxocara* spp.
- Close supervision of children is essential to minimise risk of oral exposure to contaminated material (e.g. soil, sand).
- Prompt removal of faeces on a daily basis is recommended. Dog owners should remove faeces from public areas.
- Ensure good hygiene practices are followed, including washing hands after handling pets, playing outdoors, and prior to eating.
- Cover sandpits and protect playgrounds and garden areas. Do not allow children to play in areas contaminated with animal faeces.
- Juvenile animals have the highest prevalence of patent infestation. Higher risk individuals may consider adopting older animals or should take particular care if adopting a young animal.

Sandpits should be covered when not in use to minimise contamination with animal faeces

- Veterinarians should educate dog owners regarding the potential risks of improper parasite control in dogs.
- Albendazole and mebendazole are frequently used anthelmintics in humans, effective for treating adult nematodes located within the gastrointestinal tract. As *T. canis* and *T. cati* do not develop past the larval stage in humans, the routine use of albendazole or mebendazole will have no effect on preventing or managing toxocariasis in humans and is therefore not recommended for this purpose.

AETIOLOGY AND EPIDEMIOLOGY

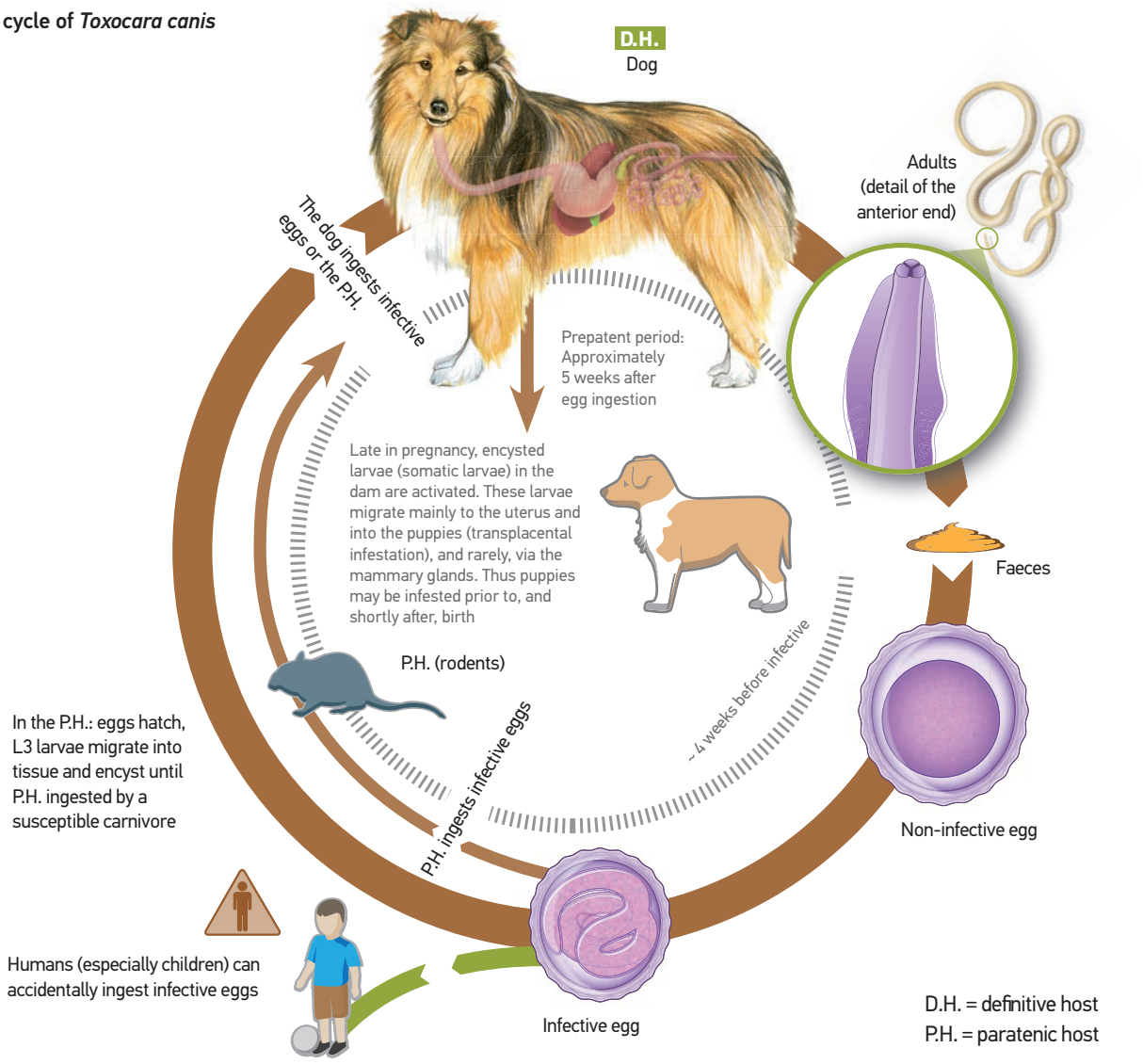
- Three species of roundworm are known to infest dogs and cats in Australia: *T. canis* (canine only), *T. cati* (feline only) and *Toxascaris leonina* (canine and feline). *Toxascaris leonina* is not a known zoonosis. In addition to domestic dogs, *T. canis* occurs widely in wildlife (foxes and dingoes) in Australia.[2]
- Companion animals can become infested through ingestion of embryonated eggs, consumption of paratenic hosts (such as snails, birds and rodents) or via transmammary transmission.[1] Transplacental transmission of larvae from the bitch to pups *in-utero* is also an important route of transmission for *T. canis.*
- When infective eggs are ingested by adult dogs, roundworms commonly undergo arrested development. Larvae travel through the intestinal wall, undergo hepatopulmonary migration and are distributed throughout the body, including the liver, lungs, muscles and other organs, without completing their life cycle. Somatically arrested *T. canis* can reactivate in pregnant bitches and be transmitted to pups.



Embryonated (infectious) *Toxocara canis* egg

- Lactogenic transmission of *T. cati* only occurs after acute infection of the queen during late pregnancy.[3] Unlike *T. canis,* transmammary infection of kittens following reactivation of arrested somatic larvae in chronically infected queens does not play a strategic role in the life cycle of *T. cati.*
- In younger dogs, tracheal, as opposed to hepatopulmonary migration predominates, so that most worms are coughed

Life cycle of *Toxocara canis*



Life cycle from Beugnet, F., et al (2018) Textbook of Clinical Parasitology in Dogs and Cat. Grupo Asis Biomedica, S.L.; Adapted from Carithers, D., et al (2012) Pet Owner Educational Atlas.

and swallowed and develop as egg-producing adults in the gut. Therefore, clinical disease caused by *T. canis* infestation typically affects young dogs less than one year of age. Patent egg-shedding infestations in older dogs are uncommon, due to a degree of acquired immunity.

PREVALENCE AND RISK FACTORS

- Companion animal roundworms are distributed worldwide. The prevalence of *T. canis* in Australia has been reported as 1.2% in domestic pet dogs and 29.4% in Aboriginal community and wild dogs.[4,5] The prevalence of *T. cati* in domestic cats in Australia has been reported as 3.2%.[5]

CLINICAL DISEASE

- Heavily infested puppies, and to a lesser extent kittens, can have a classic 'pot-bellied' appearance, with clinical signs including ill thrift, abdominal discomfort, anorexia, diarrhoea and vomiting. Young animals may have significant worm burdens due to their limited resilience to infestation, leading to biliary obstruction, intussusception and/or intestinal obstruction.
- Heavy infestations in neonatal pups may result in pneumonia and acute death.
- Female roundworms are highly fecund, laying up to 200,000 eggs per day.[6] The eggs are environmentally resistant and can survive a range of temperatures.[7]
- The prepatent period of *T. canis* varies depending on how the larvae are acquired. For transplacental or lactogenic infections, adults develop to patency within three or four weeks respectively, compared to approximately five weeks after ingestion of embryonated eggs from the environment.[8] For *T. cati* the prepatent period varies between approximately five to eight weeks regardless of route of transmission.[3,9] This is the primary reason kittens are less susceptible to clinical effects of *T. cati* infection than pups are to *T. canis*.

DIAGNOSIS

- Faecal tests for specific parasite antigens combined with centrifugal faecal flotation has been shown to assist in the diagnosis of infestation. In standard faecal flotations, detection of thick shelled pitted eggs enables identification to the genus level. The absence of eggs in samples does not rule out infestation.

TRANSMISSION

- Human infection occurs through accidental ingestion of eggs from contaminated water, food or soil or via consumption of undercooked viscera, snails and meat sources containing infective larvae.[10]
- Infective, sticky-coated eggs from the environment may also contaminate the fur of animals and pose a direct source of infection to humans in close physical contact.[11]
- *Toxocara* eggs are highly resistant to chemical disinfectants and can survive for years in the environment. Eggs possess a sticky outer coating making their removal from surfaces difficult.

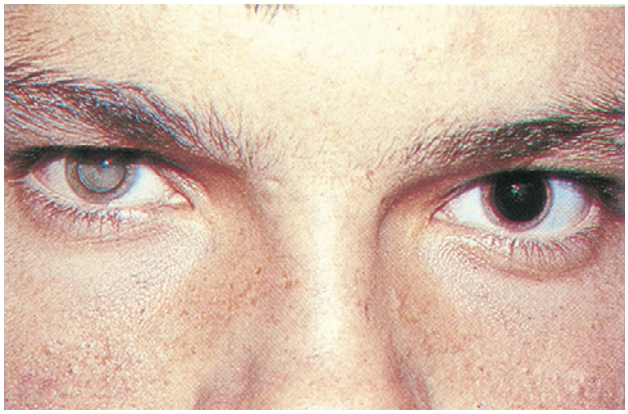
IN HUMANS

PREVALENCE AND RISK FACTORS

- A meta-analysis of published data on the seroprevalence of toxocariasis worldwide estimated that 7.0% of Australians are seropositive for *Toxocara* antibodies, indicating current or prior infection.[12]
- The primary risks for *Toxocara* infection in humans is puppy ownership, pica behaviours and low socioeconomic status given the major source of infection is via the ingestion of embryonated eggs from the environment. Young children, pet owners and individuals in regular contact with companion animals are also at increased risk of exposure.
- Zoonotic roundworms cannot complete their life cycle in humans, however developing larvae can migrate through the body and cause disease.

CLINICAL DISEASE

- Most people infected with *Toxocara* have asymptomatic infections, however a number of clinical syndromes are recognised, associated with the host's immune response to the migrating larvae and the larval burden.
- The clinical syndromes of toxocariasis in humans include visceral larva migrans (VLM), ocular larva migrans (OLM),



Unilateral leukocoria caused by toxocariasis
(© American Academy of Ophthalmology)

covert or "common" toxocariasis, and neurotoxocariasis (NLM).[13]

- Visceral larva migrans (VLM) is considered the most common syndrome and is predominantly documented in young children, but considered rare in practice. Most cases are asymptomatic or subclinical. Clinical signs associated with VLM pertain to the organs involved and may include coughing, wheezing, abdominal pain, hepatomegaly and myalgia.[13]
- Ocular larva migrans (OLM) is usually unilateral in presentation and most commonly reported in children.[13] Cases occur every year in Australia and are considered rare, estimated at one case per 1.6 million head of population annually.[2] Clinical findings include strabismus, unilateral diminished vision, leukocoria, photophobia and ocular granulomas. Blindness can occur. The extent of visual impairment depends on the larval burden. It is possible that children with OLM have a repeated small-dose inoculation of larvae over a long period of time that escape the host's immune defence mechanisms to lodge in the retinal vessels.[13-15]
- Covert toxocariasis in children or common toxocariasis in adults is typically non-specific and describes patients that demonstrate positive *Toxocara* serology linked to a number of systemic and local symptoms. Fever, anorexia, headache, wheezing, nausea, abdominal pain, vomiting and lethargy may be seen.[13,16]
- Neurotoxocariasis or neural larva migrans (NLM) is considered rare and predominantly occurs in middle-aged individuals. Migration of larvae through the central nervous system results in clinical signs such as meningitis, encephalitis, cerebral vasculitis, or myelitis, usually associated with other symptoms such as fever or headache.[13,17,18]
- Systematic reviews have identified associations between *Toxocara* seropositivity and asthma in children and epilepsy, although further studies are needed.[19,20] A cross-sectional study from the United States demonstrated reduced cognitive function in children seropositive for *Toxocara* independent of other known confounding factors (e.g. socioeconomic status), however due to the nature of the study, a causal association cannot be confirmed.[21]

KEY CONSIDERATIONS

1. Veterinarians should educate dog owners regarding the potential risks of improper parasite control in dogs.
2. As the prepatent period of *T. canis* is approximately five weeks and *T. cati* five to eight weeks, monthly deworming of dogs and cats is recommended.
3. Daily removal of pet faeces and covering sandpits when not in use is essential to reduce environmental contamination.

Monthly deworming of adult dogs and cats is recommended to reduce environmental contamination and minimise zoonotic risk.

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TOXOPLASMOSIS *(Toxoplasma gondii)*

- Toxoplasmosis is a zoonotic disease caused by the obligate intracellular protozoan parasite *Toxoplasma gondii.*
- *Toxoplasma gondii* can infect multiple warm-blooded animal species including companion animals, livestock, birds and wildlife, with cats being the definitive host.
- Most cases of toxoplasmosis in humans are thought to occur indirectly through ingestion of uncooked or undercooked meat or contaminated food, however direct contact with cat faeces is a potential risk.

ACAZAP RECOMMENDATIONS

- Preventing infection in cats requires minimising opportunities for exposure. It is therefore recommended to keep cats indoors to prevent hunting and scavenging, and to avoid feeding raw meat and poultry.
- Gloves should be worn when handling cat litter trays. Daily emptying of litter trays and prompt removal of faeces from the environment is recommended to prevent oocysts from sporulating. After being passed in faeces, oocysts can sporulate (become infective) in one to five days and can survive in the environment up to 1.5 years in optimal cool and humid conditions.
- Children's sandpits should be protected or covered when not in use and gloves worn when gardening to prevent exposure.
- Good hand hygiene is essential after contact with raw meat, soil and sand. This is especially true when handling raw meat and poultry. Prevent these foods and their juices from contacting already cooked or ready-to-eat foods and fresh produce.
- To reduce the risk of humans acquiring *T. gondii*, proper food preparation is essential. Meat should be cooked to an internal temperature of 67°C or higher to inactivate tissue cysts.[1] Freezing meat at -10°C for 3 days or -20°C for 2 days is considered sufficient to inactivate tissue cysts or bradyzoites.[2] Thorough washing of fruit and raw vegetables prior to consumption is also of benefit.
- Due to the increased risk associated with infection in pregnant women and immunosuppressed individuals, additional precautions for these higher risk groups include:
  - Avoid direct contact with soil, cat litter or areas contaminated with cat faeces.
  - When considering adopting a new cat, consider healthy adult cats rather than young kittens, as recently weaned kittens and feral/stray cats are potentially a greater source of infection.

IN ANIMALS

AETIOLOGY AND EPIDEMIOLOGY

- *Felidae*, inclusive of domestic cats, are the definitive hosts of *T. gondii*, while most other mammalian and avian species can be intermediate hosts.
- *Toxoplasma gondii* has a complex life cycle with three infectious stages:
  - Sporozoites – contained in oocysts shed in the faeces of the definitive host. The enteroepithelial life cycle results in the shedding of oocysts and occurs only in cats.
  - Tachyzoites – actively multiplying stage of the parasite in extra-intestinal tissues. Occurs in definitive and intermediate hosts.
  - Bradyzoites – latent or slowly multiplying stages, encapsulated within extra-intestinal tissue (central nervous system, muscle and viscera), also referred to as 'tissue cysts', are responsible for life-long chronic infection. Occurs in definitive and intermediate hosts.
- Cats and dogs can become infected by ingesting intermediate hosts (e.g. rodents and birds) or raw meat and poultry harbouring tissue cysts, or by consuming sporulated oocysts in soil or contaminated food and water. Sporulated oocysts are resistant to environmental conditions and many routine disinfectants. Less commonly, transplacental or transmammary transfer of tachyzoites from mother to offspring can occur in pets.[3]

PREVALENCE AND RISK FACTORS

- Pooled global meta-analysis from 1967-2017 estimated 52% of cats in Australia as seropositive to *T. gondii*.[4] A 2020 Australian study reported 39% seroprevalence of *T. gondii* in owned domestic cats in Australia.[5]
- Feeding companion animals raw meat diets (kangaroo, lamb and other pasture-consuming species) is considered a major risk factor for *T. gondii* infections. It is reported that up to 59% of cats in Australia are fed raw meat diets.[5]
- The prepatent period in cats depends on the stage of *T. gondii* ingested (3-10 days after ingestion of bradyzoites and 18 days or more after ingestion of tachyzoites).[6]
- Only about 1% of the feline population are found to be shedding oocysts at any given time.[7] Duration of shedding is relatively short (1-3 weeks) and cats that have previously shed *T. gondii* typically do not re-shed oocysts unless re-infected or immunocompromised.[3,7]

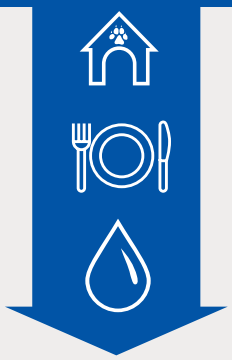
CLINICAL DISEASE

- The majority of infections with *T. gondii* are asymptomatic in cats. Clinical disease is considered rare and more commonly seen in immunocompromised cats and young kittens. Cats with iatrogenically induced immunosuppression, feline infectious peritonitis, feline immunodeficiency virus and feline leukaemia virus can be predisposed to systemic toxoplasmosis, with immunosuppression leading to recrudescence of latent infection.[8,9]
- Non-specific clinical signs and multi-system infection characterise the clinical presentation in non-immune adult cats. Pneumonia is the predominant sign of generalised toxoplasmosis in cats. Acute respiratory distress syndrome and septic shock may also occur. In cats with severe neurological or respiratory signs, toxoplasmosis can be fatal. Common sites of involvement include the central nervous system, musculature, lungs and eyes.[3,9]
- It is rare for dogs to present with clinical toxoplasmosis in the absence of underlying immunosuppressive disease.[3] Clinical signs may relate to hepatic, pulmonary, ocular or neurological involvement, resulting in fever, cough, jaundice, seizures and cranial nerve deficits. Screening dogs with neurological signs for *T. gondii* infection is recommended.[3]
- If infection occurs for the first time in immuno-naive pregnant cats, or if pregnant cats have a reactivation of latent infection (for example due to immunosuppression), tachyzoites can cross the placenta to infect the foetus. Clinical toxoplasmosis in transplacentally infected kittens can vary in severity depending on the stage of gestation, and may include foetal death and abortion in early pregnancy or the birth of stillborn or deformed kittens with infection later in pregnancy. Live born congenitally infected kittens, or those infected lactogenically, frequently die of pulmonary or hepatic disease.[3,9] Congenital infection is rarely reported in pups.

DIAGNOSIS

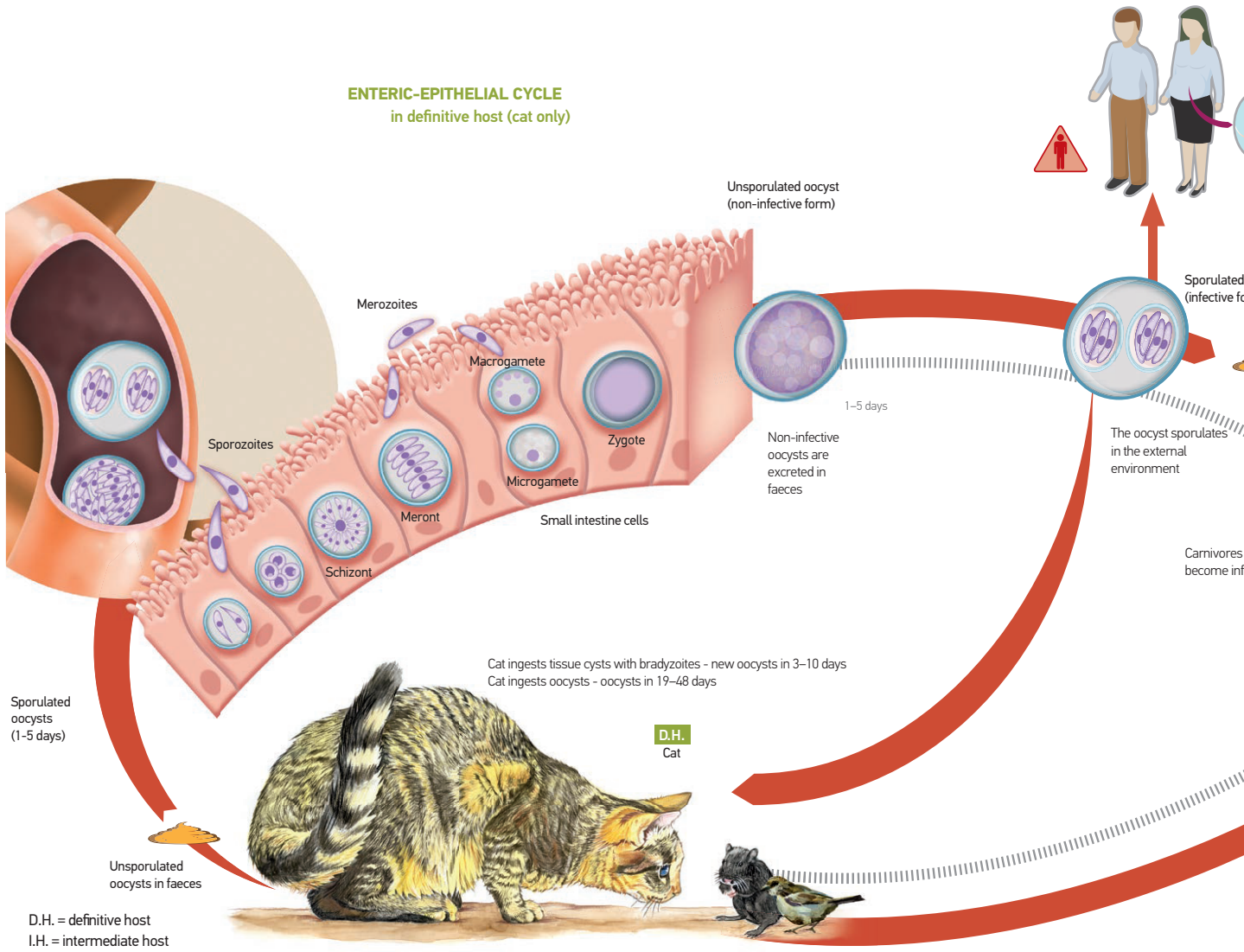
- As active shedding only occurs for 1-3 weeks after initial exposure, oocysts are rarely found in cat faeces via faecal flotation. Serological testing can determine if a cat is positive (exposed, asymptomatic or clinical) or negative (susceptible to infection). Cases of severe clinical systemic toxoplasmosis may be diagnosed by:
  - High-to extreme IgM anti-*T. gondii* antibody titres.
  - Rising paired IgG anti-*T. gondii* antibody titres.
  - Detecting presence of the organism's DNA in body fluids (cerebrospinal fluid, aqueous humour or bronchoalveolar lavage fluid) or tissue via PCR.[8,9]
- In older dogs that are iatrogenically or naturally immunosuppressed, concurrent testing and measurement of anti-*Neospora caninum* antibody titres is strongly recommended as clinical signs may be indistinguishable from those of toxoplasmosis.

TRANSMISSION



- The most common route of transmission to humans is through consumption of tissue cysts in raw or undercooked meat contaminated with *T. gondii.*
- Human infection can occur through accidental ingestion of sporulated oocysts shed in the faeces of cats, for example when gardening or consuming vegetables, fruit or water contaminated with oocysts.

Life cycle of *Toxoplasma gondii*

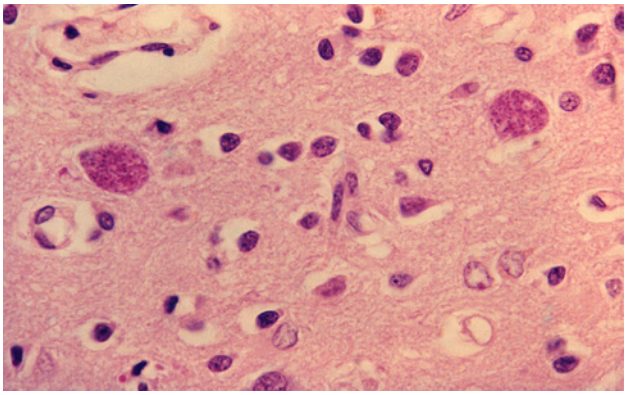


Life cycle from Beugnet, F., et al (2018) Textbook of Clinical Parasitology in Dogs and Cat. Grupo Asis Biomedica, S.L.; Adapted from Carithers, D., et al (2012) Pet Owner Educational Atlas.

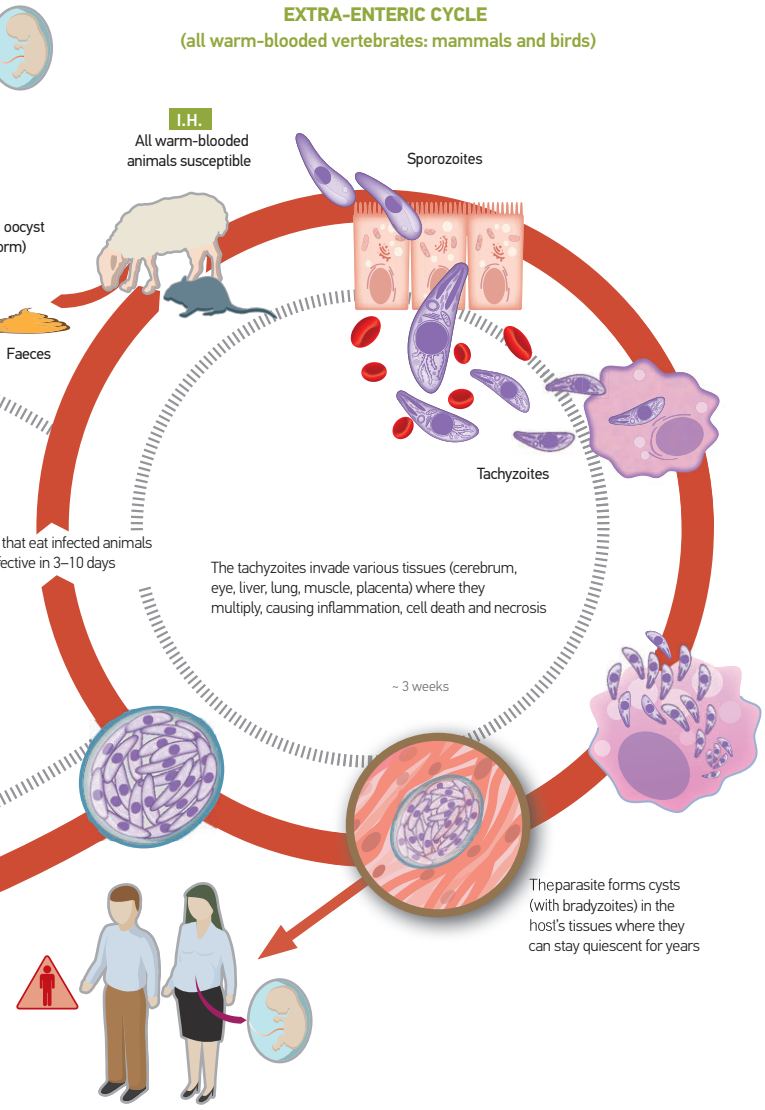
IN HUMANS

PREVALENCE AND RISK FACTORS

- It is estimated globally that approximately 25-30% of the population is infected by *Toxoplasma*, however significant geographical variation is seen, relating to climatic factors (higher prevalence in cool, humid conditions), anthropogenic factors (dietary habits) and socioeconomic factors (higher prevalence in resource-poor communities).[10]
- Whilst primary infection during pregnancy is considered rare in Australia, infection in the Australian population is relatively common, with most studies reporting a seroprevalence of between 20 and 40%.[11,12] A recent pilot study in Busselton, Western Australia, reported a seroprevalence of 66%,[13] which, if representative of the country as a whole, would be counter to the situation in many other developed countries in which *Toxoplasma* infection has declined over time.[10]



Photomicrograph of brain tissue sample from a patient with neurotoxoplasmosis. Several cysts containing *Toxoplasma gondii* bradyzoites are visible in the image
(Public Health Image Library, CDC)



- A study from Western Australia reported that seropositivity increased with age, reflecting the accumulated risk of a lifetime of exposure.[13] Interestingly in this study, owning a cat was not associated with an increased risk of infection.

CLINICAL DISEASE

- Primary infection in pregnant women may lead to congenital toxoplasmosis. The overall rate of vertical transmission following primary infection during pregnancy has been reported as approximately 30%.[14,15] The risk of vertical transmission increases with gestational age, from 6% at 13 weeks to 72% at 36 weeks.[15]
- Congenital infection may also occur in immunocompromised (e.g. HIV-positive) pregnant women with reactivated latent infection.
- Congenital toxoplasmosis may result in spontaneous abortion or stillbirth. A wide spectrum of clinical signs have been reported in congenitally infected children, including developmental delays, impaired hearing, ocular and neurological abnormalities.[16-18] The severity of clinical disease in congenital toxoplasmosis is inversely related to the stage of gestation at which infection occurs, with the greatest impact seen following infection in the first trimester.[18]

Pregnant owners should be counselled on appropriate and proportional risk mitigation strategies to minimise the risk of congenital toxoplasmosis

- Most postnatal infections in immunocompetent patients are asymptomatic or subclinical, though some patients may present with a glandular-fever like syndrome (fever, fatigue, muscle pain, a sore throat and headache) with lymphadenopathy and mononucleosis.[18] Severe clinical manifestations are rarely seen in immunocompetent patients. Although the clinical course of toxoplasmosis is usually benign, symptoms may take weeks to months to resolve.
- Immunocompromised individuals (including HIV/AIDS patients and transplant recipients) are susceptible to severe disease following primary infection. Systemic involvement can include pneumonitis, myocarditis and hepatitis in these patients.
- A significant burden of disease related to toxoplasmosis is due to reactivation of latent infections, most commonly in immunocompromised HIV-positive patients.[19]
- In HIV-infected patients with a low CD4+ T cell count, toxoplasmosis can cause opportunistic infection, presenting either as a severe systemic primary infection or more commonly an end-organ infection when associated with reactivation of tissue cysts. Affected individuals most commonly present with cerebral toxoplasmosis (toxoplasmic encephalitis) with the presence of characteristic multiple ring-enhancing intracerebral mass lesions involving the cortex and basal ganglia on CT or MRI imaging.[19] Extracerebral localisation following reactivation is less common.
- Toxoplasmosis is an unusual but potentially serious complication following organ transplantation. Patients undergoing cardiac transplantation are susceptible to myocardial reactivation of *T. gondii* as seropositive organ donors can transmit *Toxoplasma* cysts in transplanted muscle tissue.[20]

IN HUMANS *continued*

- Latent infections have been linked to a wide spectrum of human neurological and psychiatric disorders, with correlations reported for *Toxoplasma*-seropositivity and conditions including schizophrenia, bipolar disorder, addiction disorders, Alzheimer's disease, and epilepsy.[21-23] Additional research is required to determine if such associations are causal.
- Serological testing is used for the diagnosis of primary and past infection and relies on IgM and IgG testing respectively. Confirmation of a diagnosis of relapsing infection in the immunocompromised patient requires molecular testing (e.g. cerebrospinal fluid PCR if neurological signs are present) or the detection of *Toxoplasma* cysts in a tissue biopsy.
- Antenatal screening is not typically recommended in Australia, but serology is performed by some practitioners routinely upon request or based on clinical signs suggestive of acute toxoplasmosis.

KEY CONSIDERATIONS

1. Most cases of toxoplasmosis in humans are thought to occur indirectly through ingestion of uncooked/undercooked meat or contaminated food, however direct contact with cat faeces is a potential risk.
2. Although cats remain infected for life, shedding is typically of short duration (1-3 weeks) at the time of infection only, unless the cat is re-infected or immunocompromised. Shed oocysts are not immediately infectious, taking one to five days to sporulate. Prompt removal (at least daily) of cat faeces from the environment is recommended.
3. Good hand hygiene is essential after contact with raw meat, soil and sand. This is especially true when handling raw meat and poultry.

Due to the increased risk associated with infection in pregnant women and immunosuppressed individuals, additional precautions should be taken in these groups.

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ANIMALS IN CARE FACILITIES

CONSIDERATIONS FOR DOGS AND CATS IN HEALTH CARE, AGED CARE, OR CHILDCARE SETTINGS

Mirroring the positive role that pets play in broader society, visitation by animals has been associated with a range of positive mental and physical health outcomes in the health care and aged care sectors. In the school or daycare setting, animal associated activities may be for general education on interacting with pets, be part of a structured educational intervention, such as dog-assisted reading programs, or as support and therapy dogs to assist children with special emotional or behavioural needs.

Despite the benefits, visitation of animals in these settings is not without risk. Visiting animals may carry zoonotic pathogens, serve as a reservoir of resistant organisms, or act as mechanical vectors for the transmission of human pathogens between patients, residents and staff.

Careful selection and proper care of animals, as well as the implementation of appropriate controls within the facility, can greatly minimise the risk involved in such visits.

The following general guidelines are recommended for companion animal (dog and cat) associated activities in the health care, aged care, or childcare setting to minimise the risks of zoonotic disease.

ACAZAP RECOMMENDATIONS

ANIMAL SELECTION AND CARE

- Animals involved in visitation programs should undergo regular veterinary examination (at least every 12 months). Veterinary examination should specifically include an assessment of the probability of carriage and transmission of zoonotic diseases and the capacity for mitigation of risk in these animals.
- Dogs and cats should be at least 12 months of age to minimise the risk of carrying zoonotic pathogens more frequently associated with young animals (e.g. *Toxocara, Giardia*, *Salmonella*, *Campylobacter*).
- Visiting animals should have the appropriate temperament and training to interact in a safe and calm manner. Dogs should be assessed and accredited through professional organisations such as Delta Therapy Dogs.
- Pregnant, nursing, or immunocompromised animals and animals in oestrus should not take part in visitation programs.
- Animals with illness or injury must be assessed by a veterinarian for their suitability to participate in visitation programs.
- Animals with a known or suspected communicable disease should not take part in programs until cleared by a veterinarian. Similarly, animals with vomiting, diarrhoea, urinary incontinence, or skin lesions should be appropriately investigated and cleared prior to taking part in a program. Such clearance should specifically consider the risk of zoonotic diseases.
- Dogs and cats should receive monthly deworming and year-round ectoparasite control (effective against fleas and mites), and be appropriately vaccinated.
- Faecal parasite testing should be performed at least once a year. Other routine screening for potentially zoonotic pathogens IS NOT recommended.
- Animals should not be fed raw foods of animal origin (including raw meat, unpasteurised milk etc.) to minimise the risk of carrying zoonotic pathogens.
- Animals should be bathed/groomed before and after visitation. Nails should be kept short.

DURING VISITATION

- Hand hygiene is essential. All persons having contact with animals should wash their hands before and after handling animals.
- Animals should be prevented from licking people's faces and open wounds. All open wounds should be covered during visits.
- If animals are placed on bedding, a disposable, impermeable barrier should be used.
- All areas visited should be appropriately cleaned and disinfected in accordance with local infection control and disinfection guidelines.
- Appropriate records of the visit should be kept to enable contact tracing if required.
- Visitation should generally not take place in intensive or neonatal care facilities or food/medication preparation areas.

NON-TRADITIONAL PETS IN THE CHILDCARE SETTING

Although the focus of these guidelines is zoonotic diseases that may be transmitted from dogs and cats, a brief discussion of exotic, or non-traditional pets, in the childcare setting warrants specific comment due to the increased risk of disease associated with exposure to such animals in children less than 5 years of age.

Due to their increased risk for zoonotic disease transmission, the following animals are not recommended for childcare visitation programs:

- Reptiles and amphibians (salmonellosis, campylobacteriosis).
- Poultry, including hatchlings and associated hatchery equipment (salmonellosis, campylobacteriosis).
- Preweaned calves (cryptosporidiosis).

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