AIDAP | AUSTRALASIAN INFECTIOUS DISEASES ADVISORY PANEL

ANTIBIOTIC PRESCRIBING

DETAILED GUIDELINES

2ND EDITION

AIDAP | AUSTRALASIAN INFECTIOUS DISEASES ADVISORY PANEL

Dr Steve Holloway

BVSc (Syd), MVS, PhD (Melb), DACVIM, MACVSc

Steve graduated from the University of Sydney in 1983 and after several years in practice, journeyed along the pathway to becoming a specialist in internal medicine. In 1991 Steve became a Diplomate of the American College of Internal Medicine. In 1994 Steve commenced his PhD studying herpes viruses of horses at the University of Melbourne, completing in early 1998. Between 1999 and 2009 Steve lectured in infectious diseases of small animals at the Faculty of Veterinary Science at the University of Melbourne. Steve is currently a specialist in internal medicine in private practice and regularly consults on infectious disease problems in small animal patients.

Dr Darren Trott

BSc (Hon), BVMS (Hon), PhD

Darren is a veterinarian with 30 years of experience in bacterial disease research focused on zoonotic infections, enteric diseases, gastrointestinal microbial ecology, and antibiotic resistance. In 2010, after 10 years of teaching and microbe-focussed research at the University of Queensland, Darren accepted a position in the new School of Animal and Veterinary Sciences at The University of Adelaide where his research areas have expanded to include antimicrobial resistance ecology, repurposing existing drug classes for development as new anti-infectives, including pre-clinical assessment in murine bioluminescent models of infection, and the effect of antimicrobials on the gut microbiome. In 2016 he established the Australian Centre for Antimicrobial Resistance Ecology to work closely with Australia's major animal industries (both livestock and companion animal) in the areas of prudent antimicrobial use, antimicrobial stewardship and confirming/ensuring that rates of resistance in animal pathogens, zoonotic and commensal bacteria remain low by international standards.

Dr Mike Shipstone

BVSc, FACVSc, DACVD

Dr Shipstone graduated from Queensland University in 1984 and has worked in multiple private practice and industry positions. In 1995 he started a residency at the Animal Skin and Allergy Clinic in Melbourne, with additional periods of study at the University of California, Davis and Louisiana State University, Baton Rouge. Mike is Principal and Director of a specialist dermatology referral practice and adjunct Associate Professor at the University of Queensland. Mike is a Fellow of the Australian and New Zealand College of Veterinary Scientists (Veterinary Dermatology) and a Diplomate of the American College of Veterinary Dermatology. Mike has published in Australia and overseas and has presented in Australia, South East Asia, and North America.

Professor Vanessa Barrs

BVSc (hons), MVetClinStud, FACVSc (Feline Medicine), GradCertEd (Higher Ed)

Vanessa Barrs is a registered specialist in feline medicine and a Chair Professor of Companion Animal Health and Disease at City University of Hong Kong's Jockey Club College of Veterinary Medicine and Life Sciences, partnered with Cornell College of Veterinary Medicine. Before moving to Hong Kong in 2019, Vanessa was Head of Small Animal Medicine at The University of Sydney for 15 years, and Director of their Veterinary Teaching Hospital. She is a past president of the Feline Chapter of the Australian and New Zealand College of Veterinary Scientists and the International Society of Companion Animal Infectious Diseases. Prof. Barrs is well known for her infectious diseases research of companion animals. She discovered *Aspergillus felis,* which can cause severe disease in animals and humans. Vanessa is passionate about improving animal health and welfare and is the proud owner of two second-hand cats, Joey, and Albert.

Dr Richard Malik

DVSc, DipVetAn, MVetClinStud, PhD, FACVSc, FASM

Richard Malik graduated from the University of Sydney, trained in Anaesthesia and Intensive Care, and then moved to ANU where he completed a PhD in pharmacology at the John Curtin School of Medical Research. He then completed a Postdoctoral fellowship at the Neurobiology Research Centre before returning to his alma mater where he remained for 16 years in a variety of positions (1995 to 2002). Since 2003 Richard has worked as a consultant for the Centre of Veterinary Education and finds time to see cases in practices in the Eastern suburbs of Sydney. Richard has varied research interests, most notably infectious diseases, genetic diseases, and diseases of cats in general. He is a Fellow of the Australian Society of Microbiology, a member of the Australian Society of Infectious Diseases and an Adjunct Professor of Veterinary Medicine at Charles Sturt University.

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Dr Mandy Burrows

BSc, BVMS (Murd), MACVSc, FACVSc (Dermatology)

Mandy is a Fellow of the Australian and New Zealand College of Veterinary Scientists in Veterinary Dermatology and a registered specialist in veterinary dermatology. She is a Director of Animal Dermatology Clinic, Perth, and an Associate Professor in Small Animal Medicine (Dermatology) at Murdoch University, Western Australia. She lectures in dermatology and endocrinology at Murdoch University, and she teaches postgraduate veterinary students at Massey University, New Zealand. She is a past-President of the Council of the Australian and New Zealand College of Veterinary Scientists and Chair of the Science Week Committee, the President of World Congress in Veterinary Dermatology, and the current Australian and New Zealand representative of the World Association for Veterinary Dermatology. She has extensive experience with clinical dermatology in companion animals and she enjoys teaching dermatology to veterinary undergraduate and postgraduate students.

Professor Jacqueline Norris

BVSc, MVSt, PhD, FASM, MASID, Grad Cert Higher Ed. RCVS (Veterinary Microbiology)

Jacqui is a Professor of Veterinary Microbiology and Infectious Diseases at the Sydney School of Veterinary Science, at the University of Sydney. She is passionate about practical research projects and education programs for veterinary professionals, animal breeders and animal owners. For example, she is one of three designers and founding members of the AMR Veterinary Collective (www.amrvetcollective.com) which is centralising resources and training related to antimicrobial resistance and stewardship in veterinary science. Her main research areas include: 1) Epidemiology, diagnosis and treatment of companion animal viral diseases including feline coronaviruses; 2) Prevention of Q fever and role of companion animals and wildlife in its epidemiology; 3) Multidrug resistant (MDR) bacterial infections especially *Staphylococcus* species in all animals including humans; 4) Knowledge, attitudes and barriers to veterinary antimicrobial stewardship; and 5) Aetiology and diagnosis of kidney disease in domestic and zoo felids.

Dr Anthony Caiafa

BVSc BDSc MANZCVS

Anthony graduated from the University of Melbourne with a Bachelor of Veterinary Science degree in 1978. He worked in companion animal practice for 17 years and obtained his memberships by examination from Australian and New Zealand College of Veterinary Scientists in Small Animal Surgery (1992) and Veterinary Dentistry (1993). He then returned to full time studies in 1995, to graduate with a Bachelor of Dental Science, dux of the year, in 1998, from the University of Melbourne. Anthony currently works as a dentist on the Sunshine Coast, Queensland as well as in Veterinary Dental referral practice at North Coast Veterinary Specialists and Referral Centre, also on the Sunshine Coast. He lectures to the undergraduate Veterinary Science students at James Cook University, Townsville; as well as lecturing and running workshops on all aspects of Veterinary Dentistry around the world. He is also a chapter author in two respected Veterinary Dental textbooks and has published several peer reviewed journal articles.

Dr Wendy Baltzer

DVM, PhD, DACVS, DACVSMR, CCRP

Wendy graduated from UC Davis School of Veterinary Medicine in 1994 and completed a PhD in veterinary physiology and a residency in small animal surgery at Texas A&M University in 2005. She is a Diplomate of the American College of Veterinary Surgeons and of the American College of Veterinary Sports Medicine and Rehabilitation and a registered specialist in New South Wales. She was an Associate Professor at Oregon State University College of Veterinary Medicine until 2016 and a Professor at Massey University School of Veterinary Science in New Zealand before joining the faculty at the University of Sydney in 2020.

Wendy's research interests include oxidant stress and injury in sporting dogs, bone healing using omental grafting, tendon and ligament injury, osteoarthritis, lumbosacral disease, and geriatric rehabilitation. She is currently working on research involving osteoarthritis, lower back pain in dogs, bandaging techniques and omentum grafting for bone healing.

Wendy's role at the School of Veterinary Science at the University of Sydney includes surgical teaching of veterinary students, interns and residents, clinical small animal surgery practice at the veterinary teaching hospital, and research in surgery, sports medicine, and rehabilitation.

FOREWORD

Antimicrobials are essential to modern medicine for treating a range of infections in people and maintaining the health and welfare of our animals. The inappropriate and/or unrestrained use of antimicrobials occurring globally in human and animal health is exacerbating the rate of development of antimicrobial resistant microorganisms. This pervasive global threat means that antimicrobials are becoming less effective over time leading to treatment complications and failures, and increased healthcare costs for people and animals.

The veterinary profession has been paramount in combatting antimicrobial resistance. They partnered with industry and government to successfully advance antimicrobial stewardship efforts under *Australia's First National Antimicrobial Resistance Strategy 2015-19*. They have an ongoing, critical role in how we minimise antimicrobial resistance through the implementation of our latest strategy, *Australia's National Antimicrobial Resistance Strategy – 2020 and Beyond* (the Strategy).

Resistance to antimicrobials occurs naturally in microorganisms, but amplification occurs with poor antimicrobial selection and management. The AIDAP's antimicrobial prescribing guidelines directly address Objective 4 of the Strategy. In particular, the Priority Area for Action 4.1 requests our stakeholders to "ensure that coordinated, evidence-based antimicrobial prescribing guidelines and best-practice supports are developed and made easily available and encourage their use by prescribers".

These revised guidelines capture the latest scientific evidence on antimicrobial use in companion animals, so that they remain fit-for-purpose and assist in reducing the risk of antimicrobial resistance. It is a complete guide that not only covers antimicrobial indications but also assists with recommended treatments. The guide also encourages veterinarians to think about when to use antimicrobials, to avoid unnecessary use in the first instance.

I commend the work of all involved in the development and ongoing patronage of these guidelines and urge every veterinarian to regularly consider this advice. In doing so, you will help safeguard the ongoing, long-term efficacy of antimicrobials, while ensuring the health and welfare of animals under your care.

Dr Mark Schipp

Chief Veterinary Officer (Australia)

OIE Delegate (Australia)

AIDAP

RESISTANCE TIPS

Antimicrobial resistance is a critical problem in human medicine around the world, both in hospitals and in the wider community. It is emerging as a problem in veterinary medicine, especially in the USA. Although the situation in Australia is currently much better than in North America, multi-resistant *E. coli* and some methicillin-resistant *Staphs* have appeared in Australian small animal practices over the last 10 years. Although detailed discussion and analysis of this problem is currently beyond the scope of AIDAP, the group thought some **pertinent practical tips** would be a good step towards improved antimicrobial stewardship, which is the best way to prevent the emergence of a more widespread resistance problem.

1. **Choose antimicrobials based on the most likely pathogen(s) that are associated with particular infectious disease settings.** (Such as *E. coli* from lower urinary tract infections or *Staphylococcus pseudintermedius* from canine pyoderma). Published susceptibility profiles for a pathogen should be used to make an informed decision as to the antibiotic to be selected. In situations where it is not possible to accurately predict the likely pathogens and/or their antibiograms, then culture and susceptibility (C&S) testing should be performed as soon as practical. Where finances preclude this, an in-practice cytological examination using slides stained with Diff-Quik® or Gram stain can be very informative. If a dose range is provided, aim to prescribe as close to the maximum recommended dose on the label as possible.

2. **If empiric antibiotic therapy has failed, then C&S testing is advised.** If finances preclude this, choose another class of antimicrobial likely to be effective against the putative pathogen.

3. **Avoid empiric use of fluoroquinolones in general, and especially** for treating chronic *Staphylococcus* spp. infections in dogs or acute sporadic UTI. Amoxicillin or Trimethoprim-Sulphonamides are superior choices to fluoroquinolones for empiric therapy of UTIs.

4. **Avoid using combination therapy** unless there is clearly a life-threatening infection present and/or an unpredictable antibiotic susceptibility of the pathogen(s) involved. For example, life-threatening sepsis in a dog that has peritonitis from a ruptured bowel is an indication for 4-quadrant antibiotic therapy until the results of culture are known and de-escalation of drugs can occur.

6. **Ensure the length of treatment with antibiotics is appropriate.** Duration of therapy should only be sufficient to achieve clinical cure. Serious infections generally justify at least two weeks of therapy. Identify where owner or patient compliance is likely to be an issue and take appropriate measures to achieve compliance.

7. **In the hospital setting, be vigilant for infections attributable to an unusual organism,** such as *Serratia* spp., or common pathogens such as *E. coli* with a consistent antibiogram, often with a multi-resistant profile. Such organisms should ideally be forwarded to a suitable reference laboratory (Professor Darren Trott's laboratory at the University of Adelaide or Veterinary Pathology Diagnostic Services at the University of Sydney) for archiving and possibly additional molecular testing. If such case clustering occurs, consider consultation with an infectious diseases expert to identify potential sources of infection (such as foam bedding, or a staff member who is a chronic carrier of *Staphylocccus aureus*).

8. **Develop in-house infection control guidelines for every veterinary hospital.** These should include signs and policies that encourage regular hand washing with alcohol-based hand preparations. See the AIDAP Practical Infection Control Guidelines for further information.

ANTIMICROBIAL RESISTANCE SURVEILLANCE IN COMPANION ANIMAL PATHOGENS

THE NEED FOR ANTIMICROBIAL RESISTANCE SURVEILLANCE

There is broad consensus internationally that surveillance of resistance levels in key bacterial pathogens through antimicrobial susceptibility testing (AST) underpins strategies to address the issue of antimicrobial resistance (AMR), including adoption of prudent use guidelines, antimicrobial stewardship (AMS), and improved infection control. The key reasons for surveillance of AMR are to determine:

1. what is the size of the problem?
2. is it increasing?
3. are previously unknown types of resistance emerging?
4. is a particular type of resistance spreading?
5. is a particular type of resistance is associated with a particular outbreak?
6. is the use of a particular antibiotic class is associated with resistance development?

However, AMR surveillance for resistance phenotypes only provides half the story. Whole bacterial genome sequencing (WGS) of pathogens can tell us much more about their origins, molecular epidemiology, and risk factors for infection, as well as potential for zoonotic transmission or strain sharing between animals and humans.

AMR surveillance of companion animal pathogens is performed on isolates from veterinary diagnostic laboratories (VDL) derived from clinical specimens submitted by veterinarians. These isolates are convenience samples obtained from cases where a C&S test has been requested by the referring clinician. As such, there is potential for selection bias to skew the isolates towards a more resistant phenotype, given that one of the main reasons for requesting C&S is non-responsiveness to empirical antimicrobial therapy. Despite this issue, AMR surveillance of VDL isolates currently represents the most streamlined and cost-effective approach. Site of infection is also an important consideration, as the breakpoints used to classify the isolate as susceptible or resistant may differ depending upon the pharmacokinetics of the antimicrobial agent relative to the infection site, for example, the urinary tract versus skin or soft tissue infection.

WHICH ISOLATES SHOULD WE FOCUS ON?

For companion animals, the bacterial pathogens that are the main drivers of systemic antibiotic use are *Staphylococcus pseudintermedius,* mainly associated with skin, soft tissue, and surgical site infections, and extraintestinal pathogenic *Escherichia coli* (ExPEC), mainly associated with urinary tract infections (UTIs). Methicillin-resistant *S. pseudintermedius* (MRSP) in particular, requires ongoing monitoring due to its propensity to develop multidrug-resistant (resistance to three or more classes of antimicrobial) and extensively drug-resistant (resistance to all but one or two classes) profiles, which includes resistance to all ß-lactam drugs.

S. pseudintermedius is a resident of the skin microbiota of dogs and often causes secondary bacterial infections in animals with primary allergic skin disease. It is increasingly recognised as a cause of otitis externa, surgical site, and urinary tract infections, with nosocomial transmission an important feature. MRSP isolates are identified in the VDL by their resistance to oxacillin. MRSP epidemiology is characterised by widespread dissemination of resistant clones belonging to distinct multi-locus sequence types (STs).[1] Infection of humans with MRSP is rare.[2]

By contrast, highly similar strains of ExPEC may colonise the gastrointestinal tract and cause clinical infections (most commonly UTIs) in both humans and companion animals. These have been associated with multidrug resistance (MDR) including resistance to both third-generation cephalosporins and fluoroquinolones in both humans and companion animals.[3-5]

HOW IS ANTIMICROBIAL SUSCEPTIBILITY TESTING (AST) FOR RESISTANCE MONITORING PERFORMED?

AST results have historically been intended primarily to guide physicians and veterinarians regarding appropriate antimicrobial therapy. Results are generally reported qualitatively as susceptible, intermediate, or resistant after applying relevant clinical breakpoints based on pharmacokinetic and pharmacodynamic principles (see below). For the purposes of surveillance, however, qualitative results achieved using different laboratory methods or applying non-standard breakpoints are of limited value to detect trends or evaluate levels of resistance on a broader level, and almost precludes comparison of the data. In contrast, reporting and retaining quantitative data provides a mechanism to detect shifts over time, facilitate early detection of emerging resistance, as well as allowing clinicians to adjust treatment approaches to achieve better clinical outcomes. This approach supports comparison with surveillance data from other systems and allows data to be re-interpreted.

Minimum inhibitory concentration (MIC) testing is the gold standard technique for determining an isolate's individual susceptibility to an antimicrobial agent. MIC is defined as the lowest concentration inhibiting growth of the organism using a series of two-fold dilutions. Multiple techniques for determining MIC have been developed, including broth microdilution (using 96-well plates), agar dilution, E-test graded strips and automated systems. Broth microdilution is most applicable to AMR surveillance.[6] The Kirby-Bauer disk diffusion (DD) technique is often employed in VDLs for AST on individual clinical isolates from animals, however it is not readily amenable to AMR surveillance.[7,8]

CLINICAL BREAKPOINTS AND EPIDEMIOLOGICAL CUT-OFF VALUES (ECOFFs or ECVs)

The Clinical Laboratory Standards Institute (CLSI) has developed clinical breakpoints for human and veterinary pathogens for both MIC broth microdilution and disk diffusion AST techniques. Clinical breakpoints are determined using a combination of *in vitro* and *in vivo* data to predict the likelihood of clinical cure based on pharmacokinetic-pharmacodynamic (PK-PD) parameters. Breakpoints are agreed upon by CLSI's veterinary antimicrobial susceptibility subcommittee (first formed in 1982) after reviewing all available data. They do not, however, predict the likely presence of resistance mechanisms in isolates, are not available for all antimicrobials and all animal species, and are subject to change as new PK-PD data is obtained. The default is to use human clinical breakpoints if veterinary-specific breakpoints are unavailable. Based on clinical breakpoints, isolates are designated as:

- **'susceptible'** (bacterial infection may be appropriately treated with the dosage regimen recommended for that type of infection and infecting species), or
- **'intermediate'** (bacterial infection may be appropriately treated in body sites where the drugs are physiologically concentrated, or when a higher dosage of drug can be safely used), or
- **'resistant'** (bacteria are not inhibited by the usually achievable concentrations of the agent using normal dosage schedules and/or fall into the range where specific microbial resistance mechanisms are likely and clinical outcome has not been predictable in in vivo based studies).[9]

An isolate may also be described as 'non-susceptible' if its MIC is above the susceptible clinical breakpoint (it is in the intermediate or resistant range).

The European Committee on Antimicrobial Susceptibility Testing (EUCAST) is a standing committee jointly organised in 1997. EUCAST is funded by the European Union. EUCAST first developed the ECOFF (Epidemiological Cut-Off Value) term and publishes ECOFFs for specific antimicrobial and veterinary pathogen combinations based on MIC distributions of large numbers of isolates. ECOFFs classify an organism as 'wild type' or 'non-wild type' based on the normal distribution of MICs for fully susceptible isolates that do not contain any resistance determinants that could influence the MIC phenotype (phenotypically detectable acquired resistance mechanisms). The difference between clinical breakpoints and ECOFFs can best be appreciated in a MIC distribution for a hypothetical antimicrobial as seen in Figure 1.

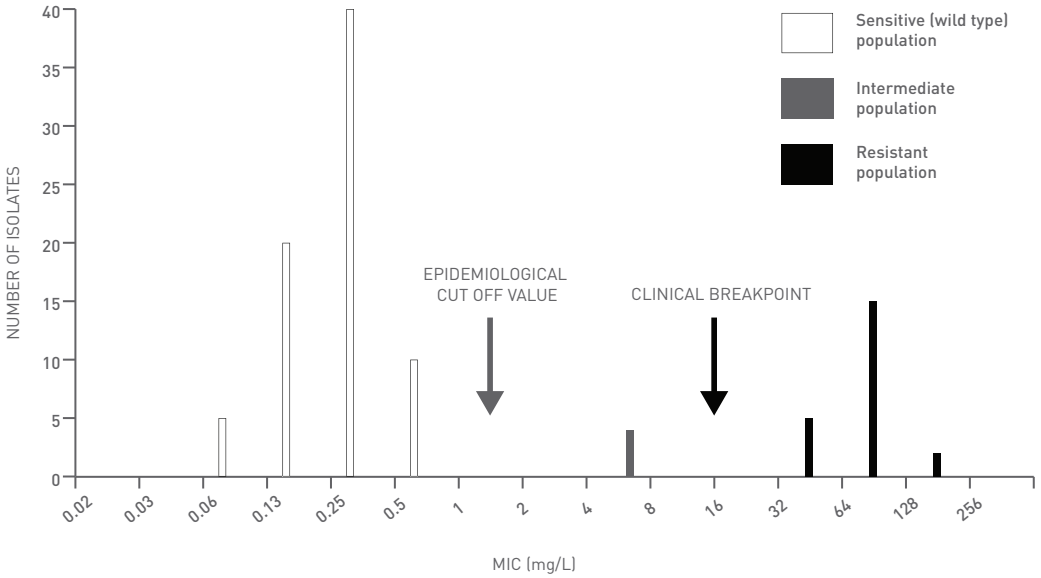

Figure 1: MIC distribution for a hypothetical bacterial species targeted in antimicrobial resistance surveillance programs. Arrows indicate the epidemiological cut-off value (ECOFF) established according to EUCAST recommendations, separating the wild type (no resistance determinants) from the non-wild type (presumed resistance determinants that could be verified by whole genome sequencing analysis), and the clinical breakpoint. Sensitive (Susceptible), Resistant and Intermediate value columns are indicated. Adapted from Simjee *et al* (2018).[10]

In many cases, the ECOFF is often the same value as the susceptible clinical breakpoint.

Difficulty in interpreting veterinary specific AST data arises where recent changes in clinical breakpoints established for veterinary species have shifted the CLSI clinical breakpoint to below the corresponding ECOFF (because of new PK data). This can be seen with current amoxicillin-clavulanate breakpoints for dogs and cats for *E. coli* isolated from skin and soft tissue infections (≥1 µg/mL) and urinary tract infections (≥16 µg/mL) now being below the current ECOFF (≥32 µg/mL), which is also the human clinical breakpoint. Using the veterinary clinical breakpoints, isolates with MICs which do not indicate the presence of resistance mechanisms and are within the wild type distribution could still be classified as resistant, but if the same isolate was obtained from a human clinical sample, it would be regarded as susceptible. The same principle also holds true for cephalexin and amoxicillin. As these assumptions are based on the label dose rate, increasing the dose rate to the upper limit (as is recommended for these antibiotics in certain situations) may compensate.

Zoetis-sponsored national antimicrobial resistance survey

In 2013, through AIDAP, Zoetis sponsored the first national Australian survey of antimicrobial resistance in coagulase-positive *Staphylococcus* spp. and *E. coli* isolated from infections in animals. Over a one-year period, Australian VDLs submitted isolates to a centralised laboratory for AST incorporating both DD and MIC techniques. Further characterisation of isolates by WGS identified major STs, virulence genes and antimicrobial resistance genes in the isolate collection and enabled detailed strain epidemiology and comparison with human isolates. Just over 2500 isolates were obtained, with the majority sourced from infections in companion animals.

Extraintestinal pathogenic E. coli (ExPEC) are part of the autochthonous microbiota of the gut but are also major causes of urinary tract and other opportunistic infections in humans and companion animals, as well as being one of the predominant sepsis-causing agents.[3] Among human ExPEC, increasing prevalence of resistance to critically important antimicrobials (CIAs) such as fluoroquinolones and extended-spectrum cephalosporins has been largely driven by the emergence and global epidemic spread of distinct genetic STs including ST131 and ST1193.[11] Both these STs are highly virulent and often associated with urosepsis in humans.

The Zoetis AMR survey confirmed that ~9-10% of ExPEC isolates from dogs in Australia (mainly derived from UTIs) were resistant to CIAs (Figure 2), and ~3-65.% from cats (Figure 3), a proportion similar to human ExPEC isolates in this country. Additionally, the breakpoint issue associated with amoxicillin, amoxicillin clavulanate and cephalexin mentioned above was apparent with high proportions of isolates resistant to these antimicrobials even though many are unlikely to possess any actual resistance mechanisms (hence the recommendation to use the upper end of the label dose range for these antibiotics for *E. coli* infections in companion animals).

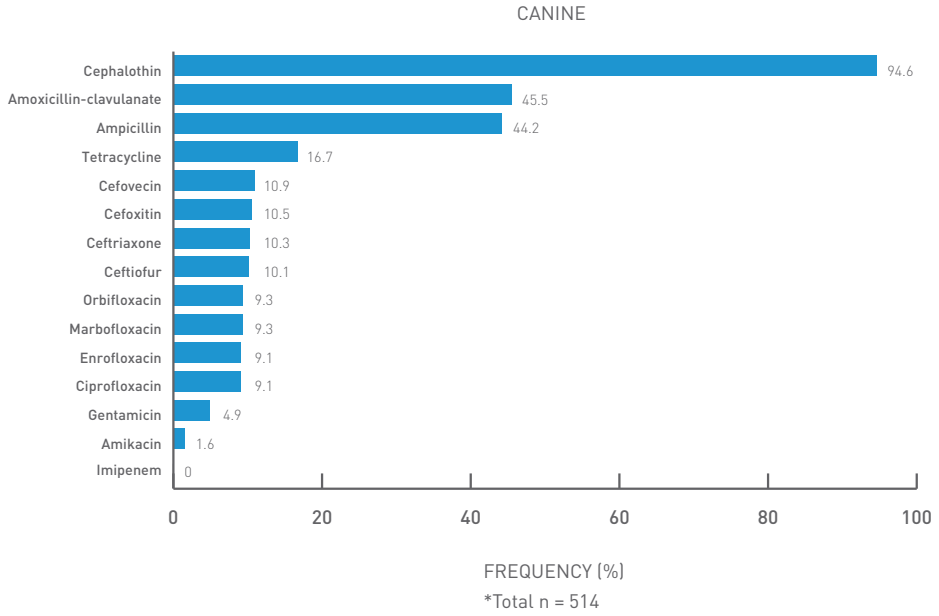

Figure 2: Frequency of resistance to antimicrobials among ExPEC isolates from dogs obtained in the first national AMR survey.

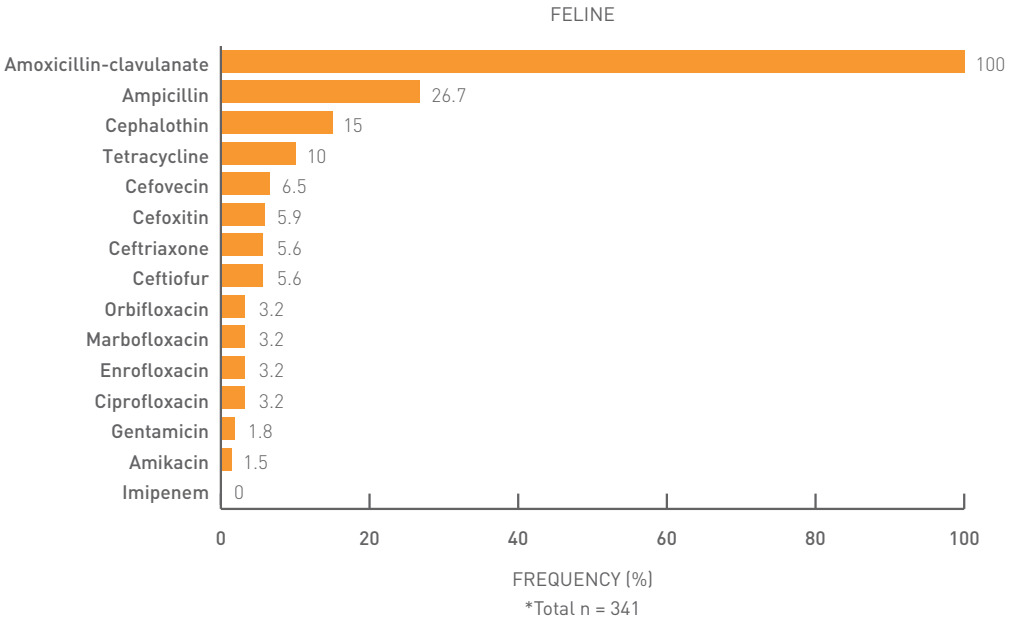

Figure 3: Frequency of resistance to antimicrobials among ExPEC isolates from cats obtained in the first national AMR survey.

ST131 and ST1193 can both cause extraintestinal infections in companion animals and the strains isolated from dogs and cats are closely related to human ST131/ST1193 strains by WGS analysis.[3] However, the Zoetis study confirmed that companion animals are likely spill-over hosts for these clonal lineages rather than natural hosts. This is based on their much lower prevalence as a cause of clinical infections in dogs and cats (~10% of CIA-resistant isolates) which has been relatively constant over time, compared to their much higher and increasing prevalence as a cause of human infections (currently representing ~50% of CIA-resistant isolates).[3,12] Nevertheless, it has been shown that dogs may carry ST131 in their gut,[13] and cyclic transmission has been demonstrated between humans and dogs within the family home.[14] Furthermore, dogs admitted to veterinary hospitals may shed high proportions of CIA-resistant *E. coli* in their faeces, particularly if they are receiving CIA therapy.[13]

Staphylococcus pseudintermedius is the major autochthonous commensal organism present on canine skin but is also an important cause of opportunistic infections in susceptible dogs. These include skin pyodermas and soft tissue infections (SSTIs), otic, urinary tract, and post-surgical infections. The first clinical *S. pseudintermedius* strain to possess the mecA gene (encoding resistance to all β-lactam drugs including methicillin) was identified in 1999, and since then, MRSP has spread epidemically among dogs throughout the globe in a similar way to *E. coli* ST131/ST1193 in humans.[1] MRSP can be described as the veterinary equivalent of hospital- and community-acquired methicillin-resistant *S. aureus* (MRSA). However, MRSP is often resistant to more antibiotic classes, with globally distributed epidemic strains such as ST68 ('North American' clone), ST71 ('European' clone), and ST45 ('SE Asian' clone) exhibiting multiple drug resistance phenotypes.[15] Although immunosuppressed humans may acquire MRSP infection, these are typically rare events.[16] Nevertheless, in localised studies, humans have been shown to be important in the carriage and cyclic transmission of MRSP strains to dogs within veterinary hospitals and in the family home.[2]

The Zoetis AMR survey found that the frequency of MRSP among *S. pseudintermedius* isolates from Australian dogs was approximately 12.7% (Figure 4).

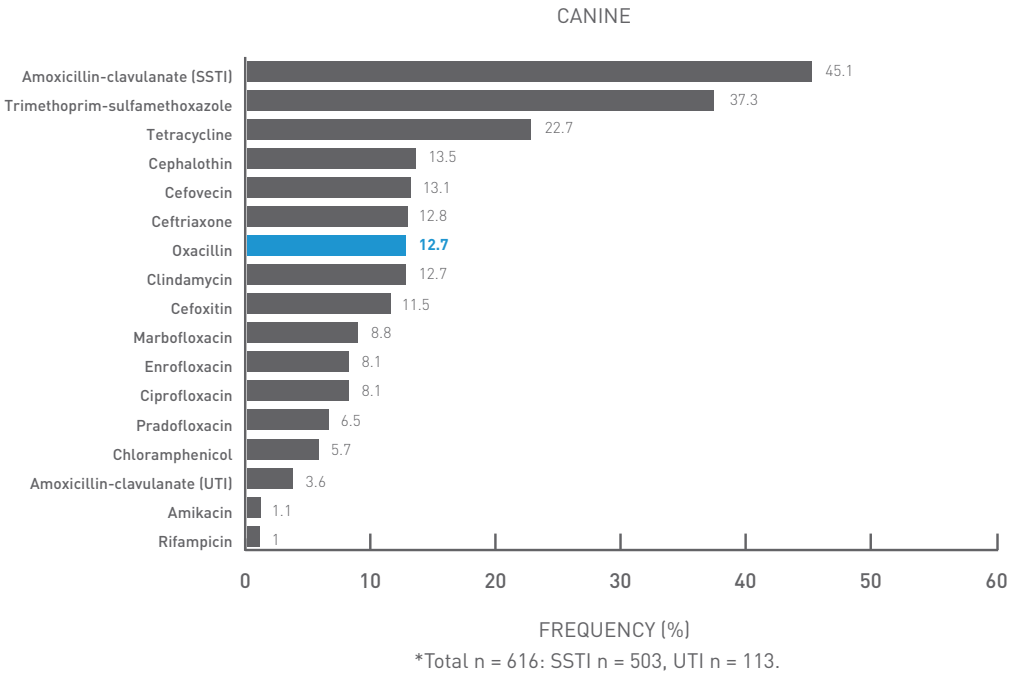

Figure 4: Frequency of resistance to antimicrobials among *Staphylococcus pseudintermedius* isolates from dogs obtained in the first national AMR survey.

Furthermore, WGS analysis has confirmed rapid evolution under antimicrobial selection pressure, wide distribution and the presence of internationally distributed epidemic clones (ST71 and ST45) in Australia[1] (Figure 5). Unique STs exhibiting extensively drug-resistant phenotypes (only susceptible to one or two antibiotic classes) were also identified, including the ST496 'Sydney' clone and ST497 'Melbourne' clone, two STs that were unique to Australia at the time. While cases of MRSP SSTI can sometimes be effectively managed with topical therapies alone, the limited range of systemic treatment options leads to increased use of CIAs normally reserved for human infections. These infections may include post-surgical joint and urinary tract infections suggestive of nosocomial transmission within veterinary hospitals with some cases requiring systemic treatment with CIAs such as linezolid (usually reserved for MRSA infections in humans) for clinical resolution.[17]

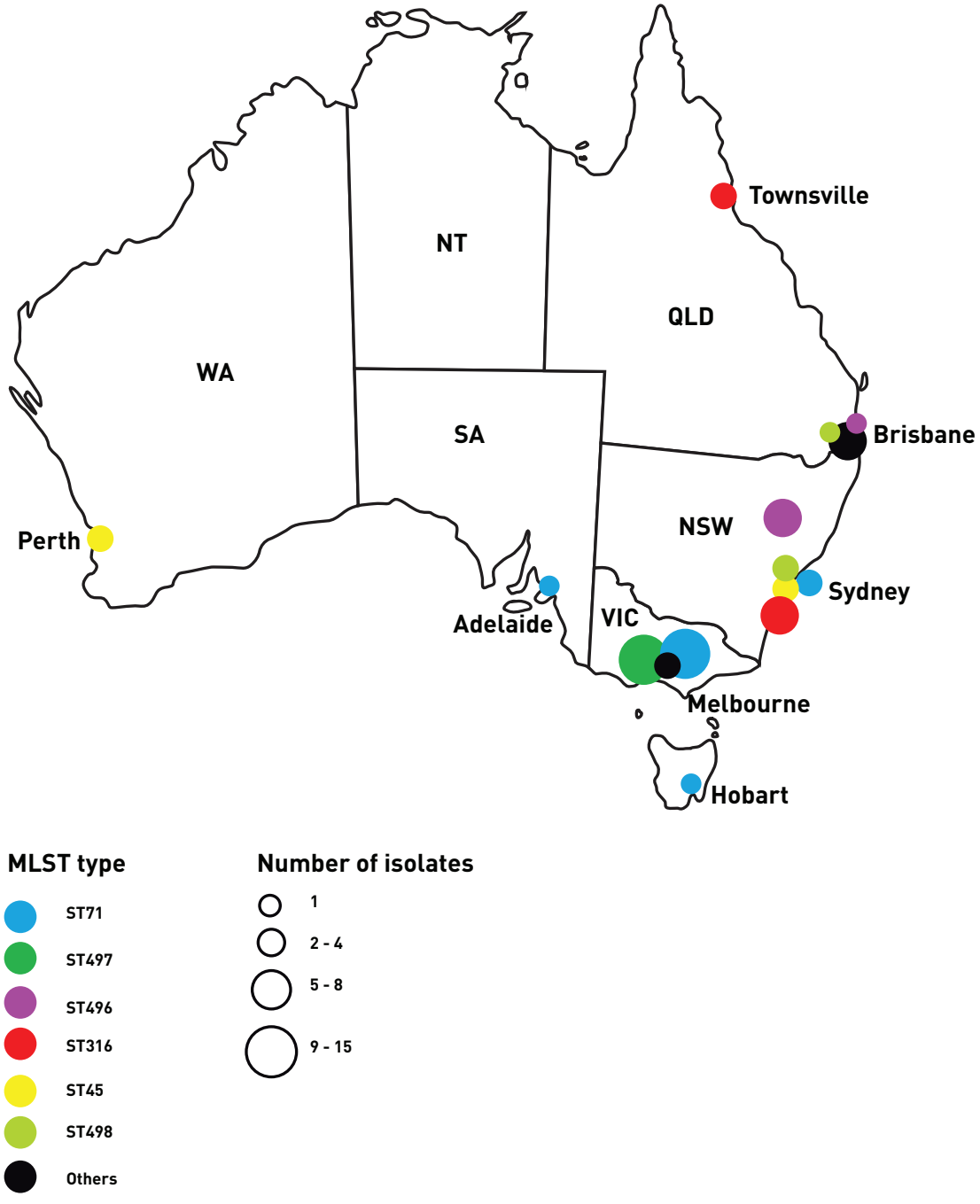

Figure 5: Distribution of Methicillin-Resistant *Staphylococcus pseudintermedius* STs in Australia.

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DOGS

CATS

DISCLAIMER

1. Not all antibiotics listed are approved for animal use and practitioners should consult https://portal.apvma.gov.au/pubcris for registered formulations.
2. The contents of this guideline are the opinions of the authors and Zoetis takes no responsibility for clinical outcomes resulting from the use of this guideline or for any off-label use of medications recommended within this guideline.

CONDITION: SUBCUTANEOUS ABSCESS/ CELLULITIS/BITE WOUNDS

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Bite wounds inflicted by dogs generally have significant lateral shearing forces. This results in extensive tearing and disruption of tissues.

Abscesses in dogs often result from migration of foreign bodies such as grass awns, with translocation of bacteria either from the oral cavity or the environment. Bite wounds inflicted by dogs generally have significant lateral shearing forces. This results in extensive tearing and disruption of tissues. For this reason, such wounds are typically presented early, often when the wound is contaminated with bacteria but not yet infected, with the aim of therapy being prophylactic.

Fight wounds are often contaminated by a variety of obligate and facultative anaerobic organisms from the oral cavity and gingival cleft. Other bacteria from the skin surface and mucous membranes, such as *Staphylococcus pseudintermedius* and *Streptococcus* spp. (which may potentially cause necrotising fasciitis [NF] and toxic shock syndromes [TSS]), can become important pathogens, and occasionally soil saprophytes which enter the wounds as contaminants (such as *Nocardia* spp., *Pseudomonas aeruginosa*, rapidly growing mycobacteria and fungi) can give rise to chronic infections that fail to respond to standard therapy.

CONDITION: SUBCUTANEOUS ABSCESS/ CELLULITIS/BITE WOUNDS

TESTS FOR DIAGNOSIS

It can be helpful to make smears of purulent exudate if present. Gram or Diff-Quik® staining may demonstrate causative bacteria. C&S testing may be helpful, especially in cases that have failed to respond to empiric therapy.

Radiography with contrast (fistulogram), ultrasonography and occasionally advanced cross-sectional imaging may be useful to detect inciting foreign bodies such as grass seeds, wood splinters, teeth, or metallic fragments. The bites of large dogs may result in internal trauma to the thorax, abdomen, or spine. This may also require radiography, ultrasonography, or CT scanning to identify organ damage. Radiography may also be useful for identifying fractured bones caused by large shearing forces associated with the bite. Penetrating bite wounds associated with fractured bones are at increased risk of developing osteomyelitis and surgical fracture repairs may have increased risk of infection. Early identification and assessment of fractured bones (open fractures) is required. Instituting prophylactic antibiotics may reduce the opportunity for bacteria to colonise and form biofilms on devitalised bone.

KEY ISSUES

01 Dog fight wounds involve lateral shearing forces, major disruption of tissues and an open draining wound.

02 There is variable contamination with a variety of different bacteria.

03 *Streptococcus* species can sometimes be important pathogens in this setting.

04 Occasionally, subcutaneous infections occur due to migrating plant foreign bodies such as grass seeds and awns.

CONDITION: SUBCUTANEOUS ABSCESS/ CELLULITIS/BITE WOUNDS

TREATMENT

- Debridement, drainage, and wound reconstruction are critical to prevent infections developing. Be very wary of using monotherapy with currently registered veterinary fluoroquinolones as this may induce superantigen expression and potentially, NF/TSS in otherwise uncomplicated *Streptococcus canis* infections in some patients, especially if combined with corticosteroid therapy. Thorough exploration of dog fight wounds is important as the 'iceberg effect' is often present, with greater disruption to subcutaneous tissues being present than suggested by the appearance of the surface wound. Opening pockets of devitalised tissues, wound debridement and the strategic placement of drains are just as critical as careful selection of antimicrobial agents. Placing latex (Penrose) or Jackson Pratt drains to facilitate removal of exudate while minimising wound dead space is often helpful.

RECOMMENDED ANTIBIOTICS

Emphasis should be placed on selecting agents active against Gram-positive cocci.

First line:
Amoxicillin-clavulanate
(20 mg/kg q12h PO; 8.75 mg/kg q24h SC or IM)

Second line:
Based on C&S

USAGE RECOMMENDATION

There is no evidence-base to guide recommendations regarding treatment duration. From experience, the panel recommends at least 4 days and ideally 7–14 days. Amoxicillin-clavulanate, initially by injection (SC or IM), and subsequently PO offers the best antimicrobial spectrum of activity.

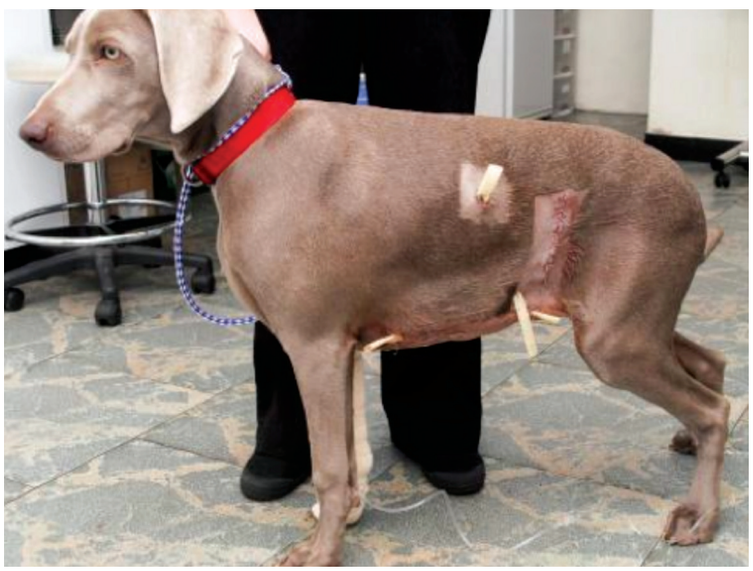
An IV combination of drugs in more severe cases may be utilised when rapid high blood levels is desirable. For example, ampicillin/amoxicillin plus gentamicin, or a first-generation cephalosporin plus gentamicin.

Fluoroquinolones should not be used unless indicated by C&S testing (for example, *Pseudomonas aeruginosa* superinfection).

CONDITION: SUBCUTANEOUS ABSCESS/ CELLULITIS/BITE WOUNDS

AIDAP TOP TIPS

Avoid empiric use of fluoroquinolones in dogs with fight wounds, as they may predispose to the development of life-threatening streptococcal infections.



Latex drain placed to allow ongoing gravitational drainage.
Photo courtesy of Dr Anne Quain.

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CONDITION: USE OF ANTIBIOTICS IN DENTAL PROPHYLAXIS (PERIODONTAL THERAPY)

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Most bacteria found in the mouths of dogs and cats are similar to those recovered from bite wounds.

Cats appear to have a more diverse oral microbiota when compared to dogs. Certain bacterial species are associated with periodontal disease (PD) in dogs and cats. Gram-negative anaerobes such as *Porphyromonas* spp., *Prevotella* spp., *Fusobacterium* spp. and spirochaetes (*Treponema* spp.) comprise most bacterial species found in mild to severe forms of PD in dogs and cats. However, Gram-positive anaerobes such as *Peptostreptococcus* also feature in significant numbers in PD in dogs and cats, unlike in humans, where gram-positive anaerobes do not feature in the pathogenesis of periodontal disease. *Pasteurella multocida* and anaerobic Gram-negative rods including *Capnocytophaga* are frequently involved, and these are all susceptible to ß-lactam antibiotics including penicillin, amoxicillin, amoxicillin-clavulanate and first-generation cephalosporins.

Numerous studies have shown that a transient bacteraemia can occur in both humans and dogs after dental procedures. This bacteraemia nearly always occurs following tooth extraction(s). In a healthy dog, the bacteraemia is cleared in about 30 minutes, however, in animals with diseases (such as renal, cardiac or hepatic dysfunction), compromised immune systems, poorly controlled diabetes mellitus or other serious endocrinopathies, it is recommended that antimicrobial therapy be given prophylactically at the time of surgery. Perioperative antibiotics may also be indicated in patients with less common systemic risk factors such as subaortic stenosis and orthopaedic implants placed in the last 12–18 months. If unsure, it is recommended to consult with the cardiologist or surgeon prior to commencing.

CONDITION: USE OF ANTIBIOTICS IN DENTAL PROPHYLAXIS (PERIODONTAL THERAPY)

If the trabecular bone and the outer cortical plates of the jaws are invaded by bacteria and their toxins, as well as by host inflammatory mediators, osteomyelitis can occur, which then requires an extended course of antimicrobials.

Pre-wiping the teeth and gingivae with chlorhexidene digluconate 0.12% is also helpful prior to performing a dental cleaning or tooth extraction. It has been shown to reduce the number of aerosolised bacteria during these procedures, reducing the risk of inhalation by both the operator and the assistant.

Most dental procedures in companion animals are classified as clean-contaminated procedures, meaning that following tooth extractions systemic antibiotics are usually not indicated unless there is marked oral inflammation or purulent discharge associated with the affected teeth.

Periodontal Disease stage 4 (PD stage 4) is where there is >50% attachment loss or class 3 furcation, which is evident on intraoral radiographs. Preoperative antibiotics commenced several days before surgery may be administered in cases of PD stage 4 for the purpose of reducing the bacterial load in the oral cavity and making tissues more amenable to surgical handling. This should also be the case when dealing with gingivostomatitis cases where selective or whole mouth extractions are planned.

CONDITION: USE OF ANTIBIOTICS IN DENTAL PROPHYLAXIS (PERIODONTAL THERAPY)

TESTS FOR DIAGNOSIS

- Diagnostic tests for PD include visual and tactile inspection, periodontal probing, and intraoral radiographs. These tests are all included in an extra- and intra-oral examination or the recently termed, comprehensive oral health assessment and treatment (COHAT), which is performed under general anaesthesia. Especially in cats (and sometimes in dogs), the detection of tooth resorptions at the cervical margin is performed both with tactile examination (using an explorer probe) as well as the use of intraoral radiographs.

  Any localised or asymmetrical lesion in the oral cavity should be biopsied to rule out other oral pathology.

  Because of the large number of bacterial species in the mouth, C&S testing is usually unrewarding.

TREATMENT

A dental prophylaxis (or the preferred term, periodontal therapy) will include a COHAT, followed by dental cleaning including removal of plaque and calculus above and below the gums (supra and subgingival debridement). Periodontal therapy will include extraction of poor to hopeless prognosis teeth (furcation 3 and/or >50 % attachment loss around the tooth).

The use of 10% povidone-iodine disinfectant following mechanical periodontal therapy has been shown in human dentistry to reduce periodontal pocket depth (at least in deeper pockets >5 mm) when used as a final subgingival irrigation (needs up to 5 minutes contact time within the pocket).

Normally, antibiotics are not required for extractions unless dealing with a surgical extraction (where a mucoperiosteal flap is raised and bone removed), or multiple extractions associated with severe periodontitis.

KEY ISSUES

- Prophylactic antibiotics (if required) are best administered prior to the procedure. For example, procaine penicillin, amoxicillin or amoxicillin-clavulanate can be administered SC or IM at the time of premedication or shorter acting benzyl penicillin or first-generation cephalosporin (cefazolin) can be given IV immediately after anaesthetic induction. These would cover the great majority of potential pathogens in this setting.

CONDITION: USE OF ANTIBIOTICS IN DENTAL PROPHYLAXIS (PERIODONTAL THERAPY)

RECOMMENDED ANTIBIOTICS

First line:

Penicillin
(Procaine penicillin 30 mg/kg q24h IM or SC dogs, SC cats)

Amoxicillin
(22 mg/kg q12h PO; 7.5 mg/Kg q24h SC or IM)

Second line:

Amoxicillin-clavulanate
(20 mg/kg q12h PO; 8.75 mg/kg q24h SC or IM)

Clindamycin
(5.5 mg/kg q12h PO)

Doxycycline monohydrate
(5 mg/kg q12h PO†)

Third line:

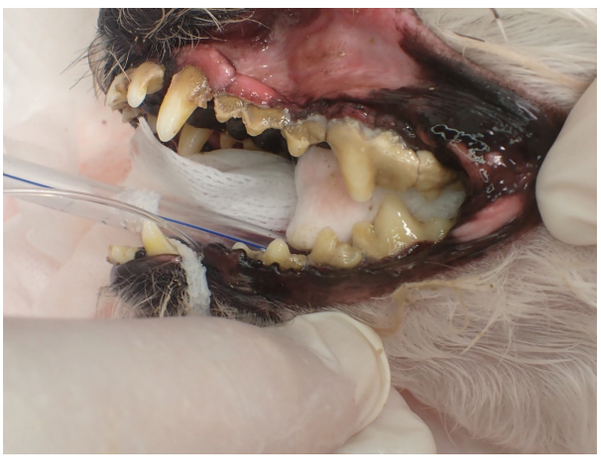
Cefovecin (8 mg/kg SC) is suitable for cases where there are concerns of compliance, or there are difficulties with oral dosing.

†Ensure doxycycline is given with food and access to drinking water

USAGE RECOMMENDATION

Penicillin or amoxicillin can be given prior to the dental procedure and may be continued q12h PO only in those situations of moderate to severe periodontitis, multiple extractions, or extensive oral surgery or those with moderate co-morbidities.

A perioperative dose of amoxicillin or amoxicillin-clavulanate can be given at the time of premedication (30 to 60 minutes prior to procedure) when performing surgical extractions. Where the patient has severe periodontitis and multiple extractions are anticipated, a preoperative and postoperative course of antibiotics may be required (determined on a case-by-case basis).



Photograph courtesy of Dr Anthony Caiafa

CONDITION: USE OF ANTIBIOTICS IN DENTAL PROPHYLAXIS (PERIODONTAL THERAPY)

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BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Most dental procedures in companion animals are classified as clean-contaminated procedures, meaning that after extractions, systemic antibiotics are usually not indicated unless there is marked oral inflammation or purulent discharge associated with the affected teeth.

Most bacteria found in the mouths of dogs (and cats) are similar to those recovered from bite wounds. *Pasteurella multocida*, and anaerobic Gram-negative rods including *Porphyromonas* spp. and *Capnocytophaga* spp. are frequently involved, and these are all susceptible to penicillins, including procaine penicillin and amoxicillin-clavulanate.

Periodontal disease

Periodontal disease (PD) is a bacterial plaque-induced inflammatory disease, involving the supporting structures of the teeth. Plaque bacteria form colonies, called plaque biofilms, and these biofilms can lead to gingival inflammation (gingivitis). Gingivitis is reversible if plaque reduction measures, such as toothbrushing, are initiated. However, if plaque accumulates, gingivitis may eventually lead to periodontitis. Periodontitis is the irreversible loss of the periodontal attachment to the tooth (including the gingiva, cementum, periodontal ligament, and the supporting alveolar bone). Some plaque bacteria can even invade gingival cells and thus potentially evade the host's defence mechanisms. PD is considered a complex inflammatory disease involving interaction between plaque bacteria and the host's immune system, which can lead to further inflammation and attachment loss around the tooth with the eventual loosening of the tooth.

Numerous studies have shown that a transient bacteraemia can occur in both humans and dogs after dental procedures. This bacteraemia nearly always occurs following tooth extraction(s). In a healthy dog, the bacteraemia is cleared in about 30 minutes, however, in animals with diseases such as renal, cardiac, or hepatic dysfunction, compromised immune systems, poorly controlled diabetes mellitus or other serious endocrinopathies, it is recommended that antimicrobial therapy be given prophylactically at the time of

CONDITION: PERIODONTAL DISEASE

surgery. Intraoperative antibiotics may also be indicated in patients with less common systemic risk factors such as subaortic stenosis and orthopaedic implants placed in the last 12–18 months.

Pre-wiping the teeth and gingivae with chlorhexidine digluconate (<0.2%) is also helpful prior to performing a dental cleaning or tooth extraction. It has been shown to reduce the number of aerosolised bacteria during these procedures, reducing the risk of inhalation for both the operator and the nearby assistant.

Most dental procedures in companion animals are classified as clean-contaminated procedures, meaning that after extractions, systemic antibiotics are usually not indicated unless there is marked oral inflammation or purulent discharge associated with the affected teeth. Systemic antibiotics may also be indicated for PD stage 3 (PD3: 25-50% attachment loss, based on periodontal probing depths and intraoral radiographs) or PD stage 4 (PD4: >50% attachment loss, based on periodontal probing depths and intraoral radiographs). Preoperative antibiotics commenced several days prior to surgery, may be administered in cases of severe PD, for the purpose of reducing bacterial induced inflammation and making tissues more amenable to surgical handling. This should also be the case when dealing with gingivostomatitis cases where selective or whole mouth extractions are planned.

If the trabecular bone and the outer cortical plates of the jaws are invaded by bacteria and their toxins, as well as by host inflammatory mediators, osteomyelitis can occur, which then requires an extended course of antimicrobials.

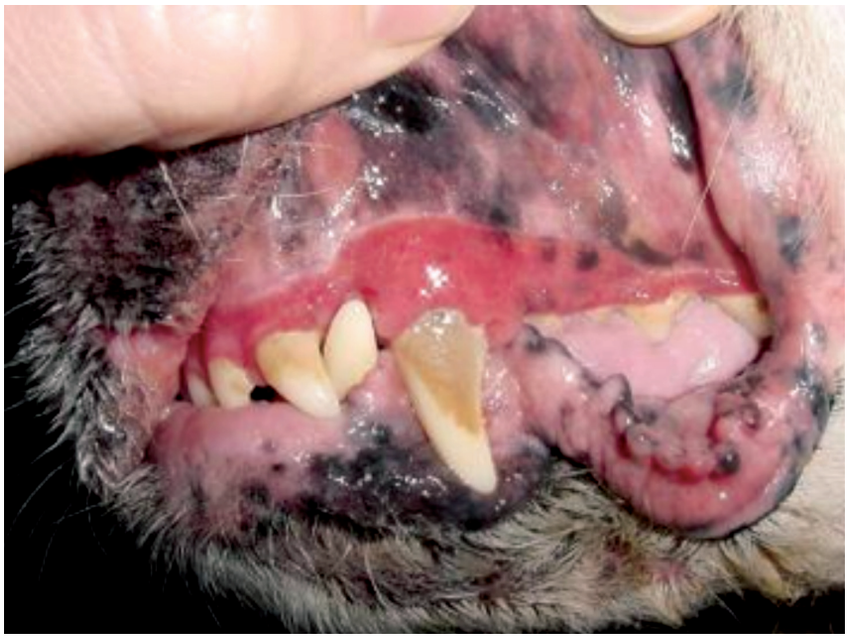
CONDITION: PERIODONTAL DISEASE

TESTS FOR DIAGNOSIS

1. Under general anaesthesia, a complete oral health assessment and treatment (COHAT) should be performed, including periodontal probing and intraoral radiographs

2. Any localised or asymmetrical lesion in the oral cavity should be biopsied to rule out other disease

3. Because of the large number of bacterial species in the mouth, C&S testing is unrewarding



Canine patient with calculus (tartar) and mild to moderate periodontal disease, especially in the vicinity of the canine and carnassial teeth of the upper dental arcade.

Photo courtesy of Dr Richard Malik.

CONDITION: PERIODONTAL DISEASE

TREATMENT

- The initial management of periodontal disease will include extra- and intra-oral examination, diagnostic tests such as periodontal probing and dental radiography, followed by mechanical scaling and root debridement (periodontal therapy) performed under general anaesthesia (comprehensive oral health assessment and treatment or COHAT).

- Periodontal therapy includes the removal of plaque and calculus above and below the gums (supra and subgingival debridement). Periodontal therapy also includes extraction of poor to hopeless prognosis teeth (furcation 3 and/or >50% attachment loss around the tooth).

- Normally, antibiotics are not required for extractions, but should be considered when dealing with a surgical extraction (where a mucoperiosteal flap is raised and bone removed) or multiple extractions associated with severe PD. A perioperative dose of an antibiotic can be given at the time of premedication when performing surgical extractions. Where the patient has severe PD and multiple extractions are anticipated, a preoperative and postoperative course of antibiotics may be required (determined on a case-by-case basis)

- The use of 10% povidone-iodine disinfectant following mechanical periodontal therapy has been shown in humans to reduce periodontal pocket depth (at least in deeper pockets >5 mm) when used as a final subgingival irrigation (needs up to 5 minutes contact time within the pocket).

- The long-term management of PD involves the regular professional removal of plaque and calculus from the tooth surface, as well as the instigation of a homecare program, with the goal of either maintaining periodontal health or, where there is severe disease, providing treatments that will predictably arrest disease progression and give long term periodontal stability, as well as comfort to the pet. The short-term use of antibiotics can play an adjunctive role in this management plan.

CONDITION: PERIODONTAL DISEASE

RECOMMENDED ANTIBIOTICS

First line:

Amoxicillin
(22 mg/kg q12h PO; 7.5 mg/kg q24h SC or IM)

Amoxicillin-clavulanate
(20 mg/kg q12h PO; 8.75 mg/kg q24h SC or IM)

Second line:

Clindamycin (5-11 mg/kg q 12h PO)
Doxycycline monohydrate (5 mg/kg q 12h PO†)

Third line:

Cefovecin (8 mg/kg SC) is suitable for cases where there are concerns of compliance, or there are difficulties with oral dosing

†Ensure doxycycline is given with food and access to drinking water

USAGE RECOMMENDATION

Prophylactic antibiotics are best administered prior to the procedure. For example, amoxicillin or amoxicillin-clavulanate administered SC or IM after premedication.

Amoxicillin or amoxicillin-clavulanate would cover the great majority of potential pathogens in this setting.

AIDAP TOP TIPS

Further therapy is directed at changing the diet to include more chewing.

Further adjunctive therapy can include the use of Veterinary Oral Health Council (VOHC) accepted "dental" diets and "dental" chews that can mechanically remove plaque from the tooth surface. These "dental" diets and chews can be used in conjunction with tooth brushing and/or chlorhexidine, which are still considered the "gold standard" for plaque and calculus (tartar) control in our pets, as they are in humans.

CONDITION: PERIODONTAL DISEASE

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CONDITION: CANINE INFECTIOUS RESPIRATORY DISEASE COMPLEX

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Canine infectious respiratory disease complex (CIRDC), a.k.a. "kennel cough" or infectious tracheobronchitis, is associated with an acute onset of cough, with or without sneezing.

Most dogs are not febrile at presentation. In some cases, nasal and/or ocular discharge may be present.

Co-infections are common.

Viral causes include Canine adenovirus 2 (CAV), Canine distemper virus (CDV), Canine respiratory coronavirus (CRCoV), Canine herpesvirus (CHV), Canine pneumovirus, Influenza viruses (H3N8, H3N2) and Canine parainfluenza virus.

Primary bacterial causes include *Bordetella bronchiseptica*, *Streptococcus equi* subspp. *zooepidemicus* and *Mycoplasma cynos*. *S. zooepidemicus* infections are mostly confined to multi-dog environments, such as breeding kennels or shelters.

H3N2 Canine Influenza has not been reported in Australia, and H3N8 was only detected in serological surveillance studies during an outbreak of H3N8 Equine Influenza in the early 2000s.

CDV has not been eradicated in Australia. CDV should be considered as a differential diagnosis in puppies with mucopurulent nasal and ocular discharge, especially if diarrhoea or neurological signs are present.

CONDITION: CANINE INFECTIOUS RESPIRATORY DISEASE COMPLEX

TESTS FOR DIAGNOSIS

1. Nasal swab bacterial C&S testing is not indicated in dogs with CIRDC (see above).
2. Molecular (PCR) respiratory panels are of low value (see above).
3. In multi-dog environments (for example shelters or breeding kennels), respiratory PCR panels are more useful if several dogs are sampled within 1-3 days of the onset of clinical signs when viral loads are the highest, or from exposed dogs that have not yet developed clinical signs. Other tests may be considered such as virus isolation or bacterial culture for primary pathogens. Necropsies and histopathology should be performed on any animals that die during an outbreak of CIRDC. Immunohistochemical stains can be performed to identify specific viruses, such as CDV.
4. If there is no response to antimicrobial therapy within 7 days, further investigations should be performed (see Acute Lower Respiratory Tract Infection) before changing antimicrobials or adding other antimicrobials.

KEY ISSUES

01 Co-infections are common in CIRDC.

02 Some bacteria are primary pathogens and cause CIRDC, for example *Bordetella bronchiseptica* and *Mycoplasma cynos.*

03 Viruses are implicated in most cases of CIRDC, and even with bacterial co-infections, disease usually resolves within 10 days without antimicrobial treatment.

04 Nasal swab bacterial C&S testing is not indicated in dogs with CIRDC since non-causative commensals will often be cultured and primary pathogens can be isolated from healthy or diseased dogs.

05 PCR is of low value in individual cases since causative agents can be detected in healthy and diseased dogs and in dogs that have been recently vaccinated with modified live pathogen strains.

06 **Antimicrobial therapy is only recommended in dogs with purulent or mucopurulent nasal discharge that are febrile, anorexic, or lethargic. Further investigation is recommended if dogs present with clinical signs of pneumonia such as crackles or wheezes on thoracic auscultation.**

CONDITION: CANINE INFECTIOUS RESPIRATORY DISEASE COMPLEX

TREATMENT

- Take a thorough history, such as any access to kennels, pounds, other dogs at shows, including vaccination history, perhaps thoracic radiography (in certain circumstances) and then trial empiric therapy using doxycycline monohydrate, which is arguably the most effective and reliable agent against *Bordetella* because of its high penetration of respiratory mucous. It also has good efficacy for *Pasteurella* spp. and secondary obligate anaerobes.
- Amoxicillin-clavulanate is a less satisfactory choice because, being charged and water soluble, it tends not to reach sufficiently high levels in respiratory mucous.
- In young animals, fluoroquinolones should not be used because of their effects on growing cartilage.
- Cough suppressants (for example opioids) may be appropriate under some circumstances (persistent dry cough). Nebulisation therapy using saline (with or without gentamicin) may also be helpful.

ANTIBIOTICS USED

In most circumstances no antimicrobials are required unless there is bacterial pneumonia or marked co-morbidities.

First line:

Doxycycline monohydrate (5 mg/kg q12h or 10 mg/kg q24h PO†)

Second line:

Amoxicillin (22 mg/kg q12h PO)
Amoxicillin-clavulanate (20 mg/kg q12h PO)

†Ensure doxycycline is given with food and the animal has access to drinking water.

USAGE RECOMMENDATION

Doxycycline generally has in vitro and/or in vivo efficacy against *Mycoplasma cynos* and *B. bronchiseptica*, and has activity against many opportunistic bacterial species.

Amoxicillin and amoxicillin-clavulanate have little or no activity against *B. bronchiseptica* and *Mycoplasma cynos* but have good efficacy against secondary bacterial infections. These agents may be used where doxycycline therapy is not possible, not tolerated, or not effective as a first line.

CONDITION: CANINE INFECTIOUS RESPIRATORY DISEASE COMPLEX

Key references:

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CONDITION: CHRONIC RHINOSINUSITIS (CRS)

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

The most common causes of chronic nasal discharge and sneezing in dogs are idiopathic lymphoplasmacytic rhinitis (LPR), sinonasal aspergillosis (SNA), foreign bodies and neoplasia.

Less common causes include allergic rhinitis and nasal or nasopharyngeal stenosis.

Bacterial rhinitis is uncommon and almost always occurs secondary to a foreign body, odontogenic infection mycotic rhinitis, maxillary/turbinate trauma, or irradiation.

Viral rhinitis due to canine distemper virus is rare but should be considered as the possible cause of mucopurulent nasal/ conjunctival discharge in young and/or unvaccinated dogs.

Underlying allergic, infectious, and immune-mediated causes have been implicated in LPR. Diagnosis of LPR requires demonstration of lymphoplasmacytic nasal mucosal infiltrates on histology and the exclusion of other causes of disease. Periodontal disease, especially associated with fractured maxillary teeth (crown or crown root fracture), can also cause rhinitis, and may be misdiagnosed as LPR. Maxillary lesions on intra-oral radiographs that may be associated with rhinitis include tooth resorption, endodontic disease, periodontal disease and retained tooth roots.

CONDITION: CHRONIC RHINOSINUSITIS (CRS)

TESTS FOR DIAGNOSIS

1. Serological testing for *Aspergillus* antibodies is a useful non-invasive blood test for SNA. ELISA is more sensitive than agar gel immunodiffusion assays. A negative result does not rule out SNA.
2. Serology to detect cryptococcal antigen has high sensitivity to rule out cryptococcal rhinitis, which is less common than SNA.
3. Other diagnostic techniques include dental radiographs, advanced diagnostic imaging (CT or MRI), endoscopic evaluation of the nasal cavity and nasopharynx, deep nasal lavage, biopsy, and histopathology.

KEY ISSUES

01 Primary chronic bacterial rhinosinusitis is is rare in dogs.

02 Mucopurulent nasal discharge is usually caused by a foreign body or a disease that causes severe turbinate destruction, especially sinonasal aspergillosis (SNA) or neoplasia.

03 Periodontal disease can cause chronic nasal discharge and sneezing in dogs, especially when associated with fractured maxillary teeth.

TREATMENT

- Antimicrobials should not be prescribed routinely to dogs presenting with nasal discharge and sneezing, since primary bacterial rhinitis is rare.

CONDITION: CHRONIC RHINOSINUSITIS (CRS)

RECOMMENDED ANTIBIOTICS

First line:

None empirically.

Second line:

May be appropriate after investigation and C&S. For example, SNA may need antimicrobial therapy for secondary bacterial infection.

Most commonly:

Doxycycline monohydrate (5 mg/kg q12h or 10 mg/kg q 24h PO†)

†Ensure doxycycline is given with food and the animal has access to drinking water.

AIDAP TOP TIPS

1. Mucopurulent nasal discharge in dogs is almost always secondary to an underlying primary cause.
2. Foreign bodies, sinonasal aspergillosis, and neoplasia, are the most common causes of mucopurulent discharge, and may also be accompanied by epistaxis. Depigmentation of the nasal planum should arouse suspicion for SNA.

CONDITION: CHRONIC RHINOSINUSITIS (CRS)

Profuse nasal discharge, crusting and depigmentation of the nasal planum in a dog with sinonasal aspergillosis.

Photos courtesy of Dr Vanessa Barrs.

Close-up of the dog in previous photo after removal of nasal discharge, illustrating crusting and depigmentation of the nasal planum, which are common findings in dogs with sinonasal aspergillosis – an important differential diagnosis for rhinitis in dogs.

Key references:

1. Lappin MR, Blondeau J, Boothe D, Breitschwerdt EB, et al. J Vet Int Med. 2017; 31:279-294.
2. Stepaniuk KS and Gingerich W. J Vet Dent. 2015; 32(1):22-29.
3. Sharman M, Paul A, Davies D, et al. J Small Anim Pract. 2010; 51(8): 423–427.
4. Nelson HS. J Allergy Clin Immunol. 2007; 119(4): 872–880.

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Bacterial bronchitis

Acute bacterial bronchitis can be caused by the same primary pathogens that cause CIRDC, including viruses (Canine adenovirus 2 (CAV), Canine distemper virus (CDV), Canine respiratory coronavirus (CRCoV), Canine herpesvirus (CHV), Canine pneumovirus, Influenza viruses and Canine parainfluenza viruses; and bacteria (*Bordetella bronchiseptica, Streptococcus equi* subspp. *zooepidemicus* and *Mycoplasma cynos). B. bronchiseptica* and Mycoplasmas can also cause chronic bronchitis or bronchopneumonia. Secondary bacterial bronchitis is relatively uncommon and occurs in association with upper airway diseases such as collapsing trachea and laryngeal bronchitis.

In some dogs, acute bacterial bronchitis can become chronic. In addition, dogs with other inflammatory or anatomic upper airway abnormalities can develop secondary bacterial bronchitis. Canine chronic bronchitis (CCB) can also be complicated by secondary bacterial infection. CCB is a non-infectious inflammatory airway disease, typically characterised by frequent cough (daily) of greater than two months duration in association with neutrophilic and/or eosinophilic airway inflammation, mucous hypersecretion, and loss of ciliated epithelial cells. In most cases CCB has no identifiable cause and disease is progressive.

Pneumonia

Bacterial infection of the lungs in dogs is usually secondary to a predisposing anatomic or physiological condition. A common cause is aspiration during the peri-anaesthetic period or in association with megaoesophagus or laryngeal paralysis. Other inciting causes include viral infections, inhaled foreign bodies and primary respiratory disorders such as ciliary dyskinesia and bronchiectasis. Common bacteria include *E. coli, Pasteurella* spp., *Streptococcus* spp., *Enterococcus* spp., *S. pseudintermedius,* other coagulase-positive *Staphylococcus* spp., and *Pseudomonas* spp.

Primary bacterial pneumonia, also known as community-acquired pneumonia (CAP) is caused by contagious bacteria such as *B. bronchiseptica* and *S. equi subspp. zooepidemicus, S. canis,* and *Mycoplasma* spp.

Molecular techniques have shown that CAP is associated with the loss of microbial diversity and overgrowth of a single bacterial species such as *B. bronchiseptica*. In secondary pneumonias, there is a loss, but not abolishment, of microbial diversity.

Clinical signs in dogs with bacterial LRTI can include coughing, dyspnoea, tachypnoea, fever, and lethargy. Some dogs with pneumonia may have purulent nasal discharge. Additionally, auscultation may demonstrate crackles, wheezes, and harsh breath sounds.

CONDITION: LOWER RESPIRATORY TRACT INFECTION (LRTI)

KEY ISSUES

01 Canine chronic bronchitis is an idiopathic inflammatory airway disorder of dogs that can be complicated by acute exacerbations associated with secondary bacterial infections.

02 Bacterial pneumonia is most commonly secondary to an underlying anatomic or physiologic abnormality. Community acquired (contagious) pneumonias are less common.

03 Since coughing can be caused by non-bacterial disorders or by cardiovascular diseases, thorough physical examination including auscultation of all heart valves, trachea and all lung fields is essential to help localise the problem and rank differential diagnoses.

04 In addition to thoracic radiographs, in most cases, airway sampling is warranted. The best samples for cytology and culture are those obtained via bronchoscopy or transtracheal wash.

05 Advanced diagnostics may be required for further investigation, such as bronchoscopy, computed tomography (CT) and echocardiography.

CONDITION: LOWER RESPIRATORY TRACT INFECTION (LRTI)

TESTS FOR DIAGNOSIS

1. Clinical examination should help rank differential diagnoses for cough, including pulmonary oedema, allergic or hypersensitivity disorder, haemorrhage, and neoplasia.

2. In dogs with chronic cough, if bronchitis is suspected, full-inspiratory thoracic radiographs help evaluate for pulmonary and cardiac causes of cough. Inspiratory and expiratory radiographs or fluoroscopy of the cervical and intrathoracic trachea helps identify collapsing airways. Other tests include heartworm testing, faecal analysis for eggs/nematode larvae, NT-proBNP as a biomarker for left atrial enlargement/congestive heart failure/pulmonary hypertension, and echocardiography. Bronchoscopy, including bronchoalveolar lavage (BAL) is recommended to obtain samples for cytology and bacterial C&S testing. PCR for *B. bronchiseptica* and *Mycoplasma cynos* in BAL fluid samples can also be used.

3. In dogs with acute cough associated with fever and dyspnoea or tachypnoea, a complete blood count (CBC) should be performed in addition to thoracic radiographs. Findings suggestive of infection include neutrophilia, which may be accompanied by a left shift. In severe acute infection, neutropenia may occur due to overwhelming demand. If CBC and thoracic radiographs indicate alveolar lung disease, a transtracheal wash (TTW), BAL by bronchoscopy, or unguided BAL via an endotracheal tube, is recommended to obtain a sample for cytology and culture.

4. For animals with severe disease (for example oxygen dependent) where anaesthesia is a risk, consider performing a TTW, which does not require anaesthesia and avoids contamination of clinical samples by oropharyngeal flora. Samples collected via bronchoscopy may also be contaminated with environmental bacteria within the scope, such as *Serratia* spp.

5. **Both aerobic and anaerobic cultures should be requested when culturing airway fluid samples.**

6. Advanced diagnostics, such as computed tomography (CT) and echocardiography, allow further investigation of primary underlying respiratory disorders.

CONDITION: LOWER RESPIRATORY TRACT INFECTION (LRTI)

TREATMENT

Bacterial bronchitis

If severe, empirical doxycycline for 7-10 days is warranted pending culture results, based on its activity against *B. bronchiseptica* and *Mycoplasma* spp. If a positive response occurs, treatment should be continued for one week after the resolution of clinical signs. If underlying CCB is present, concurrent treatment to reduce inflammation such as inhaled corticosteroids may be required and may decrease the frequency of recurrent secondary bacterial infections.

Pneumonia

Antimicrobial therapy should be guided by C&S testing. In severe pneumonia, empiric therapy is recommended and then changed as necessary. Where airway sampling is not possible, empiric "four-quadrant" antimicrobial therapy is warranted.

For dogs with acute aspiration pneumonia in the absence of sepsis, parenteral administration of an antimicrobial agent with Gram-positive (especially *Staphylococcus* and *Streptococcus* spp.) and anaerobic coverage. A ß-lactam antimicrobial alone, such as ampicillin or amoxycillin-clavulanate, may be adequate. Another option for Gram-positive and anaerobic coverage is clindamycin, although the latter is ineffective against *Bacteroides*.

Where sepsis is suspected, or in cases of severe pneumonia, addition of parenteral Gram-negative coverage (especially *Pasteurella multocida, Bordetella bronchiseptica, E. coli* and *Klebsiella pneumoniae*) is recommended, for example with enrofloxacin or marbofloxacin.

Small dogs with LRTI greatly benefit by nebulisation with saline followed by appropriate physiotherapy, viz. exercise, percussion, coupage, and elevating the hindquarters to facilitate expectoration of inflammatory exudate from the airways.

Note: *Gentamicin is not absorbed systemically when delivered via a nebuliser. Nebulisation of gentamicin (1% solution or 10 mg/mL) may have efficacy against bacteria located on the surface of the ciliary epithelium. However, it is not suitable for monotherapy in cases of bacterial pneumonia and adequate coverage with systemic antibiotic therapy is essential.*

CONDITION: LOWER RESPIRATORY TRACT INFECTION (LRTI)

RECOMMENDED ANTIBIOTICS

BACTERIAL BRONCHITIS

First line:

Doxycycline (5 mg/kg q12h or 10 mg/kg q24h PO†)

PNEUMONIA

If sepsis is not present:

Ampicillin sodium (22–30 mg/kg q8h IV or SC)
Amoxicillin (20–30 mg/kg q8h IV or IM)
Clindamycin (10 mg/kg q12h SC)

If sepsis/severe pneumonia add:

Enrofloxacin (5–20 mg/kg q24h IV or IM) or
Marbofloxacin (2.75-5.5mg/kg PO q24h) and
Metronidazole (10 mg/kg q12h IV)

Gentamicin (9–14 mg/kg q24h IV) can be given instead of enrofloxacin if the latter is contraindicated.

†Ensure doxycycline given with food or water bowl provided.

USAGE RECOMMENDATION

Empiric antimicrobial therapy of bacterial bronchitis is only recommended if clinical disease is severe.

Duration of therapy for bacterial bronchitis: Continue treatment for one week after clinical signs resolve if there is improvement within the first 7-10 days of therapy.

For initial treatment of severe acute pneumonia, parenteral therapy, preferably administered intravenously is recommended to rapidly obtain adequate plasma concentrations of antimicrobials. Transition to oral therapy when clinical signs improve.

Gentamicin is potentially nephrotoxic and is contraindicated in the presence of dehydration or renal insufficiency.

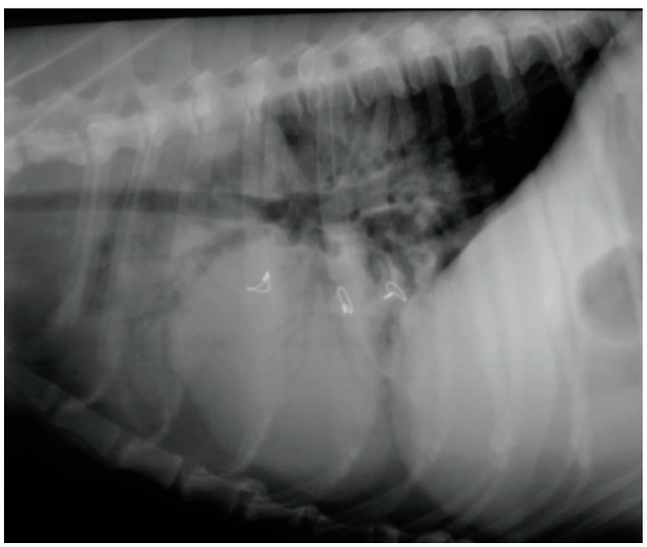
Duration of therapy for sepsis/severe pneumonia: Evaluate response to therapy after 10-14 days (physical examination, haematology, radiographs). Extend therapy as required.

CONDITION: LOWER RESPIRATORY TRACT INFECTION (LRTI)

AIDAP TOP TIPS

1. Empiric antimicrobial therapy of bacterial bronchitis is only recommended if clinical disease is severe.

2. Small dogs with LRTI greatly benefit by nebulisation with saline followed by appropriate physiotherapy, viz. exercise, percussion, coupage, and elevating the hindquarters to facilitate expectoration of inflammatory exudate from the airways.



Radiograph of a 12 year old dog with bronchopneumonia. Air bronchograms are evident. Previous metal sutures are from surgery for PDA as a puppy. This dog had a TTW with a heavy growth of *Klebsiella pneumoniae* isolated.

Photo courtesy of Dr Steve Holloway.

Key references:

1. Lappin MR, Blondeau J, Boothe D, Breitschwerdt EB, et al. J Vet Int Med. 2017; 31:279-294.
2. Jambhekar A, Robin E, Le Boedec K. J Vet Intern Med. 2019; 33(5):1880-1891.
3. Ericsson AC, Personett AR, Rindt H, Grobman ME, Reinero CR. PLoS ONE. 2020; 15(1): e0228085.
4. Jambehkar A, Robin E, Le Boedec K. J Vet Intern Med. 2019; 33:1880-1891.
5. Cannone AM, Billen F, Tual C, et al. C. J Vet Intern Med. 2016; 30(4); 1204-1209.
6. Jameson PH, King LA, Lappin MR, et al. J Am Vet Med Assoc. 1995; 206(2): 206–209.
7. Chandler JC, Lappin MR. J Am Anim Hosp Assoc. 2002; 38(2): 111–119.
8. Tart KM, Babski DM, Lee JA. J Vet Emerg Crit Care (San Antonio). 2010; 20(3): 319–329.

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Because of the wide range of organisms that may be isolated from canine pyothorax cases, C&S testing is mandatory and cost effective, as it permits targeted therapy of the specific pathogen involved.

Pyothorax is an uncommon disease in dogs. Where an underlying cause can be determined, grass awn or plant inhalation is the most common cause. Inhaled plant material is frequently contaminated with oral bacterial commensals such as *Actinomyces* spp.

In contrast to feline pyothorax, *Enterobacteriaceae*, especially *E. coli* and *Klebsiella pneumoniae* are more commonly implicated (*E. coli* in up to 54% of cases compared with 0–7% of cats), and *Nocardia* spp. infections are also more common (up to 22%, compared with 0–7% in cats). Therefore, empiric 'four quadrant' antimicrobial therapy is warranted pending results of antimicrobial susceptibility testing.

Because of the wide range of organisms that may be isolated from canine pyothorax cases, C&S testing is mandatory and cost effective, as it permits targeted therapy of the specific pathogen involved.

CONDITION: PYOTHORAX

TESTS FOR DIAGNOSIS

1. Haematology may reveal anaemia and leucocytosis and serum biochemistry may include elevations in liver enzymes, electrolyte imbalances, hypoproteinaemia and hypoglycaemia or hyperglycaemia.

2. Pleural fluid should be submitted for cytology (both Diff-Quik® and Gram staining) and C&S (aerobic and anaerobic). Gram-positive filamentous rods are suggestive of *Actinomyces, Nocardia* and occasionally some obligate anaerobes.

3. Successful anaerobic culture requires exclusion of all air from the diagnostic specimen (for example, inoculation into an anaerobic blood culture bottle at the time of thoracocentesis).

4. Post-drainage thoracic radiographs, or preferably CT, should be performed to aid in the identification of focal/lobar pneumonia, foreign bodies, lung abscesses etc.

5. In cases where a foreign body is suspected, bronchoscopy may be useful to identify and remove the instigating cause, although grass awn migration to the pleural space may have already occurred by the time of diagnosis.

KEY ISSUES

01 In contrast to cats, dogs with pyothorax are more likely to be infected with *Enterobacteriaceae* (especially *E. coli* and *Klebsiella pneumoniae*) and anaerobes, thus four-quadrant empirical antimicrobial therapy is warranted.

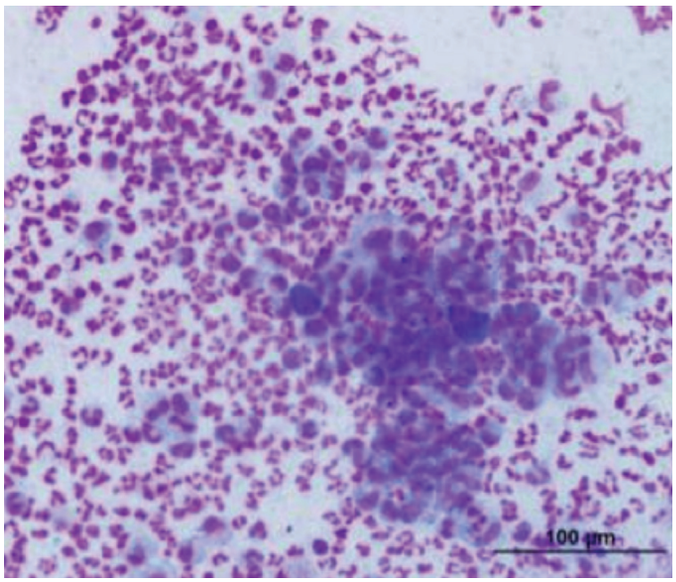
02 Inhaled plant material, especially grass awns are the most common cause of bacterial pyothorax in dogs, although in most cases, no underlying cause is identified.

03 Severe compartmentalisation of infection in the thorax and lung abscessation may require surgical intervention.

CONDITION: PYOTHORAX

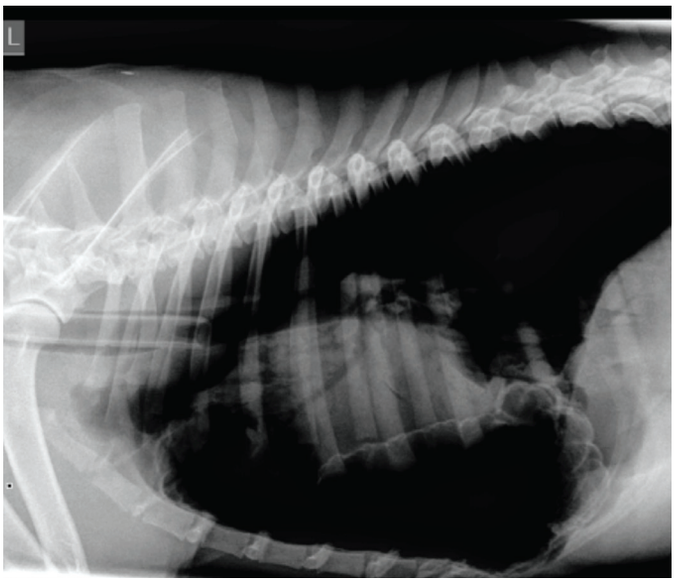
TREATMENT

1. Successful treatment requires pleural drainage and lavage plus antimicrobial therapy.
2. Before anaesthesia or sedation for chest tube placement, the pleural effusion should be drained as completely as possible using needle thoracocentesis to minimise anaesthetic complications.
3. If the pleural effusion is bilateral, bilateral indwelling thoracostomy tubes should be placed, with twice daily aspiration and lavage until pleural fluid formation is <2 mL/kg/day together with resolution of infection on pleural fluid cytology and on radiographic evaluation.
4. Therapy should be modified, if necessary, in the light of C&S results.
5. If response to pleural fluid drainage, lavage and antimicrobial therapy is not rapid, advanced diagnostic imaging (thoracic CT) and/or bronchoscopy may be warranted.



Diff-Quik® stained smear of purulent exudates from a dog which presented with tension pneumothorax and purulent pleurisy attributable to migration of grass awn(s).

Photo courtesy of Dr Shane Raidal.



Tension pnuemothorax and lobar pneumonia in a 'pig dog' following migration of a grass awn.

Photo courtesy of Dr Peter Young.

CONDITION: PYOTHORAX

ANTIBIOTICS USED

First line empiric therapy with three agents:

Ampicillin sodium (22–30 mg/kg q8h IV or SC) or Amoxicillin (20–30 mg/kg q8h IV)

and

Enrofloxacin (5–20 mg/kg q24h IV or IM)

and

Metronidazole (10 mg/kg q12hrs IV)

Gentamicin 9–14 mg/kg q24h IV can be given instead of enrofloxacin if the latter is contraindicated.

USAGE RECOMMENDATION

Thoracic instillation of antimicrobials via thoracostomy tubes is not recommended because data to support efficacy is lacking.

Treat IV initially then base on C&S and clinical response.
Change to oral antibiotics after a few days, after the animal is responding and has commenced eating.

Avoid gentamicin in dehydrated patients, or in dogs with renal insufficiency.

Duration of therapy: Repeat thoracic radiographs 10-14 days after being discharged from hospital on oral antimicrobials. Extend therapy as required. Previously, antimicrobial therapy for 3-6 weeks was recommended. Evidence to recommend a minimum period of administration is lacking and serial monitoring is recommended.

CONDITION: PYOTHORAX

AIDAP TOP TIPS

1. Needle thoracocentesis to obtain a diagnostic sample may be accomplished initially via needle aspiration (a 19-gauge butterfly is convenient in many patients) followed by placement of indwelling thoracic drains, to facilitate daily drainage and lavage with warm crystalloid solutions.

2. To confirm a diagnosis of pleural effusion in a dog presenting with a restrictive respiratory pattern (rapid, shallow breathing, inspiratory dyspnoea) thoracic ultrasound is a cost-effective diagnostic tool. Alternatively, a single view dorsoventral thoracic radiograph can confirm the presence of pleural effusion, with reduced risk of respiratory decompensation compared to acquisition of routine three views (ventrodorsal and both laterals).

3. Pyothorax is uncommon in dogs. Where possible, advanced diagnostic imaging, particularly CT is recommended at diagnosis to obtain information about possible underlying causes such as migrating grass awn.

Key references:

1. Epstein SE, Balsa IM. Vet Clin North Am Small Anim Pract. 2020; 50(2): 467-487.
2. Stillion JR, Letendre J-A. J Vet Emerg Crit Care. 2015; 25(1):113-119.
3. Boothe HW, Howe LM, Boothe DM, et al. J Am Vet Med Assoc. 2010; 236(6): 657–663.

CONDITION: ACUTE LOWER UTI/SPORADIC BACTERIAL CYSTITIS

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

In a freshly collected sample, urine pH can be a good indicator of the causative agent.

Sporadic bacterial cystitis (previously called simple or uncomplicated UTI) is an acute bacterial infection of the bladder leading to LUT clinical signs such as haematuria, stranguria, and pollakiuria. It refers to the frequency of occurrence rather than the health status of the animal, with sporadic bacterial cystitis describing an acute infection seen less than three times in the last 12 months. Infections more frequent than this are referred to as 'recurrent bacterial cystitis'.

Most UTIs result from ascending infections. Infections occur most commonly in older female dogs, with a median age of 8 years. LUT signs in dogs may also be due to urolithiasis, bladder neoplasia or prostatic disease (prostatitis, prostatic neoplasia, benign prostatic hypertrophy) with or without concurrent bacterial infection.

In published and unpublished Australian studies, the most common cause of sporadic bacterial cystitis in dogs is *E. coli* (up to 60%), followed by *Proteus mirabilis, Staphylococcus pseudintermedius* and *Enterococcus faecalis* which collectively represent >92% of cultured isolates. Less frequently isolated bacteria include *Enterobacter* spp., *Klebsiella* spp., and *Pseudomonas aeruginosa*. Most *E. coli* (>75%), *P. mirabilis* (>90%), *S. pseudintermedius* (>90%) and *Enterococcus faecalis* (>95%) remain susceptible to amoxicillin, while >90% of isolated *E. coli*, *P. mirabilis,* and *S. pseudintermedius* are susceptible to trimethoprim-sulphonamide.

In a freshly collected sample, urine pH can be a good indicator of the causative agent. Acidic pH (<7) is seen in *E. coli, Klebsiella, Enterococcus* and *Pseudomonas* infections. Alkaline pH (>8) is seen in urease-producing bacteria such as *Staphylococcus, Proteus* and less frequently in *Corynebacterium* spp. Urease-positive mycoplasmas that are adapted for life in the urogenital tract (*Ureaplasma* spp.) can be uncommon but important causes of UTI in dogs and are resistant to ß-lactams. *Mycoplasma* infection should be considered in dogs with high urine pH ± crystalluria and a high white cell count that returns a diagnostic microbiology report yielding no significant growth.

CONDITION: ACUTE LOWER UTI/SPORADIC BACTERIAL CYSTITIS

TESTS FOR DIAGNOSIS

1. Rectal examination for prostatic disease is important in male dogs while vulval examination is important in female dogs.
2. A full urinalysis is recommended for all dogs presenting with LUT signs, using a urine sample collected by cystocentesis or sterile urinary catheter where possible. If this is not possible, a mid-stream urine sample can be used for urine analysis, but it is more difficult to interpret microbial culture results in this type of sample.
3. Diagnosis of sporadic bacterial cystitis requires LUT signs with supporting evidence of UTI (epithelial cells, RBC, WBC, and bacteria) on sediment examination by wet preparation (see instructions on 'Wet Prep"), or stained urine sediment (Gram or Diff-Quik®). Beware of stain precipitates mimicking bacteria. Commercial stain additives should be avoided for this reason.
4. Free-catch urine samples are inferior and should generally be avoided. However, when this is the only option, urine analysis should be performed to determine the presence or absence of organisms by sediment exam and can provide good preliminary information on whether a UTI is likely. Normal distal UT commensals will not be visible in any substantial numbers on a wet prep or stained sediment.

KEY ISSUES

01 Increasing age and female gender are risk factors for sporadic bacterial cystitis in dogs.

02 Where subclinical bacteriuria is identified (positive urine culture or cytological evidence of bacteriuria in the absence of clinical signs and pyuria) treatment is not recommended.

03 Look for markers of underlying disease or bladder dysfunction/ abnormalities such as urolithiasis, prostatic enlargement, PU/PD, diabetes, and hyperadrenocorticism so that complications predisposing to infection can be addressed, reducing the risk of recurrent cystitis.

04 The presence of a comorbidity doesn't exclude categorisation of a UTI in dogs as sporadic or acute.

CONDITION: ACUTE LOWER UTI/SPORADIC BACTERIAL CYSTITIS

TREATMENT

1. Empiric antimicrobial therapy is reasonable pending urine culture results although evidence in human medicine suggest the use of anti-inflammatory/pain relief achieves similar results in the first 24-48 hours.
2. The recommended first-line choices for empiric therapy are amoxicillin (or amoxicillin clavulanate) or trimethoprim-sulphonamide due to high susceptibility of most causative bacteria in Australian dogs.
3. The recommended treatment duration is 3-5 days.
4. The likelihood of adverse side effects associated with the use of trimethoprim-sulphonamide is low with short treatment courses, only reported in the literature in treatment courses greater than 6 weeks.

RECOMMENDED ANTIBIOTICS

First line:

Amoxicillin (11–15 mg/kg q8-12h PO)

Trimethoprim-sulphonamide (15 mg/kg q12h PO)

Second line:

Based on C&S testing

USAGE RECOMMENDATION

Recommended duration of therapy for sporadic bacterial UTIs is 3-5 days.

CONDITION: ACUTE LOWER UTI/SPORADIC BACTERIAL CYSTITIS

AIDAP TOP TIPS

Amoxicillin is a water-soluble drug that reaches high concentrations in the urine. For empirical (first line) therapy of canine sporadic cystitis there is no evidence for the need for ß-lactamase inhibitors such as clavulanic acid as high concentrations of amoxicillin are reached in the bladder.

Key references:

1. Weese et al. The Veterinary Journal. 2019; 247: 8–25.
2. Thompson MF, Litster AL, Platell JL et al. Vet J. 2011; 190: 22–27.
3. Weese JS, Blondeau JM, Boothe D et al. The Veterinary Journal. 2019; 247: 8–25.
4. Gottlieb S, Wigney DI, Martin PA, et al. Australian Veterinary Journal. 2008; 86(4): 147–152.
5. Scarborough R, Bailey K, Galgut B, et al. Antibiotics. 2020; 9: 924. doi10.3390/antibiotics9120924.

CONDITION: RECURRENT BACTERIAL CYSTITIS

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Recurrent bacterial cystitis is defined as three or more episodes in the preceding 12 months or two episodes in the preceding six months.

It may be the result of persistent infection or reinfection. It may be associated with an unresolved co-morbidity such as urolithiasis, endocrinopathies (hyperadrenocorticism, diabetes mellitus), congenital or acquired conformational abnormalities, bladder neoplasia or other disorders of growth, prostatic disease, or urinary retention. Therefore, a diagnostic plan that identifies likely potential comorbidities is required for successful resolution. Equally it may be the result of inadequate or inappropriate treatment of an acute bacterial cystitis due to an inappropriate dose or choice of antimicrobial and/or the presence of a more resistant organism.

While there has been an increase in the number of multidrug resistant (MDR) *Staphylococcus pseudintermedius, E. coli, Enterococcus faecalis, Corynebacterium urealyticum* and *Enterobacter* spp. isolated from canine urine in Australian veterinary diagnostic laboratories, they remain infrequent even in referral medicine settings.

CONDITION: RECURRENT BACTERIAL CYSTITIS

TESTS FOR DIAGNOSIS

1. Diagnosis of recurrent bacterial cystitis requires an appropriate history of repeated UTI together with lower urinary tract (LUT) signs with supporting evidence of UTI (epithelial cells, RBC, WBC and bacteria) on sediment examination by wet preparation (see instructions on 'Wet Prep" AMR Vet Collective | Continuing Education), or stained urine sediment (Gram or Diff-Quik®).

2. A full urinalysis is recommended for all dogs presenting with recurrent cystitis, using a urine sample collected by cystocentesis or sterile urinary catheter, where possible.

3. Urine C&S testing is important for the correct identification of the causative bacterium and the appropriate choice of antimicrobial agent.

4. A broader diagnostic plan may include diagnostic imaging, endocrine assays, full serum biochemistry, haematology, neurological exam, rectal and/or vulval exam may be required, as appropriate to the patient's clinical signs.

KEY ISSUES

01 Look for markers of underlying disease or bladder dysfunction/ abnormalities such as urolithiasis, prostatic enlargement, PU/PD, diabetes, and hyperadrenocorticism so that complications predisposing to infection can be addressed, reducing the risk of recurrent cystitis.

02 The presence of a comorbidity does not categorise the infection as recurrent but failure to address the comorbidity where possible may result in recurrent bacterial cystitis.

CONDITION: RECURRENT BACTERIAL CYSTITIS

TREATMENT

1. Empiric antimicrobial therapy is reasonable pending urine culture results although evidence in human medicine suggest the use of anti-inflammatory/pain relief achieves similar results in the first 24 to 48 hours.

2. **The recommended first-line choices for empiric therapy for recurrent bacterial cystitis are amoxicillin or trimethoprim-sulphonamide due to high susceptibility of most causative bacteria in Australian dogs.**

3. Urine C&S testing from an aseptically collected sample is important in recurrent bacterial cystitis as is the identification of complicating co-morbidities.

4. Treatment duration may be short (3-5 days) or longer (7-14 days) depending on the results of the broader diagnostic plan and assessment of the impact of any co-morbidities.

RECOMMENDED ANTIBIOTICS

First line:
(until C&S available)

Amoxicillin (11–15 mg/kg q8-12h PO)
Trimethoprim-sulphonamide (15 mg/kg q12h PO)

Second line:

Based on C&S testing

USAGE RECOMMENDATION

Short duration courses (3-5 days) may be considered for reinfection.

Longer courses (7-14 days) may be considered when persistent infections are likely.

Ensure duration, dose and choice of antimicrobial is reviewed following C&S testing and further diagnostic tests to ensure good penetration of an appropriate antimicrobial.

CONDITION: RECURRENT BACTERIAL CYSTITIS

AIDAP TOP TIPS

Identification of unresolved co-morbidity and/or antimicrobial-resistant bacteria is critical for the successful resolution of recurrent bacterial cystitis.

Key references:

1. Weese et al. The Veterinary Journal. 2019; 247: 8–25.
2. Briscoe K, Barrs V, Lindsay S, et al. Journal of Feline Medicine and Surgery. 2010; 12: 972-977.

CONDITION: PYELONEPHRITIS

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

The bacteria involved in pyelonephritis are the same as for bacterial cystitis, with *Escherichia coli* being the most frequent bacterium involved.

Pyelonephritis is defined as inflammation of the renal pelvis and parenchyma, most commonly the result of ascending bacterial infection or less frequently bacteraemia. Ascending infection can occur with or without overt clinical signs of lower urinary tract disease. Medullary infection extends from the renal pelvis due to poor blood supply and other factors.

Pyelonephritis can be acute or chronic and there is notably overlap in clinical signs. In acute pyelonephritis, clinical signs often include fever, pain on renal/abdominal palpation, variation in kidney size, anorexia, lethargy, vomiting, diarrhoea, uraemia, azotaemia, leucocytosis, pyuria, cylinduria, and/or sepsis, but clinicopathologic signs can be much less overt and required higher index of suspicion. Chronic pyelonephritis is likely underdiagnosed as it has greater variation in clinical signs but may lead to changes in kidney shape due to chronic inflammation and fibrosis. The bacteria involved in pyelonephritis are the same as for bacterial cystitis, with *Escherichia coli* being the most frequent bacterium involved.

CONDITION: PYELONEPHRITIS

TESTS FOR DIAGNOSIS

1. A diagnostic plan should include full serum biochemistry, haematology, endocrine testing, diagnostic imaging, as appropriate to the patient's clinical signs.
2. A full urinalysis is recommended for all dogs presenting with pyelonephritis, using a urine sample collected by cystocentesis or if possible, pyelocentesis.
3. Urine C&S testing is very important to guide the appropriate choice of antimicrobial agent.

KEY ISSUES

01 Look for markers of underlying disease such as urolithiasis, kidney disease, diabetes mellitus, as possible predisposing factors to infection and address the impact of infection rapidly.

02 Definitive diagnosis of both acute and chronic pyelonephritis can be challenging, with clinical signs varying from overt to covert, requiring thorough diagnostic investigation.

TREATMENT

1. Empirical treatment should commence immediately pending C&S testing given the serious potential consequences of delayed treatment.
2. The recommended first-line choices for empiric therapy for pyelonephritis in dogs are marbofloxacin or enrofloxacin, due to their lipophilic qualities, good penetration into renal interstitial tissue, and susceptibility of the main pathogens involved.
3. The identification of complicating co-morbidities is very important.
4. Treatment duration should be 10-14 days depending on the results of the broader diagnostic plan.
5. Re-examination and evaluation of the patient, including physical examination, serum biochemical assessment of renal analytes, and aerobic urine culture is highly recommended one week after the end of the antimicrobial course.

CONDITION: PYELONEPHRITIS

RECOMMENDED ANTIBIOTICS

First line:
(until C&S available)

Marbofloxacin (5.5 mg/kg q24h PO)
Enrofloxacin (5-20 mg/kg q24h PO)

Second line:

Based on C&S testing

USAGE RECOMMENDATION

Treatment duration should be 10-14 days depending on the results of the broader diagnostic plan.

Re-examination and evaluation of the patient including physical examination, serum biochemical assessment of renal analytes, and aerobic urine culture is highly recommended one week after the end of the antimicrobial course.

Ensure duration, dose and choice of antimicrobial is reviewed following C&S testing and further diagnostic tests.

AIDAP TOP TIPS

Pyelonephritis has a wide range of clinical signs with fever being the most consistent. A high index of suspicion is required to correctly diagnose pyelonephritis in more subtle presentations.

Key references:

1. Weese et al. The Veterinary Journal. 2019; 247: 8–25.
2. Bouillon et al. Journal of Veterinary Internal Medicine. 2018; 32(1): 249-259.

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Acute febrile illness in the dog may be infectious, immune-mediated, or attributable to malignancy.

Fever is less common due to infectious agents in the dog than in the cat, where occult fight injuries and other bacterial and viral infections are common. Cats may respond to empiric antimicrobial therapy when presented with fever and no localising signs, whereas dogs less commonly do.

In the dog, bacterial infections causing fever include endocarditis, peritonitis, pneumonia, prostatitis, pyothorax, pyometra, and abscesses (which may be in body cavities). In certain breeds such as German Shepherds, disseminated fungal disease should also be considered.

Immune-mediated diseases accounted for 22-36% of febrile diseases of the dog in one study. Diseases such as corticosteroid-responsive meningitis (also known as aseptic suppurative meningitis), immune-mediated polyarthritis, metaphyseal osteopathy (also known as hypertrophic osteodystrophy), and panosteitis should also be considered depending on the age and presenting signs. Recently described immune-mediated lymphadenopathies may have clinical similarities to abscessed lymph nodes. Culture of lymph nodes may be consistently negative in such cases but a search for infectious aetiologies is essential. Pancreatitis is another common cause of fever in the dog. In some dogs, unexplained neutropenia may occur due to immune-mediated mechanisms, further complicating the diagnosis. Therefore, a thorough search for an infectious cause should be undertaken, but with the knowledge that some dogs may have immune-mediated disease as the primary cause of fever.

In young dogs, acute febrile diseases may be infectious and include diseases such as parvoviral enteritis or acute bacterial enteritis (*Salmonella, Campylobacter,* some *E. coli*). This is discussed in the guidelines on the treatment of diarrhoea. In these scenarios, antibiotic selection and efficacy is problematic.

CONDITION: ACUTE FEBRILE ILLNESS

TESTS FOR DIAGNOSIS

Ideally samples for bacterial culture should be collected prior to empiric antibiotic use. Blood and joint fluid culture require enrichment culture bottles to facilitate bacterial growth. Many laboratories will provide culture bottles to veterinarians, so they are immediately on hand when such cases are presented.

KEY ISSUES

01 A thorough physical examination is mandatory. Pay particular attention to identifying regions of pain such as the neck or joints. New cardiac murmurs may signal development of bacterial endocarditis. Look at the eyes for uveitis. Retinal examination can occasionally be rewarding.

02 Haematology, biochemistry, urinalysis, and urine culture are required, unless physical findings point to a specific anatomical region as a potential cause of the infection.

03 Diagnostic imaging of the thorax/abdomen – radiographs, ultrasonography and cross-sectional imaging are all useful.

04 Blood and urine cultures may be appropriate.

05 Specific searches for infectious agents/immune diseases may be required such as cardiac ultrasound for endocarditis, CSF analysis, joint fluid analysis. Culture or PCR may be used for the detection of *Bartonella* spp, *Ehrlichia canis* and other tick-borne agents, especially in areas where such diseases are endemic.

06 In young dogs with febrile disease and lameness, radiographs of long bones are required to investigate for metaphyseal osteopathy, panosteitis and fungal osteomyelitis.

CONDITION: ACUTE FEBRILE ILLNESS

TREATMENT

- If a high index of suspicion is present for infection then antibiotic selection should be based on likely pathogenic bacteria and a bactericidal, broad-spectrum antibiotic should be selected. If overwhelming sepsis is suspected, 'four-quadrant therapy' may be instituted after collection of appropriate samples for testing, with de-escalation of antimicrobials used once results of culture and susceptibility are available.

RECOMMENDED ANTIBIOTICS

First line:

Amoxicillin clavulanic acid (20 mg/kg q12h SC or PO)

Second line:

Four-quadrant therapy IV if overwhelming sepsis is suspected:

Amoxicillin (20 mg/kg q6-8h) and

Metronidazole (10 mg/kg q12h) and

Gentamicin (6 mg/kg q24h, in a well hydrated patient) or Enrofloxacin (5-20 mg/kg q24h)

USAGE RECOMMENDATION

Antibiotic use may interfere with subsequent diagnostics and should be reserved for severe cases with a high index of suspicion for an infectious cause. Every attempt should be made to collect suitable samples prior to commencing treatment.

CONDITION: ACUTE FEBRILE ILLNESS

AIDAP TOP TIPS

Acute abdominal inflammation is a special subcategory of these where multiple species of bacteria may be identified. Therefore, if there is danger of intestinal perforation or translocation of enteric organisms, then the use of 'four-quadrant' therapy may be required until appropriate testing and surgical therapy is undertaken.

Key references:

1. Dunn KJ and Dunn JK. J Small Anim Pract. 1998; 39(12): 574–580.
2. Battersby IA, Murphy KF, Tasker S, et al. J Small Anim Pract. 2006; 47(7): 370–376.
3. Ribas Latre, J et al. J Small Anim Pract. 2019;60(5):280-290.
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CONDITION: GASTROINTESTINAL INFECTIONS

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Infectious agents are an uncommon cause of vomiting in dogs when not accompanied by diarrhoea.

Gastrointestinal disease in dogs is a frequent problem. In most instances, the presenting clinical signs are either vomiting, diarrhoea or both. Infectious agents are an uncommon cause of vomiting in dogs when not accompanied by diarrhoea. The possible exception being gastric *Helicobacter* infection, although *Helicobacter* spp. are normal inhabitants of the gastric mucosa, confounding the issue of determining if *Helicobacter* is the cause of chronic vomiting in dogs. In tropical and subtropical environments containing fresh-water lakes and ponds, the motile oomycete *Pythium insidiosum* may cause gastric or intestinal wall thickening and mass formation.

Diarrhoea with or without vomiting is very common. The nature of the diarrhoea may vary with the underlying aetiology and the duration may be acute (<5 days) or chronic (>5 days). Many acute cases of diarrhoea may be non-infectious in nature or self-limiting and these do not require antibiotic therapy.

Despite the paucity of evidence for bacterial infections being a common cause of diarrhoea in the dog, widespread empirical use of antimicrobials is common. This practice will alter the normal microbiome of the dog, especially the anaerobes which predominate, allowing pathogenic non-residents such as *Salmonella* an ecological niche to further proliferate. It may promote antibiotic resistance by selecting for resistance and sharing of resistance genes between bacteria.

When assessing a patient with clinical signs of gastrointestinal disease, the attending clinician should consider the history, duration of illness, signalment, dietary factors, and clinical severity prior to selecting various diagnostic tests for infectious agents. In most scenarios, diagnostic testing should be used to determine an aetiology prior to any empirical use of antimicrobial therapy.

CONDITION: GASTROINTESTINAL INFECTIONS

Faecal analysis (including a parvo antigen test, faecal flotation +/- faecal smear), ELISA or PCR testing, testing for parasites (nematodes, *Giardia*, coccidia) and a stool bacterial culture should be considered where a diagnosis of infectious agent needs to be confirmed.

Anaerobic and aerobic culture of the faeces may be performed but selective media may be required to isolate a pathogenic bacterium (for example *Salmonella* or *Campylobacter* spp). Diagnosis of *Campylobacter* or *Salmonella* involves detection in faeces together with appropriate clinical signs. Clinically silent colonisation can occur, perhaps more commonly in dogs fed raw food diets. Detection of the organism in faeces of an animal with clinical signs consistent with salmonellosis provides a good presumptive diagnosis but is not definitive. Isolation of *Salmonella* from blood or other typically sterile sites in the presence of disease is diagnostic.

Faeces from dogs may be tested for enterotoxin genes of *C. perfringens* by quantitative PCR. However, the association of the CPE toxin and enterotoxin A with disease is uncertain, as normal animals may have high levels of toxin DNA present also.

Recently a new *C. perfringens* toxin gene netF has shown correlation with the presence of bloody diarrhoea in dogs.

Quantitative PCR is also available for viral pathogens such as parvovirus and coronavirus.

Fluorescent *in situ* hybridisation (FISH) testing has been used for identification of enteroinvasive *E. coli* in biopsy specimens obtained in granulomatous colitis.

There are four species of coccidia in dogs. These are *Cystoisospora canis, C. ohioensis, C. burrowsi, and C. neorivolta*. Coccidiosis is more common in young animals and animals in unhygienic environments. Co-infections with other infectious agents are common. Clinical signs may be severe with depression, weakness, loss of appetite, diarrhoea, and dehydration the most common findings. Diagnosis is by identification of the unsporulated oocysts on faecal flotation.

CANINE CHRONIC ENTEROPATHIES

Canine chronic enteropathies (chronic diarrhoea) are currently subdivided into food-responsive enteropathy, antibiotic responsive diarrhoea (ARD) and steroid-responsive enteropathy (equivalent to idiopathic inflammatory bowel disease). The investigation of chronic enteropathies requires exclusion of aetiologies such as known infectious disease agents and neoplasia and is based on the response to empirical treatment trials (diets, antibiotics, and corticosteroids). It is recognised that a subset of dogs without identifiable pathogens may respond to antibiotics (ARD). Typically, these dogs have had multiple investigations including, blood tests, intestinal biopsies, novel food/hypoallergenic food trials and treatment with fenbendazole for occult *Giardia* or helminth infection. In such cases, an antibiotic trial with tylosin has been successful. The mode of action of tylosin, a macrolide, in ARD is unknown, but possibly corrects microbiome dysbiosis or has an unknown anti-inflammatory effect, or possibly both.

CONDITION: GASTROINTESTINAL INFECTIONS

THE FOLLOWING INFECTIOUS AGENTS HAVE BEEN COMMONLY IMPLICATED AS CAUSING DIARRHOEA IN THE DOG

Viral: Canine parvovirus, canine coronavirus (other viruses have also been implicated such as rotavirus, norovirus, and circovirus).

Protozoal: *Giardia* spp., *Cryptosporidium, Coccidiosis.*

Algal: *Prototheca* spp.

Bacterial: *Salmonella* spp., *Campylobacter jejuni,* enterotoxigenic *Clostridium perfringens, Clostridium difficile,* some *E. coli* (enteropathogenic, enteroinvasive, enterotoxigenic, enterohaemorrhagic biotypes).

Parasitic helminths: *Toxocara canis, Ancylostoma caninum, Trichuris vulpis.*

SPECIFIC INDICATIONS FOR THE USE OF ANTIMICROBIALS IN DIARRHOEA:

1. Identification of a bacterial agent as listed below (C&S, qPCR). Note many cases of *Campylobacter* or *Salmonella* may be self-limiting and do not require antibiotic therapy.
2. Granulomatous colitis in Boxer dogs and French Bulldogs due to the causal association with enteroinvasive *E. coli.*
3. Chronic diarrhoea suspected to be ARD where other measures such as food trials have been explored.
4. Diarrhoea characterised as severe with likely compromise of the epithelium to the extent that bacterial translocation is likely, as in canine parvovirus.
5. Diarrhoea with marked neutropenia. Typically, neutrophils less than 1 x 10^9/L.
6. Acute haemorrhagic diarrhoea with strong suspicion of *Clostridial perfringens* enterotoxaemia (PCR positivity with associated history and clinical signs).

CONDITION: GASTROINTESTINAL INFECTIONS

TESTS FOR DIAGNOSIS

Selection of diagnostic methods is based on history, physical examination findings, and nature of the diarrhoea. Duration of clinical signs (acute versus chronic) may also influence choice of testing regimes.

1. Wet slide preparation of faecal material and microscopic evaluation for *Giardia* trophozoites.
2. Faecal flotation for identification of helminths, *Giardia* cysts, coccidia, possibly *Cryptosporidium*.
3. Rectal scrapings/faecal smears and cytology examination may be used in some instances for presence of less common pathogens including spirochaetes and *Prototheca*.
4. Antigen testing of faeces for canine parvovirus, canine coronavirus, *Giardia*.
5. Multi-agent PCR panels examinations may be used in cases of chronic diarrhoea. It is important to recognise that some agents may be detected but are associated with a "carrier state". Positive results must be related to clinical signs prior to treatment being instituted.
6. Faeces placed in a sterile container for specific culture of specific pathogens on specified media. C&S testing is often required for correct choice of antimicrobial.
7. Enteric biopsy may identify *Cryptosporidium* in some infrequent cases of chronic diarrhoea.
8. Colonic epithelial pinch biopsy for FISH testing for enteroinvasive *E. coli* in suspected granulomatous colitis, also for diagnosis of *Prototheca* and *Pythium*.

KEY ISSUES

01 Many cases of acute diarrhoea in dogs are self-limiting and do not require antimicrobial therapy.

02 If antibiotic therapy is to be used, diagnostic samples should be collected prior to starting therapy.

03 Diarrhoea with marked neutropenia or potential for translocation of bacteria across severely damaged intestinal epithelium may require prophylaxis of bacterial sepsis, however studies supporting improved patient survival have a low evidence base.

CONDITION: GASTROINTESTINAL INFECTIONS

TREATMENT

Where dehydration is evident with diarrhoea, hospitalisation for treatment with intravenous fluids is strongly advised.

RECOMMENDED ANTIBIOTICS

Acute non-life-threatening diarrhoea with no evidence of patient dehydration or haematochezia:

No antibiotic is required.

Bismuth subsalicylate (Peptosyl) has some antibacterial and anti-diarrhoeal effects by binding of toxin produced by *E. coli*. Bismuth may have some antibacterial properties.

Acute diarrhoea with marked neutropenia or strong evidence of sepsis:

First line:
Amoxicillin (20 mg/kg q8h IV) and
Enrofloxacin (5-20 mg/kg q24h SC)

Second line:
Amoxicillin (20 mg/kg q8h IV) and
Gentamicin (6–10 mg/kg q24h SC)

USAGE RECOMMENDATION

There is no evidence-base regarding treatment duration, but the panel recommends at least 4 days, and ideally 7–14 days.

For ARD. Treatment is initially for 4 weeks. If relapse occurs, then treatment is indefinite.

CONDITION: GASTROINTESTINAL INFECTIONS

RECOMMENDED ANTIBIOTICS

***Clostridium perfringens* overgrowth with clinical signs:**

First line:
Metronidazole (15 mg/kg q12h PO)

Second line:
Amoxicillin-clavulanate (20 mg/kg q12h PO)

Granulomatous colitis:

First line:
Enrofloxacin (10 mg/kg q24h PO for 6-10 weeks) or
Marbofloxacin (10 mg/kg q24h PO for 6-10 weeks)
(Resistance has been reported and if encountered then C&S is required)

Antibiotic responsive diarrhoea (ARD):

First line:
Tylosin (20 mg/kg q12h PO)

Second line:
Doxycycline (10 mg/kg q12h PO)

Specific infectious agents if conclusively identified as the cause of the diarrhoea:

Salmonella:
Based on C&S

CONDITION: GASTROINTESTINAL INFECTIONS

RECOMMENDED ANTIBIOTICS

Campylobacter:

Most cases are mild and self-limiting and rely on normal microbiota to assist in recovery, so treatment is only used in severe cases or when issues of zoonosis in immune compromised households arise.

First line:
Clarithromycin (7.5 mg/kg q12h PO)

Second line:
Based on C&S
Enrofloxacin (5 mg/kg q12h PO for 3-5 days)

Helicobacter:

If the clinician is convinced that *Helicobacter* is the cause of chronic vomiting, which would require biopsies of the gastric mucosa that show fibrosis, lymphoid inflammation that is associated with large numbers of *Helicobacter*.

First line:
Amoxicillin-clavulanate (12.5 mg/kg q12h PO) and
Metronidazole (15 mg/kg q12h PO)

Second line:
Clarithromycin (7.5 mg kg q12h PO) and
Metronidazole (15 mg/kg 15 BID PO)

USAGE RECOMMENDATION

Clarythromycin is better tolerated than erythromycin which may cause vomiting. Clarithromycin is not registered for use in animals. It should not be used off-label except in exceptional circumstances for individual animals.

For *Helicobacter:*

Add acid inhibition with omeprazole 10 mg per dog q24h PO for 14 days.

CONDITION: GASTROINTESTINAL INFECTIONS

RECOMMENDED ANTIBIOTICS	USAGE RECOMMENDATION
Giardia:	
First line: Fenbendazole (50 mg/kg q24h PO for 3-5 days)	Febantel is a prodrug of Fenbendazole.
Second line: Febantel (25 mg/kg q24h PO for 3 days)	Prolonged metronidazole therapy can cause neurological signs.
Third line: Metronidazole (15 mg/kg q12h PO for 7 days)	
Coccidia:	
First line: Toltrazuril (10 mg/kg q24h PO)	Toltrazuril (Baycox®) is effective in reducing oocysts and clinical signs.
Cryptosporidium:	
Most cases are mild and self-limiting.	
The following drugs have been used with some success in cases where animals have persistent diarrhoea with oocyst shedding:	
First line: Azithromycin (5-10 mg/kg q12h PO for 5-7 days)	Azithromycin is not registered for use in animals. It should not be used off-label except in exceptional circumstances for individual animals.
Pythium insidiosum:	
First line: Itraconazole (8-12 mg/kg q24h PO) and Terbinafine (12 mg/kg q24h PO)	Itraconazole and terbinafine have been reported successful in 3 dogs when used with prednisolone at 0.5 mg/kg q12h PO for 2 weeks then 0.5 mg/kg q24h PO for 4 weeks. The off-label use of the agricultural fungicide Mefenoxam (4 mg/kg q12h PO) has been used in one case report successfully in combination with itraconazole and terbinafine.

CONDITION: GASTROINTESTINAL INFECTIONS

AIDAP TOP TIPS

1. Always take samples for diagnostic testing prior to treating with antimicrobials.

2. Antimicrobials should only be used for animals definitively diagnosed with a specific pathogen that is not known to create self-limiting disease or those that are at risk of life-threatening sepsis.

Key references:

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BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

In surgical practice, antimicrobials may be used for treatment of a pre-existing infection, or for prevention of an infection resulting from the procedure, known as antimicrobial prophylaxis.

For pre-existing infections, C&S should be performed prior to the administration of any antimicrobial.

In the case of a potential break in sterile technique, the surgeon must weigh the risk of an infection against the risk of antimicrobial resistance development.

Regardless of the timing of infection, surgical site infections (SSI) represent significant cost, morbidity and even mortality in veterinary medicine. The rate of SSI depends upon the type of surgery but may be reduced by following the principles of surgical asepsis and meticulous use of aseptic techniques.

When antimicrobials are used appropriately in surgical prophylaxis, the antimicrobial chosen is often empirical, however the minimum inhibitory concentration (MIC) of the drug for the most likely contaminant/s should be reached at the surgery site prior to the first incision and should remain at or above the MIC for the duration of surgery.

Surgery is commonly classified as:

Clean - no infection present and not likely to become contaminated.

Clean-contaminated - surgery of the respiratory, gastrointestinal, genitourinary tracts without likely contamination.

Contaminated/dirty - infection already present at surgical site or contamination likely during the procedure.

The reported rates for SSI are approximately 5% for clean surgeries, 12% for clean-contaminated surgeries and 10% for contaminated/dirty surgeries.

CONDITION: USE OF ANTIMICROBIALS IN SURGERY

Clean surgical procedures (for example, routine desexing or neurological procedures of short duration) do not require antimicrobial prophylaxis in animals without risk factors; antisepsis and aseptic technique reduces the risk of infection to negligible levels.

Appropriately administered antimicrobials can reduce the risk of SSI by 6-7 fold. Improper selection of antimicrobials or incorrect administration results in increased SSI and the development of antimicrobial-resistant organisms.

Clean surgical procedures (for example, routine desexing or neurological procedures of short duration) do not require antimicrobial prophylaxis in animals without risk factors; antisepsis and aseptic technique reduces the risk of infection to negligible levels.

Antimicrobial prophylaxis during the procedure and for up to 24 hours following the surgery is indicated in animals undergoing the following:

Clean surgical procedures with risk factors such as longer duration surgery time or American Society of Anesthesiologists (ASA) patient classifications 3, 4, or 5.

Clean-contaminated surgeries with implant placement.

Contaminated/dirty surgeries.

Generally, in cases with the following: increased numbers of people present in the operating room; prolonged anaesthesia time; presence of a drain; or administration of propofol.

This reduces SSI to acceptable levels of 4.2-6.3%. Even in surgeries where SSI rates have been reported as high as 21%, no definitive evidence has been found to support the use of antimicrobials beyond 24 hours postoperatively. Risk factors indicating postoperative use of antimicrobials beyond 24 hours postoperatively include a dirty surgical site, increasing body weight (dogs >50 kg), duration of postoperative intensive care unit stay, extremes of age, morbid obesity, removal of hair greater than 4 hours preoperatively, hypothermia, and hypotension.

Risk factors for development of SSI with multiple drug resistance include the type of bacteria present (Enterobacteriales, *Salmonella*, methicillin-resistant *Staphylococcus pseudintermedius*), feeding a homemade diet or feeding raw food to the animal.

CONDITION: USE OF ANTIMICROBIALS IN SURGERY

Even in surgeries where SSI rates have been reported as high as 21%, no definitive evidence has been found to support the use of antimicrobials beyond 24 hours postoperatively.

When empirically choosing an antimicrobial, a narrow spectrum of activity is recommended to preserve the animal's normal microbiota and reduce the development of drug resistance.

The drug levels at the surgical site must be maintained at greater than MIC for the expected pathogens throughout the surgery, therefore repeat administration at intervals of 1-2 times the elimination half-life of the drug is recommended.

The antimicrobial must be present in the tissue prior to commencement of surgery, and therefore should be administered at least 30 minutes prior to surgical incision, but to reduce development of MDR and SSI, no earlier than 60 minutes prior to surgical incision. This means there is a 30-minute window of appropriate timing for antimicrobial administration.

SCENARIO BASED DECISIONS ON ANTIBIOTIC USE:

Antimicrobial prophylaxis is not a substitute for aseptic preparation of the patient, staff, facilities, or equipment.

In animals at risk of carrying MDR organisms such as those living in a household with humans carrying resistant organisms, animals fed homecooked or raw diets, animals with distant infections or recent antibiotic exposure, prior culture of nasal or aural tissues is warranted since such animals are at increased risk of SSI following surgery.

Selection of antimicrobials to be used prophylactically should be based upon the procedure, potential pathogens, and risk factors of the animal and surgical environment, such as the number of people in the operating room.

CONDITION: USE OF ANTIMICROBIALS IN SURGERY

TIMING OF ANTIMICROBIAL PROPHYLAXIS

The following recommendations are the standard of care in humans (National Surgical Infection Prevention Project):

1. Parenteral antimicrobials administered 30-60 minutes prior to surgical incision.
2. Repeat administration every 1–2 elimination half-lives during the procedure.
3. Discontinuation of prophylactic antimicrobials is recommended less than 24 hours following conclusion of surgery unless patient or surgical risk factors determine postoperative use is indicated.

KEY ISSUES

Numerous studies have been conducted on the use of antibiotics in surgery using animal models for human medical purposes. Studies specific to dogs and cats in veterinary practice recommend the following:

01 The success of antimicrobial prophylaxis at reducing surgical site infections is affected by the surgical site, likely pathogens present, pathogens most likely to cause a surgical site infection, risk factors present in the animal, and adherence to strict aseptic preparation and technique.

02 Antimicrobials administered following surgery are ineffective at reducing the risk of SSI and may increase the risk of multidrug-resistant (MDR) infections.

03 The risk of SSI doubles approximately every 70 minutes of surgery time and increases by 30% for each additional hour of anaesthesia time. Minimally invasive surgical procedures reduce the risk of SSI.

04 The likelihood of SSI increases with the number of people present in the operating theatre, increased hospitalisation postoperatively, and with increasing contamination of the surgical site.

CONDITION: USE OF ANTIMICROBIALS IN SURGERY

ANTIBIOTICS RECOMMENDED

Clean surgical procedures:

No antimicrobial is required.

Surgical procedures where contamination is associated with severe consequences, but infection is not clearly established:

Elective Orthopaedic surgery:
Cefazolin (30 mg/kg IV given every 90 minutes until wound closure)

Gastric, urogenital, and small intestinal surgery:
Cefazolin (30 mg/kg IV)

Large intestinal surgery:
Cefazolin (30 mg/kg IV)
Cefoxitin (30 mg/kg IV)

Pyometra (contained, no leakage):
Cefazolin (30 mg/kg IV)
Amoxicillin (20 mg/kg IV)

Abdominal surgery:
Cefazolin (30 mg/kg IV)
Amoxicillin (20 mg/kg IV)

USAGE RECOMMENDATIONS

Clean surgical procedures for which antimicrobial use is unnecessary:

1. Ovariohysterectomy.
2. Castration.
3. Removal of skin masses (lipomas for example).
4. Dental procedures (scaling and polishing) with minimal periodontal disease or risk of bacteraemia (see other chapters in this guideline on dentistry).
5. Clean surgical procedures without implant placement and procedures <70 minutes duration such as some neurological surgeries.

In all cases antibiotics are administered 30-60 minutes prior to surgery and repeated at 1-2 elimination half-lives during the procedure.

For elective orthopaedic surgery (total hip replacement, cruciate ligament surgery TPLO, TTA, others that involve cutting bone, or use of surgical implants), the anticipated pathogen is *Staphylococcus* spp. First generation cephalosporins can be continued parenterally every 8 hours for up to 24 hours postoperatively.

For upper gastrointestinal surgery, anticipated pathogens are Gram-positive cocci and Gram-negative bacilli.

For lower gastrointestinal surgery, anticipated pathogens are Gram-negative bacilli, enterococci and anaerobes.

If there is a ruptured gastrointestinal tract prior to surgery, use an extended Gram-negative antibiotic treatment in addition to ampicillin, such as a fluoroquinolone or gentamicin.

For pyometra (contained at surgery), anticipated pathogens are *Escherichia coli, Streptococcus* spp. and anaerobes.

For abdominal surgery (splenectomy, liver lobectomy) without contamination, anticipated pathogen is *Staphylococcus* spp.

CONDITION: USE OF ANTIMICROBIALS IN SURGERY

RECOMMENDED ANTIBIOTICS

Contaminated surgery where infection is already apparent or likely to be present:

Abscesses, hepatobiliary surgery, removal of organs, marsupialisation and drain insertions.

INITIAL THERAPY:

First line:

Cefazolin (30 mg/kg IV) or
Amoxicillin (20 mg/kg IV) and
Gentamicin (6 mg/kg SC)

Second line:

Cefoxitin (30 mg/kg IV)

ONGOING THERAPY:

Based on C&S. Narrow spectrum if possible.

USAGE RECOMMENDATIONS

Gentamicin is added and given prior to surgery if enteric or Gram-negative bacteria possibly involved.

Cefoxitin is suggested if anaerobic infection likely and possibly resistant to amoxicillin or cefazolin. For example, in hepatobiliary, urogenital, or lower GI surgery.

For head and neck surgery, clindamycin or cefazolin are appropriate choices, and anticipated pathogens are *Staphylococcus* spp., *Streptococcus* spp., anaerobes.

For abscesses such as cat fight abscess and anal sac abscess where drained, refer to the chapters in this guideline relating to soft tissue infections.

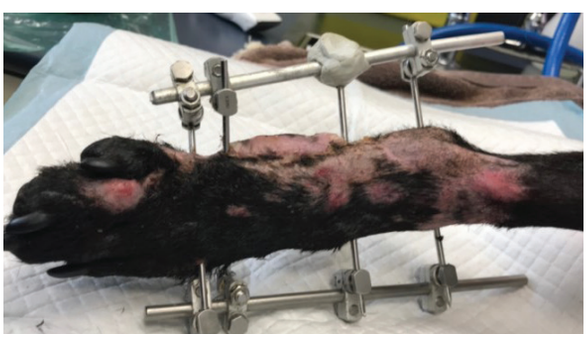
For hepatobiliary, anticipated pathogens are *Clostridium* spp., Gram-negative bacilli, anaerobes.

CONDITION: USE OF ANTIMICROBIALS IN SURGERY

AIDAP TOP TIPS

1. The risk of SSI must be weighed against the risk of MDR organism establishment when determining whether to administer prophylactic antimicrobials to surgical patients.
2. Knowledge of the surgery type, the most common organisms from SSI following the surgery, and the risk factors for SSI development will determine what type of antimicrobial prophylaxis should be administered.
3. Strict surgical personnel, facilities, and equipment aseptic preparation as well as adherence to aseptic technique in the surgery reduce the risk of SSI and the requirement for antimicrobial prophylaxis in many surgeries.
4. In surgical procedures where antimicrobial prophylaxis is used, timing of administration between 30 and 60 minutes prior to procedure commencement is vital.

Degloving wound on a dog which was treated successfully with C&S of wound as it was healing to determine correct antibiotic.



Post-op degloving wound on a dog which was treated successfully with C&S of wound as it was healing to determine correct antibiotic.

Photos courtesy of Dr Wendy Baltzer.

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CONDITION: SURFACE BACTERIAL INFECTIONS – INTERTRIGO (LIP FOLD, TAIL FOLD)

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Most bacterial skin infections involve Gram-positive organisms and particularly the coagulase-positive staphylococci.

Bacterial skin infections can be classified based on lesion depth and distribution pattern.

Surface pyodermas involve the epidermis. As infection does not cross the basement membrane, the dermis remains intact. Most bacterial skin infections involve Gram-positive organisms and particularly the coagulase-positive *staphylococci.* The most common of these is *Staphylococcus pseudintermedius* which represents over 90% of infections in dogs. Infections with *S. aureus* are relatively uncommon. *S. schleiferi coagulans* and *S. schleiferi schleiferi*) are rarer causes of infection but are found particularly in otitis externa and in certain geographic regions.

Coagulase-negative staphylococci such as *S. sciuri*, *S. xylosus* and *S. felis* are rarely involved in disease despite being common skin commensals.

They are normally only involved in disease if immunity is greatly reduced or when implants are used. Gram-negative bacteria are sometimes found in pyoderma, particularly when lesions are moist. Organisms such as *Proteus* spp. and coliforms may be secondary invaders but fail to persist when more significant pathogens are removed. *Pseudomonas aeruginosa* is more serious and requires specific therapy.

The most common surface pyodermas are skin fold-pyoderma (intertrigo) and pyotraumatic dermatitis. These clinical entities are only reported in dogs. These are surface pyodermas with secondary bacterial involvement and typically respond to topical therapy, but recurrence is frequent if a primary disease process cannot be identified and controlled.

CONDITION: SURFACE BACTERIAL INFECTIONS – INTERTRIGO (LIP FOLD, TAIL FOLD)

TESTS FOR DIAGNOSIS

- Perform an impression smear for cytological evaluation.
- Examine under the microscope using immersion oil (x100) and evaluate for the presence of healthy or degenerate neutrophils (swollen and pale nuclei) and extracellular cocci and bacilli and intracellular phagocytosed bacteria. If there are abundant bacilli, and if there is no response to empirical antibacterial therapy then submit a swab of the exudate for C&S. Collecting a skin biopsy to rule out immune-mediated disease may be indicated if the lesions fail to respond to appropriate topical and systemic antimicrobial therapy, however these diseases are rare.

KEY STEPS

01 Identify bacterial overgrowth and/or infection via surface cytology.

02 Commence trial treatment with appropriate topical and/or systemic antibiotic.

03 Consider bacterial C&S testing if failure to respond to therapy.

04 Correct underlying anatomical abnormality.

SAMPLE COLLECTION TECHNIQUE FOR C&S TESTING (SUPERFICIAL AND SURFACE INFECTIONS):

NO surface preparation is required.

PUSTULE:
Use a 25 g needle to gently pierce the pustule. Collect purulent material from within the pustule with sterile swab and submit in transport media.

COLLARETTE/CRUST:
Gently peel the crust from the skin surface. Rub sterile swab on the under-surface of the crust and skin surface. Repeat from 4–6 lesions to obtain a representative sample from body. Submit in transport media.

SKIN FOLDS:
Rub sterile swab deep in affected folds. Submit in transport media.

CONDITION: SURFACE BACTERIAL INFECTIONS – INTERTRIGO (LIP FOLD, TAIL FOLD)

TREATMENT

There is very little evidence-based data concerning the treatment of surface pyoderma in dogs.

For uncomplicated surface bacterial pyoderma, we recommend topical antimicrobial therapy in lieu of systemic antimicrobial therapy. Fusidic acid (Fucidin®) is bacteriostatic and active against all *Staphylococcus* spp. including those that are penicillin-resistant, and its lipophilic nature allows good tissue penetration.

Shampoo therapy is a suitable topical agent for the treatment of pyoderma and benefits most dogs by removing tissue debris, hydrating the skin, and reducing or eliminating the surface bacterial population. Chlorhexidine is usually the active ingredient of choice for superficial bacterial infections.

Hydrocortisone (topical corticosteroid) in combination with neomycin is not favoured by dermatologists due to the high rates of contact sensitisation associated with neomycin.

RECOMMENDED ANTIBIOTICS

First line:
Cephalexin (20 mg/kg q12h PO) or (30 mg/kg q24h PO)

Second line:
Clindamycin (11 mg/kg q12h PO)

Third line:
Based on C&S.

See section on methicillin-resistant *Staphylococcus pseudintermedius*.

Cefovecin (8 mg/kg q14d for 21-28 days) is suitable for cases where there are serious concerns of compliance, or there are difficulties with oral dosing.

USAGE RECOMMENDATIONS

Topical therapy

Application rate of topical agents is 1–2 times daily until cure and then twice a week as maintenance.

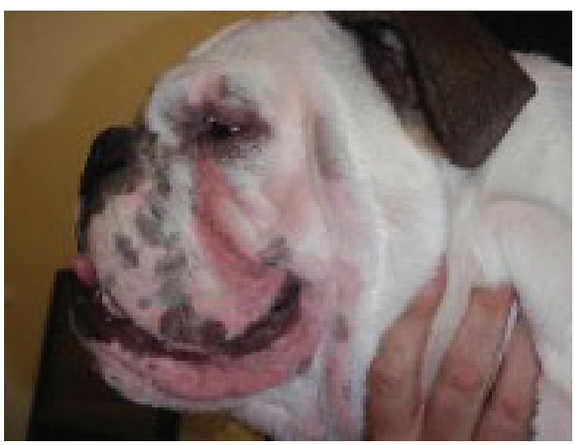
For cephalexin, vomiting may occur at higher doses. Dose and interval may need to be adjusted.

Fluoroquinolones should be avoided for staphylococcal pyodermas unless bacterial resistance to first or second-line antimicrobials has been demonstrated and susceptibility has been established by C&S.

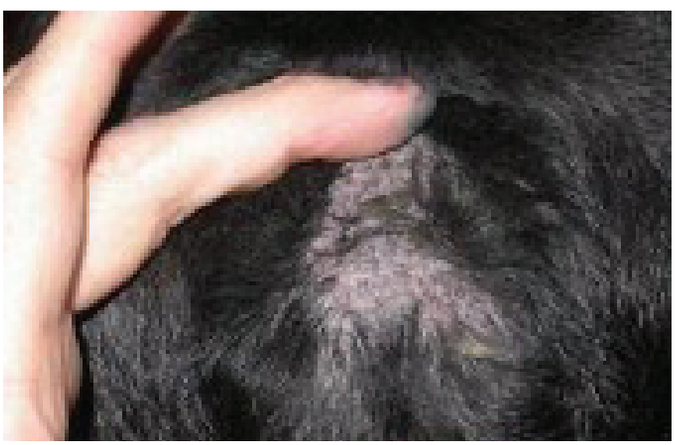
CONDITION: SURFACE BACTERIAL INFECTIONS – INTERTRIGO (LIP FOLD, TAIL FOLD)

AIDAP TOP TIPS

1. These lesions often respond well to a combination of gentle wiping with 1% chlorhexidine impregnated plagette or 'wipe' and application of topical therapy; if bacilli are detected on cytologic evaluation, then topical silver sulfadiazine cream can be useful.



Facial fold pyoderma.



Tail fold pyoderma.

Photographs courtesy of Dr Mandy Burrows & Dr Mike Shipstone.

Key references:

1. Fitzgerald JR. Vet Dermatol. 2009; 20(5–6): 490–495.
2. Gemma C. Ma, Kate A. Worthing, Michael P. Ward & Jacqueline M. Norris. Microbial Ecology. 2020; 79: 164–174. https://doi.org/10.1007/s00248-019-01382-y.

CONDITION: SUPERFICIAL BACTERIAL INFECTIONS - MUCOCUTANEOUS PYODERMA, BACTERIAL FOLLICULITIS, BACTERIAL OVERGROWTH

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Superficial bacterial infections are common and include superficial bacterial folliculitis, bacterial overgrowth and mucocutaneous pyoderma.

Superficial bacterial folliculitis is a bacterial skin infection affecting the hair follicle. *Staphylococcus pseudintermedius* is the primary pathogen. Most bacterial folliculitis is seen secondary to coexistent disease or other predisposing factors, such as atopic dermatitis, flea allergy dermatitis, food allergy, hypothyroidism, spontaneous and iatrogenic hyperglucocorticoidism, primary keratinisation disorders and genodermatoses such as colour dilution alopecia. Other predisposing factors include pruritus of any origin, defects in the immune system and poor grooming.

Distribution: axillae, inguinal region, dorsal trunk, interdigital.

Skin lesions: follicular pustules with a central protruding hair (unless the hair has been shed). Pustules are fragile and transient and rupture, forming crusted papules. After pustules rupture, collarettes may form with peripheral scaling and post inflammatory hyperpigmentation. Alopecia is variable but common and distinct, circular patches of transient alopecia may form around previously affected hair follicles and give the coat a 'moth-eaten' appearance, particularly in short-coated dogs.

Mucocutaneous pyoderma (MCP) affects mucocutaneous junctions of dogs, most commonly the lips and perioral skin. German Shepherds are predisposed. The pathogenesis is unknown but the response to antimicrobial therapy supports the role of bacterial infection in the aetiology, however the response is variable, and relapses are common. The predisposing or initiating factors are not known and mucocutaneous pyoderma may have a more complex immunologic pathogenesis.

Distribution: lips and perioral skin, and less commonly nasal planum, nares, eyelids, vulva, prepuce, and anus.

Clinical features: erythema and swelling of the lips; erosion and ulceration with adherent crusting may occur in more severe cases. Depigmentation of the lips can occur. The adjacent philtrum may also be affected. The lesions are sometimes painful, and the dogs rub areas and resent examination and palpation.

CONDITION: SUPERFICIAL BACTERIAL INFECTIONS - MUCOCUTANEOUS PYODERMA, BACTERIAL FOLLICULITIS, BACTERIAL OVERGROWTH

TESTS FOR DIAGNOSIS

- The diagnosis of superficial bacterial folliculitis and bacterial overgrowth is usually straightforward based on history, clinical signs, cytology, and response to antimicrobial therapy.
- A direct smear of the pustular contents or an impression smear of greasy skin should be performed for cytological evaluation.
- If the lesion fails to respond to therapy, or coccoid organisms persist after apparently appropriate antimicrobial therapy or there are abundant bacilli, then a swab of the exudate should be submitted for C&S.
- Histopathology is of little to no value in the diagnosis of superficial bacterial infection but may be useful to differentiate between discoid lupus erythematosus (nasal lupoid dermatosis) and MCP. Antibiotic trial therapy should be instituted before sample collection. The method for sample collection for C&S is described in the previous section on surface bacterial infections.

KEY STEPS

01 Identify bacterial overgrowth and/or infection via surface cytology.

02 Commence trial treatment with appropriate topical and/or systemic antibiotic.

03 Consider bacterial C&S testing if failure to respond to therapy.

04 Correct underlying predisposing factors.

TREATMENT

Most canine skin infections are caused by the coagulase-positive *S. pseudintermedius*. Empirical antibiotic selection is justified for superficial bacterial pyoderma. Selecting an antibiotic that penetrates to the site of infection given at the correct dosage and frequency and for an adequate length of treatment is important. For superficial bacterial infections, treat for 14 days and then re-evaluate to confirm that infection has resolved.

Shampoo therapy is a suitable topical agent for the treatment of pyoderma and benefits most dogs by removing tissue debris, hydrating the skin, and reducing or eliminating the surface bacterial population. Chlorhexidine is usually the active ingredient of choice for superficial bacterial infections.

CONDITION: SUPERFICIAL BACTERIAL INFECTIONS - MUCOCUTANEOUS PYODERMA, BACTERIAL FOLLICULITIS, BACTERIAL OVERGROWTH

RECOMMENDED ANTIBIOTICS

First line:
Cephalexin (20 mg/kg q12h PO)

Second line:
Clindamycin (5.5-11 mg/kg q12h PO)

Third line:
Based on C&S (see section on methicillin-resistant *Staphylococcus pseudintermedius).*

Cefovecin (8 mg/kg q14d for 21-28 days) is suitable for cases where there are serious concerns of compliance, or there are difficulties with oral dosing.

USAGE RECOMMENDATIONS

Full therapeutic dose for 3 weeks, or 10 days beyond complete clinical resolution.

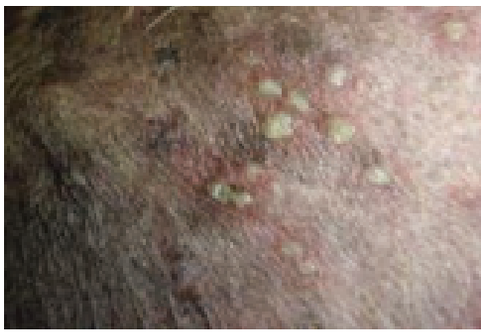
Fluoroquinolones should be avoided for staphylococcal pyodermas unless bacterial resistance to first or second-line antimicrobials has been demonstrated and susceptibility to fluoroquinolones has been demonstrated by C&S.

AIDAP TOP TIPS

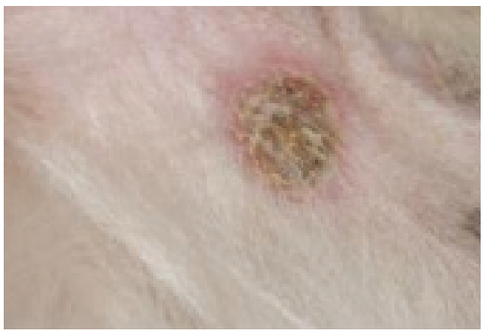
1. It is important to remember that many superficial bacterial infections will respond to the regular topical application of leave-on antimicrobial products containing chlorhexidine.

2. If required, systemic antibiotics must be given for a sufficient duration. A minimum of two weeks is recommended for superficial bacterial infections in dogs and cats.

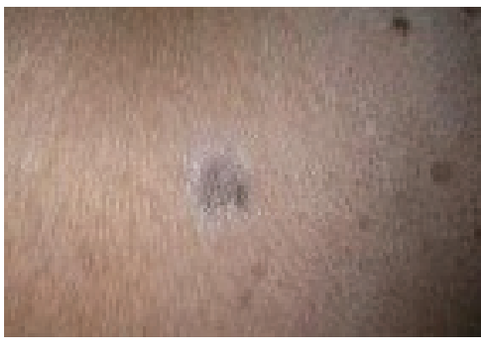
CONDITION: SUPERFICIAL BACTERIAL INFECTIONS - MUCOCUTANEOUS PYODERMA, BACTERIAL FOLLICULITIS, BACTERIAL OVERGROWTH



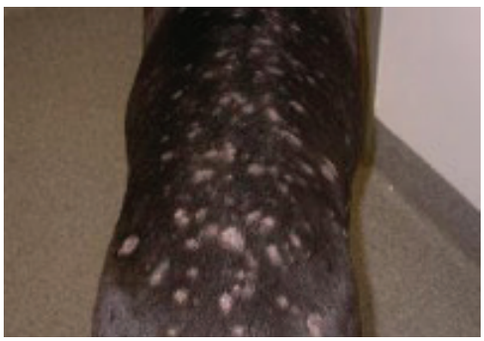
Pustule



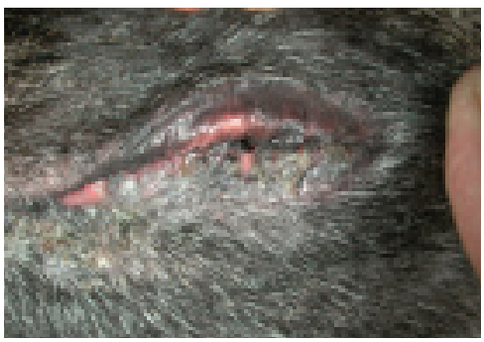
Epidermal collarette



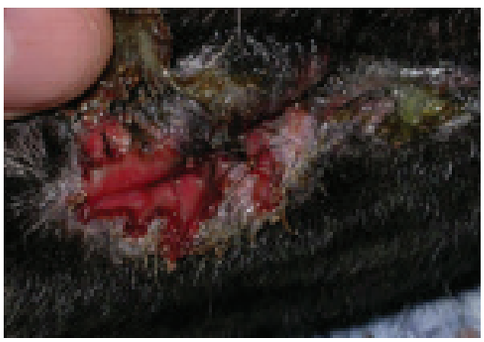
Target lesion – indicative of resolving epidermal collarette



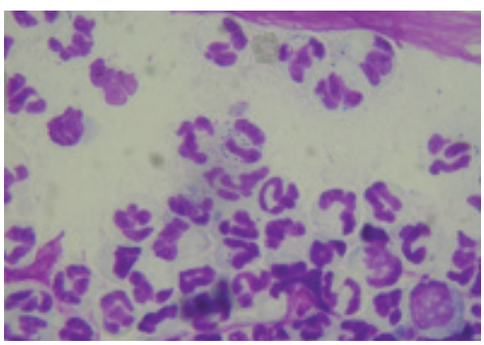
Multiple epidermal collarettes with annular alopecia



Lip fold pyoderma



Ulcerative lip fold pyoderma



Impression smear demonstrating intracellular cocci

Photographs courtesy of Dr Mandy Burrows & Dr Mike Shipstone.

Key references:

Hillier A, Lloyd DH, Weese JS, et al. Veterinary dermatology. 2014; 25(3): 163-e43.

CONDITION: DEEP BACTERIAL INFECTIONS - FURUNCULOSIS WITH DRAINING TRACTS

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Generalised demodicosis is the most common cause of deep pyoderma.

Deep pyoderma may be seen without prior superficial pyoderma or as a sequel to superficial bacterial folliculitis. Deep follicular inflammation leads to follicular rupture releasing hair shaft keratin, bacteria and bacterial products into the dermis resulting in a furunculosis or infection of the dermis and subcutis. Bacteria present are usually *S. pseudintermedius*, but *Proteus, Pseudomonas* and *E. coli* are seen more frequently in deeper infections. *Pseudomonas aeruginosa* has also been isolated from dorsal furunculosis following shampoo therapy.

Generalised furunculosis: Underlying triggers commonly initiate deep pyoderma but are not always identified.

Generalised demodicosis is the most common cause of deep pyoderma.

Others include hyperadrenocorticism, the inappropriate use of glucocorticoids (iatrogenic hyperglucocorticoidism), actinic disease and immunologic defects. Comedonal diseases such as calluses, actinic comedones, and Schnauzer comedone syndrome also predispose to deep pyoderma via rupture of the abnormal follicle.

Distribution: glabrous skin of axillae, inguinal region.

Skin lesions: papules, pustules that rupture forming fistulous tracts discharging seropurulent or haemorrhagic exudate with necrotic, friable tissue and haemorrhagic crusts; erosions and ulcers develop secondary to inflammation, necrosis, and self-trauma. Haemorrhagic bullae are a distinctive clinical feature of canine deep pyoderma. Well circumscribed, firm nodules exhibit a deep dark red to blue hue. Lethargy, depression and fever, pain and pruritus are common with accompanying regional or generalised lymphadenopathy.

Localised syndromes: Deep bacterial folliculitis and furunculosis may be manifest in skin diseases with a traumatic component and may be termed 'traumatic furunculosis' in the

CONDITION: DEEP BACTERIAL INFECTIONS - FURUNCULOSIS WITH DRAINING TRACTS

context. Diseases featuring traumatic furunculosis include post grooming furunculosis, canine acne, callus pyoderma and interdigital furunculosis.

Interdigital furunculosis: is a common presentation. Lesions occur predominantly in the interdigital webs but may affect the digits as well. Although interdigital furunculosis is multifactorial, trauma to hair follicles is a common precipitating cause. Licking/trauma of the interdigital skin leads to penetration of hair shaft into dermis or follicular rupture resulting in a foreign body reaction to the hair shaft keratin with pyogranulomatous inflammation and secondary bacterial infection. Allergic skin disease such as canine atopic dermatitis and adverse food reactions may initiate this syndrome.

Interdigital pyoderma may also present as a sequel to rupture of obstructed, keratin-filled follicles that result from chronic friction or other trauma.

Large/giant breeds and dogs with abnormal foot conformation are predisposed and most commonly the front feet are involved due to increased weight bearing. Follicular dilation and comedone formation result, forming follicular cysts. Follicular cysts rupture and the release of the keratin protein results in a foreign body reaction.

Skin lesions: nodules, haemorrhagic bullae, fistulae discharging serosanguinous to seropurulent exudate; alopecia from constant licking leading to maceration, chronic moistness, and surface secondary bacterial and *Malassezia* overgrowth; varying degrees of pain and lameness, pruritus and paronychia may be present.

Distribution: dorsal interdigital webs; the front feet are most commonly and usually most severely affected.

CONDITION: DEEP BACTERIAL INFECTIONS - FURUNCULOSIS WITH DRAINING TRACTS

TESTS FOR DIAGNOSIS

- Perform a direct smear of the contents of the pustule, nodule, or bulla by puncturing or squeezing the lesion; transfer and spread the contents directly onto a microscope slide for cytological evaluation and stain the sample with Diff-Quik®. If there are no intact lesions then sample the exudate draining from a fistulous tract and make an impression smear of the fluid contents, although microorganisms are usually less evident than in surface and superficial bacterial infections. If there are abundant bacilli, then tissue should be submitted for C&S testing. In all deep pyodermas, it is important to rule out underlying demodicosis by collecting multiple deep skin scrapings and/or trichograms.

- **Histological evaluation is often useful in the diagnostic work-up of deep bacterial pyoderma.** A skin biopsy allows division of the sample with some submitted fresh for bacterial and fungal culture and susceptibility testing and the rest formalinised for histological examination to rule out pododemodicosis. Skin biopsy all other cases of localised and generalised deep bacterial folliculitis and furunculosis.

- Further diagnostic workup involves evaluation of spontaneous or iatrogenic hyperglucocorticoidism (CBC, biochemistry, urinalysis, ACTH stimulation testing), hypothyroidism (free T4 with TSH) and for interdigital furunculosis, evaluation of underlying allergic skin disease (elimination diet trials and intradermal/serological allergy testing) as well as mechanical and traumatic factors.

- **Sample collection for C&S testing (deep infections):** Perform normal aseptic surgical preparation on target skin surface. Collect deep tissue sample aseptically using either biopsy punch or wedge biopsy. Cut epidermis from sample and submit remaining tissue aseptically in saline soaked gauze.

KEY STEPS

01 Collect surface/exudative cytology from draining or intact lesions.

02 Multiple deep skin scrapings or trichograms to evaluate for demodicosis.

03 Biopsy for deep tissue bacterial and fungal culture for recurrent/ non-responsive cases.

04 Treat for adequate length of time (4–6 weeks minimum) with systemic antimicrobial therapy.

05 Evaluate for underlying cause/s.

CONDITION: DEEP BACTERIAL INFECTIONS - FURUNCULOSIS WITH DRAINING TRACTS

TREATMENT

Systemic antibiotics such as cephalosporins, other ß-lactams and fluoroquinolones (particularly if large numbers of bacilli are seen on cytology) should be used for the treatment of deep bacterial infections.

Interdigital furunculosis is usually managed with either cephalosporins or other ß-lactams. If Gram-negative organisms are implicated, then either trimethoprim-sulphonamide or a fluoroquinolone is used for an extended treatment period of 8–12 weeks. Metronidazole may be used as an adjunct treatment.

Most affected animals are allergic and so diagnostic investigation of the underlying allergic skin disease and the use of cyclosporin and other drug modalities to control the underlying allergic symptoms may be indicated once the secondary infection is resolved.

Laser ablation of the interdigital follicular cysts and surgical fusion podoplasty have also been described for these cases, particularly the large or giant breeds that present with comedone formation.

RECOMMENDED ANTIBIOTICS

First line/Second line:
Cephalexin (20 mg/kg q12h PO)
Trimethoprim-sulphonamide (30 mg/kg q12h PO)

Third line:
If first and second-line therapy fails.

Enrofloxacin (10–15 mg/kg q24h) (off-label) or
Marbofloxacin (5.5 mg/kg q24h)

USAGE RECOMMENDATIONS

Full therapeutic dose for 6 weeks, or 10 days beyond complete clinical resolution.

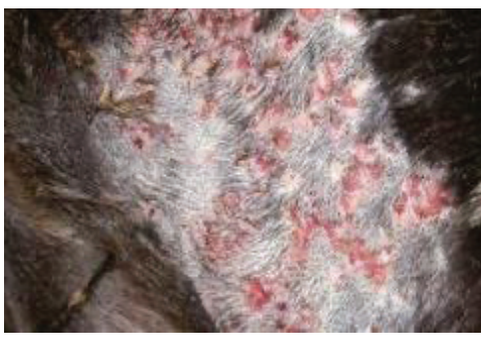
If using Trimethoprim-sulphonamide, measure tear production to avoid KCS.

Topical therapy recommended with 3% chlorhexidine shampoo/lotion/solution.

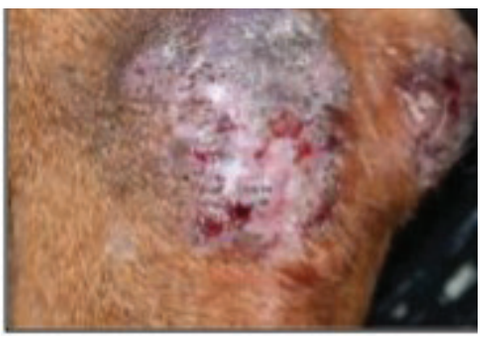
CONDITION: DEEP BACTERIAL INFECTIONS - FURUNCULOSIS WITH DRAINING TRACTS

AIDAP TOP TIPS

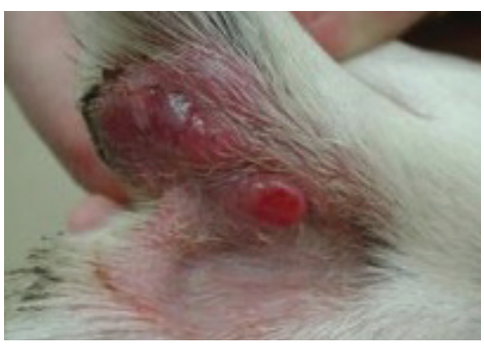
1. For interdigital furunculosis, always look for an underlying cause of self-trauma, including allergic, mechanical and weight bearing factors.



Deep bacterial infection on lateral thigh with multiple discharging sinuses

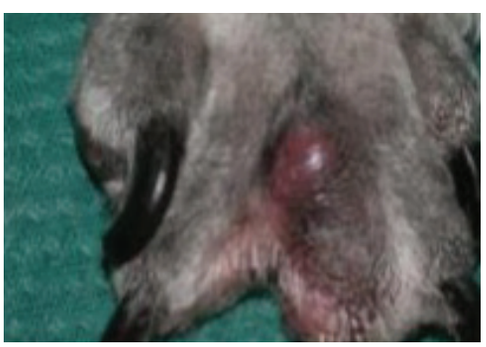


Deep bacterial infection of callus



Interdigital cyst formation

Photographs courtesy of Dr Mandy Burrows & Dr Mike Shipstone.



Interdigital cyst formation

CONDITION: METHICILLIN-RESISTANT STAPHYLOCOCCAL INFECTIONS

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

The clinical lesions of infection by a methicillin-resistant *Staphylococcus* (MRS) show NO variation from that resulting from methicillin-susceptible *staphylococcus* (MSS).

Staphylococcus pseudintermedius, S. schleiferi (including the coagulase negative variant) and *S. aureus* are the primary pathogens seen in small animal dermatology. However, several other species of coagulase-negative *staphylococcus* (CoNS) have been reported to cause skin and soft tissue infections and the clinical relevance of an isolated organism must be determined in an individual case by the clinician.

Methicillin (or Meticillin) is a staphylococcal ß-lactam-resistant antibiotic that was released in the early 1960s. Shortly after, *S. aureus* developed resistance to methicillin through the acquisition of the *mecA* gene that codes for a specific penicillin-binding protein (PBP2a) with low affinity for ALL ß-lactam antibiotics including cephalosporins, carbapenems, monobactams and penems. Not only are these organisms Methicillin (pan-ß-lactam)-resistant, but they commonly express co-resistance to other drug classes and may be multidrug-resistant (MDR, resistant to at least 3 different antibiotic classes), extremely drug-resistant (XDR, resistant to all but 1 or more antibiotics in less than 2 different classes) or pan drug-resistant (PDR, resistant to all common antibiotics) as well.

Methicillin resistance has emerged as a serious concern in veterinary medicine and is mostly seen in *S. pseudintermedius*. Prevalence rates vary enormously (0.58-30%), but this may reflect differences in target animals and disease states. Risk factors seem to be previous use of antibiotics, previous hospitalisation, living in an urban environment, and older age of animal. Frequency of isolation varies geographically. Across Australia, infrequent cases of MRSP are seen in Western Australia and South Australia, while NSW and Victoria see frequent cases in primary small animal practice. On C&S reports, methicillin resistance is indicated by resistance to the surrogate antibiotic oxacillin.

Clinical Aspects: The clinical lesions of infection by a methicillin-resistant *staphylococcus* (MRS) show NO variation from that resulting from methicillin-susceptible *staphylococcus* (MSS). (See Surface, Superficial and Deep skin infection sections). The clinical signs of superficial bacterial infection are the same regardless of whether caused by MRS or MSS and include pustules, papules, crusted papules, epidermal collarettes, crusts, hair loss leading to "moth eaten" appearance to the coat, target lesions.

CONDITION: METHICILLIN-RESISTANT STAPHYLOCOCCAL INFECTIONS

TESTS FOR DIAGNOSIS

Cytology (with samples collected using acetate tape preparation, impression smear, cotton bud smear, "glue" slides) to demonstrate coccoid organisms from lesional skin is considered mandatory.

C&S testing (see techniques described in previous sections) is considered mandatory in the following situations as it may indicate the development of resistance:

1. Less than 50% reduction in lesions after 2 weeks of appropriate therapy.
2. Development of new lesions ≥2 weeks of appropriate therapy.
3. Presence of residual lesions after 6 weeks of appropriate therapy, with cocci on cytology.
4. Rod shaped bacteria on cytology.
5. Prior history of MDR infection in the pet or in a pet from the same household as the affected individual.

One of the reported risk factors for MRSP is the prior use of systemic antibiotics. Thus C&S should always be considered in cases of recurrent infections or repetitive antibiotic use.

Empirical drug selection for systemic medication is contraindicated when a MRS infection is suspected, due to the high prevalence of multidrug resistance.

Rather than relying on the owner to determine the end point for antimicrobial therapy a revisit should always be scheduled to allow the clinician to determine if the infection has been successfully controlled.

Client education is also vital so that they understand not to stop the course of treatment early, nor continue in the face of non-response or development of new lesions after 2 weeks of therapy.

TREATMENT RECOMMENDATIONS

It is important to determine if the animal has any underlying disease such as endocrinopathy or atopy that may predispose it to recurrent infection. These must be addressed at the same time as the treatment for the infection is commenced.

Topical antimicrobial therapy

Topical therapy should be used in all cases of MRS pyoderma when a pet or owner can be compliant. In cases of surface bacterial infection and overgrowth it is often the only treatment modality required. It will reduce bacterial numbers and recolonisation, reducing the time to resolution of initial infections (when combined with systemic therapy), helping to prevent relapse and can be of great value when treating and controlling chronic and recurrent cases.

Topical therapy alone is recommended in cases of localised lesions, and early stages of generalised infection (when lesions mild), to help prevent relapse whilst causes of relapsing infection are investigated.

For focal/localised lesions:

Gels, creams, ointments, lotions, and wipes containing the following active ingredients:

Antiseptics: Hydroxyl acids such as acetic, lactic, or malic acid, benzyl peroxide, silver sulfadiazine, chlorhexidine.

Antimicrobials: Bacitracin, fusidic acid (should only be used following C&S).

CONDITION: METHICILLIN-RESISTANT STAPHYLOCOCCAL INFECTIONS

Extensive/generalised lesions:

Shampoos, conditioners, rinses, sprays, and lotions containing the following active Ingredients:

Antiseptics: Chlorhexidine, benzyl peroxide, iodine. Use 2-3 times a week until 7 days beyond resolution then weekly as prophylaxis. Contact time 10 minutes if rinsed from skin.

In cases of highly resistant strains, Dakin's Solution has been used and has been shown to be very effective. A 2013 study by Parisier et al of 16 strains of MRSP showed a Minimum Bactericidal Concentration of 1:32, with most strains susceptible between 1:64 & 1:128 of a 6.15% concentrate bleach solution.

Dakin's Solution (0.5% sodium hypochlorite):

1. **Method:**
   1. Boil 1 litre water and allow to cool
   2. Add ½ teaspoon baking soda
   3. Add 100 mL of 5.25% sodium hypochlorite (bleach)
2. **Storage**
   1. In tightly sealed glass jar
   2. Protected from light (aluminum foil)
   3. Unopened jars may be stored for 30 days
   4. Throw away unused open solution after 48 h
   5. Label with preparation date & disposal date
3. **Adverse effects**
   1. Xerosis (most common)
   2. Pain
   3. Irritation
   4. Erythema
   5. Pruritus
   6. Urticaria
   7. Vesicle formation

Systemic Antimicrobial Therapy

Systemic antimicrobial therapy may be used for surface or superficial pyoderma caused by MRS if pet or owner compliance prevents the use of topical treatment or in the case of deep bacterial infections. The antimicrobial should be used for 7 days beyond clinical cure (as assessed by a veterinarian).

If the case becomes recurrent a C&S is indicated but does NOT necessarily require an elevation in the tier of drug to be selected.

Empirical systemic therapy should only be selected from first line/second line antibiotics when risk factors for resistance do not exist, such as uncomplicated pyoderma that is not recurrent, have only been treated previously with topical antiseptics and where there is no history of multiple antibiotics used or previous MDR bacteria in the household). See previous sections on MSS for antimicrobial recommendations.

CONDITION: METHICILLIN-RESISTANT STAPHYLOCOCCAL INFECTIONS

RECOMMENDED ANTIBIOTICS

Based on positive culture for MRS and C&S testing indicating susceptibility.

First line/Second line:

Clindamycin (11-15 mg/kg q12h PO)

Trimethoprim-potentiated Sulphonamides (30 mg/kg q12h PO)

Doxycycline (5 mg/kg q12h PO) or Minocycline (5-1 2.5 mg/kg q12h PO), commonly used in combination with another drug for MRS infection to avoid resistance

Chloramphenicol (40-50 mg/kg PO q8h dog; 50 mg/cat or 12.5-20 mg/kg q12h cat)

Aminoglycocides: Gentamicin (9-14 mg/kg q24hr IV, IM, SC dog; 5-8 mg/kg q24h IV, IM, SC cat) for short term (5-7 days) treatment only: monitor renal function

Third line:

Marbofloxacin (5 mg/kg q24h PO)

Pradofloxacin (7.5 mg/kg q24h PO)

Rifampicin (10 mg/kg q24h PO) - Note that the human therapeutic guidelines recommend rifampicin is used in combination with another drug to avoid resistance

Aminoglycosides: Amikacin (6.5 mg/kg q8h IV, IM, SC dog and cat or 15-30 mg/kg q24h IV, IM, SC dog; 10-14 mg/kg q24h IV, IM, SC cat) for short term (5-7 days) treatment only: monitor renal function)

USAGE RECOMMENDATIONS

First or second line antibiotics recommended for MRS infections based on C&S are no less potent than third line if the organism is susceptible.

First/second line recommendations are based on low to medium ASTAG importance ratings.

The third line drugs are reserved for use when the MRS has been shown to be resistant to first line or second line options due to the presence of a MDR, XDR or PDR MRS, or where client/patient factors prevent the use of a first or second-line drug.

Systemic drugs (apart from aminoglycosides) should be continued for 7 days beyond clinical resolution (as assessed by a veterinarian NOT owner).

Concurrent glucocorticoid use is discouraged as it may suppress the inflammatory response and make judgment of the end point difficult.

There is little evidence for any difference in outcome between MRS and MSS infections and the prognosis for successful treatment is good, depending on the underlying cause and co-morbidities present.

Amikacin is not registered for use in animals and should not be used off-label except in exceptional circumstances for individual animals.

CONDITION: METHICILLIN-RESISTANT STAPHYLOCOCCAL INFECTIONS

AIDAP TOP TIPS

1. First or second-line antibiotics recommended for MRS infections based on C&S are no less potent than third line if the organism is susceptible.

2. Systemic drugs (apart from aminoglycosides) should be continued for 7 days beyond clinical resolution (as assessed by a veterinarian NOT owner).

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CONDITION: MYCOBACTERIA AND NOCARDIA AS CAUSES OF DEEP DRAINING SINUS TRACTS

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Mycobacteria cause two major types of disease affecting the skin and subcutis:

(i) Infections of the subcutaneous panniculus generally with rapidly growing mycobacteria and

(ii) Granulomatous or pyogranulomatous masses of the skin and subcutis (generally due to non-cultivable mycobacteria such as feline leprosy-like syndromes, leproid granulomas, and sometimes *Mycobacterium avium* complex infections). These infections will not be discussed further here but generally combination therapy is required using rifampicin, clarithromycin, pradofloxacin and clofazimine in various combinations.

The taxonomy of these organisms continues to evolve, and currently they are divided into complexes:

a. *M. smegmatis* complex (including *M. goodii*) – drugs of choice are doxycycline, pradofloxacin, (gentamicin, amikacin).

b. *M. fortuitum* complex– drugs of choice are clarithromycin, pradofloxacin, (gentamicin, amikacin).

c. *M. chelonae/abscessus* complex– drugs of choice are clarithromycin and linezolid, otherwise depends on susceptibility testing.

In general, they are all resistant to rifampicin, and all susceptible to clofazimine (which is hard to source but good for some refractory cases).

Draining sinus tracts should alert the practitioner to the presence of saprophytic pathogens such as mycobacteria, *Nocardia* spp. and fungi.

Involvement of the inguinal panniculus is suggestive of mycobacterial and nocardial disease.

Preliminary cytology stained with Diff-Quik® can be very helpful in cases where *Nocardia* and fungi are involved, whereas culture on routine media is far more expedient a way to diagnose mycobacterial infections caused by rapidly growing saprophytic mycobacteria.

CONDITION: MYCOBACTERIA AND NOCARDIA AS CAUSES OF DEEP DRAINING SINUS TRACTS

KEY ISSUES

a. Rapidly growing mycobacteria, *Nocardia* spp. and fungi can all give rise to deep draining tracts that discharge to the skin surface.

b. Rapidly growing mycobacteria (and to a lesser extent *Nocardia nova*) have a predilection for the fatty subcutaneous panniculus, especially in the inguinal region.

TREATMENT

These infections require months to years of antimicrobial therapy, and in some cases, surgery is required to debulk lesions to enable a clinical cure to be achieved. The timing of surgery is a value judgement and ideally it should occur after preliminary medical therapy, so the lesion is smaller and blood levels of effective drugs are present at the time of surgery and during the healing phase.

Topical therapy is not useful in the management of these infections as the disease process is situated in the subcutis and involves the skin secondarily.

TESTS FOR DIAGNOSIS

1. The cornerstone of therapy is obtaining a positive culture.
2. This is obtained by aspirating purulent exudate present in the subcutis through intact skin, after preparation of the skin with 70% ethanol (and allowing time for drying).
3. Primary isolation can be done in a veterinary laboratory, although it important to keep the plates for the 4-5 days it takes for the colonies to appear.
4. Positive cultures should be forwarded to a human mycobacteria reference laboratory for species identification and C&S testing.

REFERENCE LABORATORIES MANAGING CULTURE AND PCR OF MYCOBACTERIA AND *NOCARDIA*:

NSW

Charlotte Webster
Concord Hospital
Charlotte.Webster@health.nsw.gov.au

Institute of Clinical Pathology and Medical Research
Westmead Hospital (via Vetnostics)

WESTERN AUSTRALIA

Dr Ammie Higgins
PathWest Laboratory Medicine WA
Ammie.higgins@health.wa.gov.au

QEII Medical Centre, Nedlands
Western Australia

CONDITION: MYCOBACTERIA AND NOCARDIA AS CAUSES OF DEEP DRAINING SINUS TRACTS

RECOMMENDED ANTIBIOTICS

C&S is strongly advised in these cases.

Mycobacteria:

First line:

Doxycycline (5 mg/kg q12h PO†) and

Pradofloxacin (5-8 mg/kg q24h PO) for *M. smegmatis* complex infections.

Clarithromycin (5-15 mg/kg q12h PO) and

Pradofloxacin (5-8 mg/kg q24h PO) for other *M. fortuitum*.

Clarithromycin/linezolid for *M. chelonae/abscessus.*

Second line:

Clofazimine (4-10 mg/kg q24h PO compounded for cats) and

Amikacin (10-15 mg/kg q24h IV/IM/SC)

***Nocardia* spp.**

First line:

Trimethoprim-sulphonamide (12.5 to 30 mg/kg q12h PO)

Second line:

Amoxicillin (20 mg/kg BID PO) for *N. nova* (not amoxicillin-clavulanate)

Third line:

Clarithromycin (5-15 mg/kg q12h PO)

Pradofloxacin (5-8 mg/kg q24h PO)

†Ensure doxycycline given with food or a water bolus.

USAGE RECOMMENDATIONS

See current textbooks for detailed guidelines.

Clarithromycin, amikacin and linezolid are not registered for use in animals and should not be used off-label except in exceptional circumstances for individual animals.

In some refractory cases linezolid can be lifesaving but is expensive. When using linezolid, best to determine peak and trough levels to optimise efficacy and avoid toxicity.

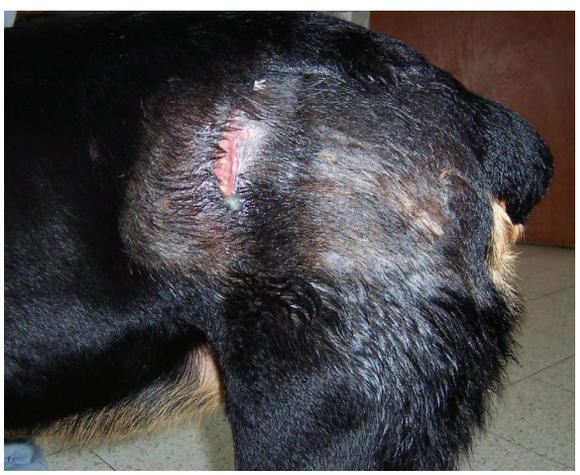
When using trimethoprim-sulphonamide combinations, do not split or otherwise divide the coated tablet. If coated tablets are unavailable give inside a gelatine capsule coated with margarine) combined with a second drug depending on C+S testing. Beware keratoconjunctivitis sicca during therapy.

CONDITION: MYCOBACTERIA AND NOCARDIA AS CAUSES OF DEEP DRAINING SINUS TRACTS

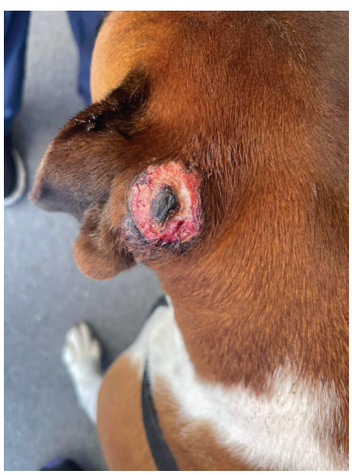
AIDAP TOP TIPS

1. Getting susceptibility data and species identification is expensive in the short term but good value in the long term. Compliance and diligent owners are important as long courses of therapy are required.

2. Richard Malik is available to provide free advice on optimal management of these infections – Richard.Malik@sydney.edu.au.



Saprophytic mycobacterial infection in a dog.
Photo courtesy of Dr Richard Malik.



Canine leproid granuloma.
Photo courtesy of Dr John Roberts.

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CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Microsporum canis is responsible for 70 to 95% of canine infections and 94 to 99% of feline infections

Dermatophytoses are superficial fungal infections that involve the skin, hair, and claws.

Microsporum canis is responsible for 70 to 95% of canine infections and 94 to 99% of feline infections.

Microsporum gypseum and *Trichophyton mentagrophytes* account for most of the remaining cases. Infections with unusual species such as *Microsporum persicolor* have been reported; it is uncertain how commonly these infections occur, but prevalence maybe related to local climate and environmental factors.

Skin lesions: There are many clinical presentations of feline dermatophytosis. Pruritus is variable and can range from nil to severe. In kittens, irregular, annular to circular patches of alopecia with scale, crust and erythema affecting face, ears and forelegs are common. In adult cats, focal, multifocal, or generalised patchy alopecia with or without scale occurs frequently, especially in long haired cats.

Dermatophyte infections are not common in dogs. They are most common in young dogs, dogs from animal shelters or dogs that are debilitated. Circular patches of alopecia with scale, crust, central hyperpigmentation, and follicular papules on the periphery affecting the face, pinnae, paws, and tail is the most frequent presentation in the dog.

Less commonly, dermatophytosis can present with folliculitis that may be localised, regional (facial) or generalised; often with furunculosis. Nodular (kerion) lesions on face and legs are an exudative, circumscribed type of furunculosis with multiple draining tracts usually associated with *M. gypseum* or *T. mentagrophytes*.

Onychomycosis is a rare, chronic subungual fold inflammation with or without footpad involvement, paronychia, claw deformity and fragility in the dog.

CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON

TESTS FOR DIAGNOSIS

No single test has been identified as the "gold standard".

Surface cytology: acetate tape preparations or firm impression smears may identify fungal hyphae and arthrospores in the stratum corneum.

Trichograms: examine the follicular debris of anagen follicles for the presence of ectothrix fungal elements. This may be aided by clearing agents, such as chlorphenolac.

Wood's lamp examination: positive in most cases of *M. canis* dermatophytosis. Fluorescing hairs are most often detected in untreated infections; fluorescence may be difficult to find in treated cats.

Fungal culture: a soft bristle, sterile toothbrush technique is the recommended method for collecting hair samples. An alternate method of equal sensitivity is to collect the sample using a 4 cm length of acetate tape attached to the end of a glass slide for submission.

Histopathology: a biopsy can be useful.

PCR: a positive test does not always indicate active infection, as DNA from inactivated organisms and non-infected fomite carriers can be detected. A negative PCR in a treated cat is compatible with cure.

KEY STEPS

01 Perform a trichogram to evaluate for ectothrix arthrospores.

02 Wood's lamp examination for *Microsporum canis* infections only.

03 Collect hair and scale for fungal culture using haemostat (dogs).

04 Avoid 'spot' therapy with topical antifungal ointments.

05 Implement topical/systemic/environmental treatment.

CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON

TREATMENT

In most healthy animals, dermatophytosis is a self-curing disease, with full resolution of disease in 10–16 weeks without therapy.

The best treatment protocol is a combination of three modalities:

1. **Topical treatment:** to kill infective material and prevent its dissemination into the environment.
2. **Systemic treatment:** to shorten the time of infection in the individual animal.
3. **Environmental treatment:** to help prevent recurrence of infection or spread to other animals or people in the household.

TOPICAL THERAPY:

i. Total body treatment

Topical therapy inactivates fungal spores and mycelia on and within hair shafts, reducing environmental contagion, and results in a faster cure than systemic therapy alone. Shampoo therapy, dipping or rinsing with topical antifungal agents is preferred. Twice weekly application of miconazole/chlorhexidine shampoo and/or 2% enilconazole rinse are currently recommended. Chlorhexidine as monotherapy is poorly effective and is not recommended.

ii. Localised (treating only the spots) or whole-body topical therapy

Focal lesions in difficult-to-treat locations such as the face and ears should receive additional specific topical localised therapy with 2% miconazole cream. In animals, not all the lesions may be visible due to the long hair coat. It is almost certain that there are infective spores in non-lesional areas. Therefore 'spot treatment' alone with topical drugs is not recommended.

SYSTEMIC THERAPY:

Systemic antifungal therapy targets the active site of fungal infection and proliferation on the infected animal. Until the infection is eliminated in this site, the infected animal is at risk for further spread of lesions on its body, continued seeding of the hair coat with infective spores, and being a source of infection for other animals and people. Systemic therapy is the treatment of choice for dermatophytosis. It is important to remember that systemic treatment does not rapidly reduce contagion and should be used in conjunction with topical antifungal therapy.

Itraconazole (non-compounded) and terbinafine are the most effective and safe treatments for dermatophytosis.

CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON

ITRACONAZOLE (SPORANOX®)

Itraconazole persists in the skin and nails for weeks to months after dosing and is frequently prescribed for skin infections or onychomycosis.

Itraconazole is generally well tolerated; reported side effects include vomiting and/or anorexia in cats. Signs of dose related hepatotoxicosis have been reported rarely in cats and an idiosyncratic cutaneous vasculitis has been reported in dogs. Itraconazole is reportedly not teratogenic when used at a dose of 5 mg/kg. Compounded or reformulated itraconazole should not be used as studies in both dogs and cats have shown poor bioavailability.

TERBINAFINE

There is limited data on the use of terbinafine in dogs and cats and this drug appears to offer no advantages over itraconazole.

Terbinafine is generally well tolerated; reported adverse effects include vomiting and asymptomatic elevation in liver enzymes. Idiosyncratic acute hepatotoxicity has been reported occasionally. No teratogenicity has been reported. The drug reaches very high concentrations in sebum and stratum corneum and fungicidal concentrations persist in the skin for several weeks after administration in humans and cats, but not in dogs.

FLUCONAZOLE

Fluconazole has poor antifungal efficacy against dermatophytes. It has the highest MIC compared to itraconazole, terbinafine, ketoconazole and griseofulvin for both *Microsporum* spp. and *Trichophyton*. It is not recommended for the treatment of dermatophytosis.

MONITORING AND CURE

Monitoring of response to therapy includes clinical improvement, Wood's lamp evaluation and fungal culture. Clinical cure will precede mycological cure. A lack of resolution of clinical signs and/or development of new lesions indicates a treatment problem or misdiagnosis. Wood's lamp examination can be used to monitor cats for resolution of *M. canis* infections.

In most cats, systemic antifungal treatment is needed for four to eight weeks until clinical resolution of lesions. Topical antifungal therapy should be continued until mycological cure. There is no scientifically established definition, but a negative PCR test is compatible with mycological cure. For *M. canis* infections, a negative fungal culture and a negative Wood's lamp examination (except for glowing tips) are compatible with mycological cure.

ENVIRONMENTAL TREATMENT:

The two reasons for environmental disinfection are:

1. To minimise the risk of disease transmission to people and other animals.
2. To minimise fomite carriage causing false positive fungal culture or PCR results.

Infection from the environment alone is rare. Contact with a contaminated environment alone in the absence of concurrent microtrauma is an exceedingly rare source of infection in both people and animals. Infected owners are found only in households containing cats and owner infection is mostly associated with direct contact with a cat (or kittens). The primary mode of dermatophyte transmission is animal to animal contact even in the presence of a contaminated environment.

CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON

Minimising contamination can be achieved via clipping of affected lesions, topical therapy, and routine cleaning.

CLIPPING THE HAIR COAT

Clipping the hair coat is not necessary in every case of dermatophytosis. In most cases, extensive clipping requires sedation to minimise patient injury and fear. Clipping the hair coat removes fragile hairs that will fracture and release spores into the environment. The owner should be warned that a temporary exacerbation of lesions may occur after clipping.

If the animal is to be clipped in the clinic all debris produced is infectious with zoonotic potential and so rigorous infection control measures should be observed, such as covering table surfaces with disposable drapes, using gowns and gloves, collecting all materials and double bagging, thoroughly disinfecting the room and all equipment used with an appropriate antifungal agent.

TOPICAL THERAPY

The major owner actions that minimise confinement and decrease risk of infection to susceptible people are compliance with oral antifungal therapy and use of topical therapy twice a week.

ROUTINE CLEANING

Twice weekly cleaning/disinfection is recommended. This includes mechanical removal of hair, washing and disinfection of target areas. Daily removal of pet hair from the room/area where the pet is being confined using mechanical removal is recommended (dust cloths, flat mops, sweeping). Use of a daily one-step cleaner can be used on days between more thorough cleaning.

Infective material is easily removed from the environment; if it can be washed, it can be decontaminated.

ANTIFUNGAL DISINFECTANTS

1. **Accelerated hydrogen peroxide (AHP)** is currently available in concentrates, ready-to-use formulations, and over-the-counter products. Its antifungal efficacy against *M. canis* and *Trichophyton* spp. have been shown in several studies.
2. **Sodium hypochlorite (household bleach)** is an effective disinfectant at concentrations ranging from 1:10 to 1:100 even with short contact times. Household bleach diluted at 1:100 and not stored in a brown opaque container retains only 40 to 50% of chlorine after 30 days. If household bleach is used it should be prepared at least once weekly and stored in a dark opaque container. There are many reasons not to use bleach and these include a lack of detergency which is a critical factor for disinfection, potential to react with other chemicals to create toxic gases, unpleasant odour, damage to hard surfaces, discolouration of fibres and coloured surfaces, damage to floor finishes, rapid loss of efficacy once diluted and human health concerns. The product is an irritant to both animals and people.

DISINFECTION OF NONPOROUS SURFACES

Disinfection of nonporous surfaces involves three steps. The first is the mechanical removal of all debris using vacuuming or sweeping as disinfectants will not work in the presence of organic debris. The second is

CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON

the washing of the target surface with a detergent to remove debris. Detergents must be rinsed from the target surface because some may inactivate disinfectants. These two steps are the most important and will often decontaminate a surface. The final step is the application of a disinfectant to kill any residual spores.

DISINFECTION OF LAUNDRY

Fabrics contaminated with infective spores and hairs can be washed in any water temperature without the addition of sodium hypochlorite. Two washings on the longest wash cycle are effective. Spray the surface of the washing machine and the dryer with accelerated hydrogen peroxide after use.

DISINFECTION OF CARPETS AND WOOD FLOORS

Vacuuming alone does not decontaminate carpets exposed to infective *M. canis* hairs and spores but is recommended to remove debris including infective hairs. Disinfect the vacuum with AHP spray and/or wipes.

Wash exposed carpeting twice with a carpet shampooer with detergent or use a steam cleaner. Steam cleaning has the fastest drying time and no discolouration. Pre-treat heavily contaminated carpets with disinfectant and then wash with a beater brush carpet shampooer. Disinfectants (chlorhexidine and sodium hypochlorite) may discolour carpets.

There are no safe surface disinfectants for wooden floors. Wooden floors can be decontaminated via daily removal of hair and dust using commercial disposable cleaning cloths designed for dry mopping and washed twice weekly with a wood oil soap.

CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON

ANTIFUNGAL AGENTS USED

First line:

Dogs: Itraconazole (5–10 mg/kg q24h PO)

Cats: Itraconazole (5 mg/kg q24h PO)

Second line:

Terbinafine (30-40 mg/kg q24h PO)

USAGE RECOMMENDATIONS

Significant duration: 6–10 weeks.

Treat until cured.

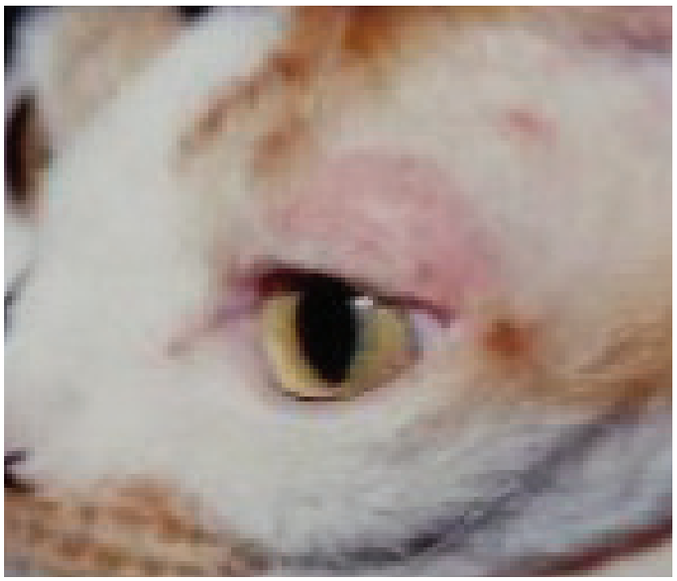
For itraconazole in cats a regime of 5 mg/kg q24h PO for three one week-on and one week-off cycles is recommended.

AIDAP TOP TIPS

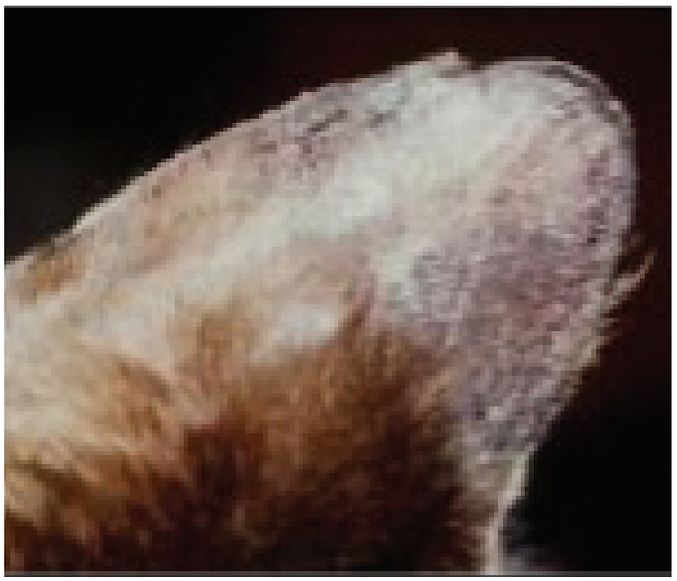
Our treatment recommendations for dermatophytosis for dogs and cats:

1. 2% miconazole, 2% chlorhexidine shampoo baths twice a week.
2. 0.2% enilconazole (Imaverol®) rinse twice a week (not registered for use in cats).
3. Environmental decontamination: important but zoonotic infection unlikely from just environmental exposure.

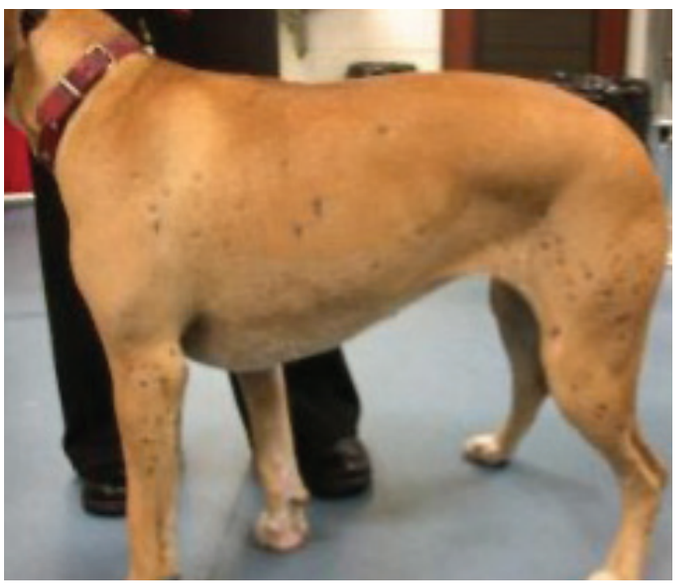
CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON



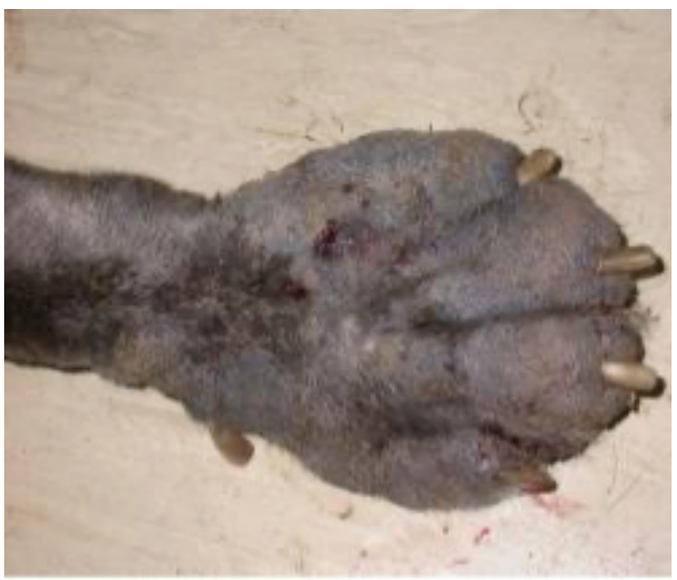
M. canis infection on preauricular skin showing mild inflammation.



M. canis infection of pinnal tip producing alopecia with minimal skin changes.



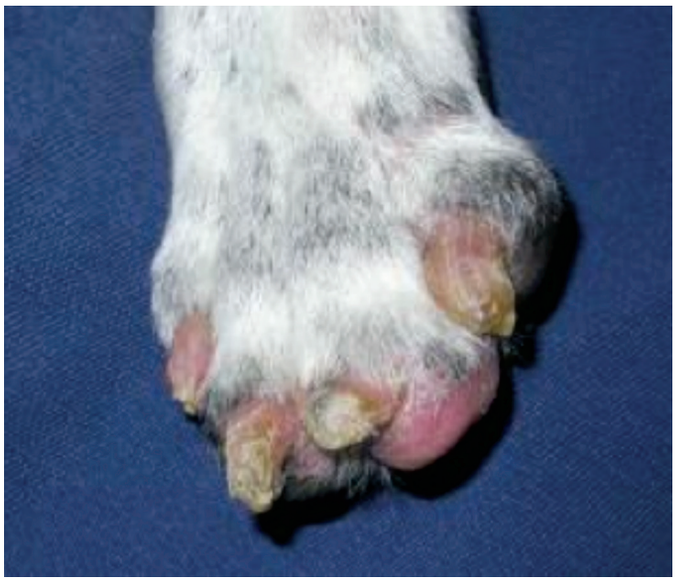
Multifocal alopecia due to *Trichophyton* infection inflammation.



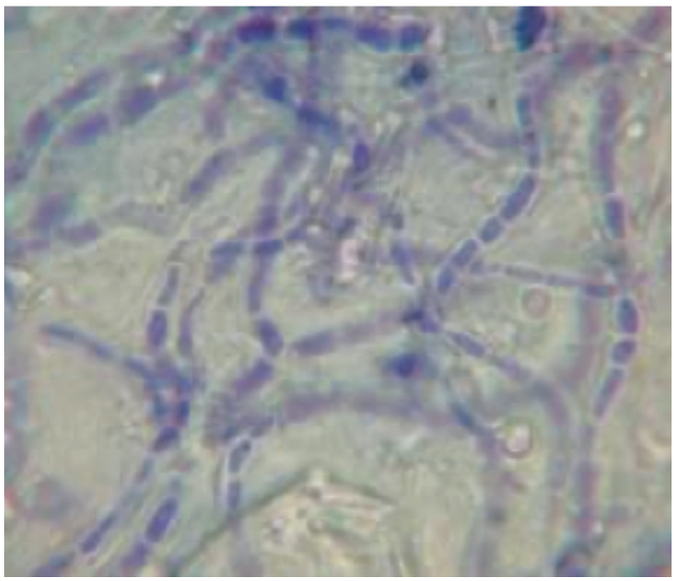
Severe dermatophytosis causing soft tissue swelling.

Photos courtesy of Dr Mandy Burrows & Dr Mike Shipstone.

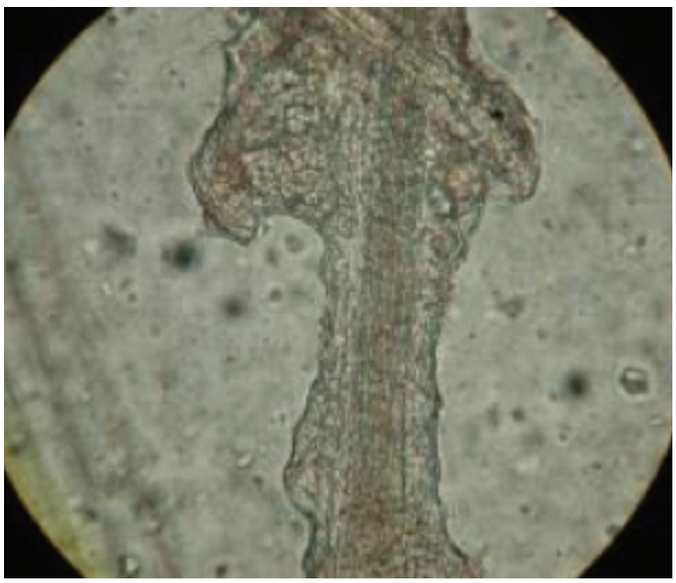
CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON



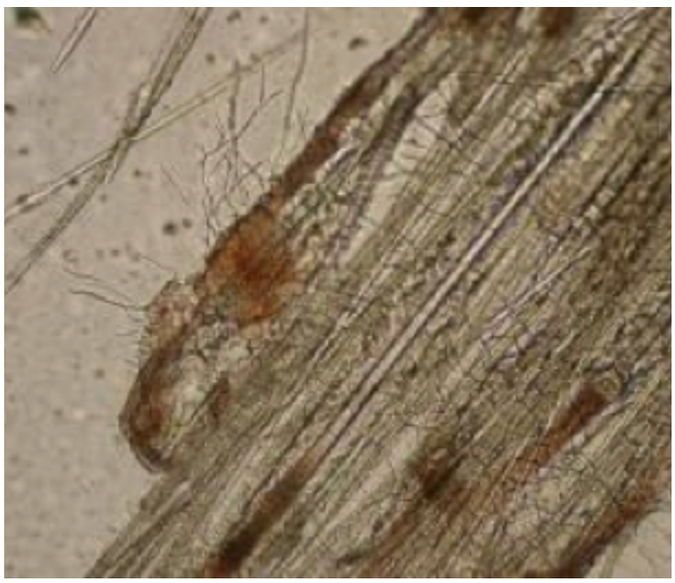
Onychomycosis causing nail deformation.



Fungal hyphae on surface cytology.



Fungal hyphae surrounding the hair shaft.



Fungal hyphae surrounding the hair shaft.

Photos courtesy of Dr Mandy Burrows & Dr Mike Shipstone.

CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON

Dermatophyte lesions of *M.canis* affecting a child. Photo courtesy of Dr Richard Malik.

Key references:

Moriello KA, Coyner K, Paterson S, Mignon B. Veterinary Dermatology. 2017; 28(3): 266-e68.

CONDITION: SUPERFICIAL YEAST (*MALASSEZIA*) INFECTIONS OF THE SKIN (NOT INCLUDING EARS)

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Skin lesions in dogs with *Malassezia* dermatitis can be localised or generalised. Regional dermatitis commonly occurs on the muzzle, lips, ventral neck, axillae, ventral abdomen, medial hindlimbs, interdigital skin, perineum and in the external ear canal and intertriginous areas.

Malassezia pachydermatis is a normal inhabitant of healthy canine skin and mucosae. Populations vary markedly between anatomical sites and between different breeds. There have been significant and recent advances in understanding of the mechanisms of interaction between *Malassezia* yeasts and dogs and cat. The outcome of *Malassezia* growth on the skin (commensal existence or inflammation and disease) is dependent upon the metabolic activities of the yeasts (expression of cell wall and secreted virulence attributes) and the host's innate and adaptive immune defensive responses. Interactions with other skin commensals (especially staphylococci) may also play a role in determining the outcome of colonisation in animals, especially in dogs and cats.

Commensal *Malassezia* populations provide a reservoir of yeasts that might proliferate and induce an inflammatory response under the influence of various host predisposing factors. Dogs breeds at increased risk of *Malassezia* dermatitis include West Highland white terriers, English setters, Shi-Tzus, Basset hounds, American cocker spaniels, Boxers, Dachshunds, Poodles and Australian silky terriers. The presence of skin folds is a common risk factor for localised disease. Dogs with *Malassezia* dermatitis often have concurrent hypersensitivity disorders, cornification defects or endocrinopathies. Many dogs with *Malassezia* dermatitis have concurrent dermatoses, especially hypersensitivity disorders, bacterial pyoderma, endocrinopathies or cornification defects. This can complicate the diagnosis or lead to misdiagnoses due to overlapping of clinical signs.

CONDITION: SUPERFICIAL YEAST (*MALASSEZIA*) INFECTIONS OF THE SKIN (NOT INCLUDING EARS)

Distribution: skin lesions in dogs with *Malassezia* dermatitis can be localised or generalised. Regional dermatitis commonly occurs on the muzzle, lips, ventral neck, axillae, ventral abdomen, medial hindlimbs, interdigital skin, perineum and in the external ear canal and intertriginous areas.

Lesions: diffuse erythema and variable amounts of keratosebaceous scale that can be brown, yellow, or grey in colour. The skin and hair coat may become greasy and self-induced alopecia can occur due to the pruritus. When paronychia is involved, there is red-brown staining of the proximal claw or hair and a waxy exudate in the claw fold, with inflammation of the surrounding soft tissue. Pruritus may be quite marked. In chronic or severe lesions there may be excoriation and lichenification. Dogs with generalised lesions often have an offensive, rancid odour. *Malassezia* overgrowth in the ears typically results in pruritic, erythematous, ceruminous otitis externa, which results in an accumulation of a brownish discharge.

TESTS FOR DIAGNOSIS

Diagnosis is confirmed by routine cytological sampling of skin sites by light microscopical examination (50x or 100x oil objectives) of tape strip samples, slide impressions or dry scrapes stained with Diff-Quik® showing elevated populations of *Malassezia*. Cytology using cotton tipped swabs is normally best restricted to use in the ear canal.

Variations in anatomical site, breed, sampling method and host immune status commonly thwart the interpretation of the clinical significance of an observed population but a general guide is that the presence of *Malassezia* above 1 to 2 per high power x1000 oil immersion is sufficient to warrant trial therapy.

For diagnosis of *Malassezia* paronychia, the broken end of a wooden cotton-tip swab or a toothpick can be used to scrape the claw fold, and exudate is pressed and rolled onto a glass slide.

For examination of ear exudate in dogs with ceruminous or exudative otitis externa, rolling of exudate in a thin layer on glass slides with a cotton-tip swab is the preferred method.

KEY ISSUES

01 Identify yeast overgrowth or infection via surface cytology.

02 Response to trial treatment with appropriate topical and systemic antimicrobial therapy.

CONDITION: SUPERFICIAL YEAST (*MALASSEZIA*) INFECTIONS OF THE SKIN (NOT INCLUDING EARS)

TREATMENT

Treatment of *Malassezia* dermatitis typically involves the use of topical and/or systemic antifungal medications. Topical treatment such as shampoos, gels and lotions are appropriate since the yeast is located within the stratum corneum.

In severe cases of microbial overgrowth or when washing/spraying of the affected areas is not practical, systemic therapy with imidazoles such as ketoconazole or itraconazole can be utilised. The use of these drugs may depend on regional differences in availability, regulatory status, and cost. Compounded formulations of ketoconazole should be avoided due to unreliable bioavailability. Rationale for itraconazole instead of ketoconazole includes the potential for intermittent dosing and a perceived tendency for itraconazole to be better tolerated. There is limited evidence for the use of fluconazole and terbinafine.

RECOMMENDED ANTIBIOTICS

First line:

Topical 2% chlorhexidine/2% miconazole shampoo twice weekly, with or without

Ketoconazole (5-10 mg/kg q12h to q24h PO)

Second line:

Itraconazole (5 mg/kg q24h PO) on two consecutive days a week

USAGE RECOMMENDATIONS

Shampoos:

2% chlorhexidine and 2% miconazole used twice a week and 3% chlorhexidine (active against staphylococci and *Malassezia*).

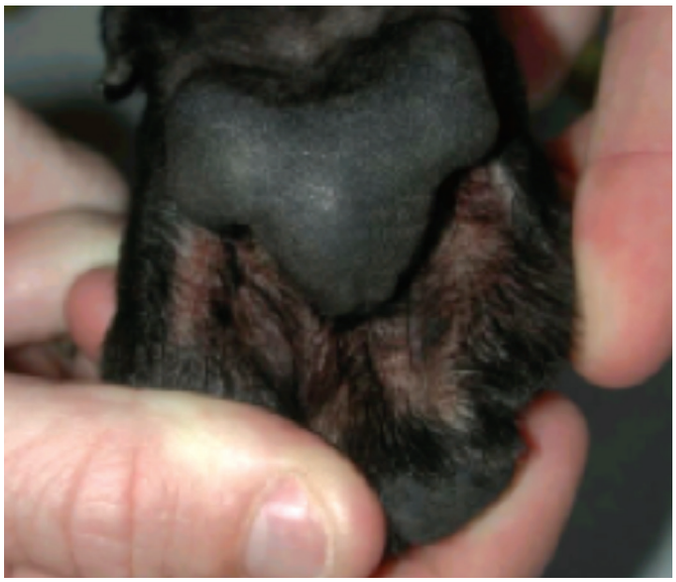
CONDITION: SUPERFICIAL YEAST (*MALASSEZIA*) INFECTIONS OF THE SKIN (NOT INCLUDING EARS)

AIDAP TOP TIPS

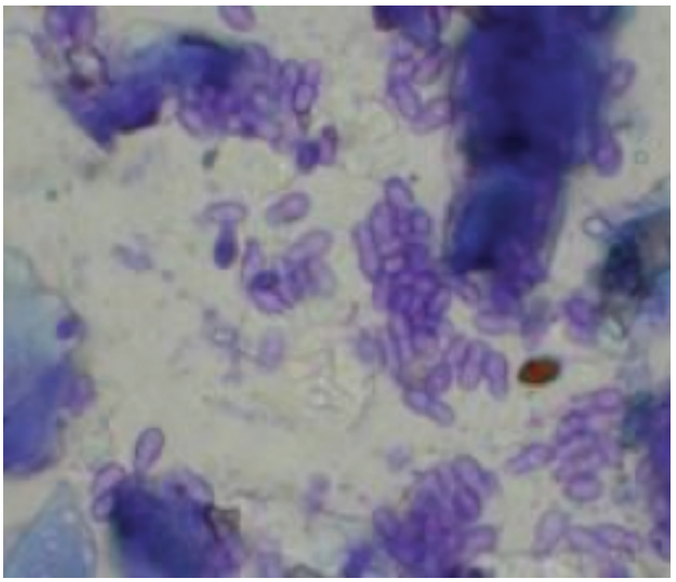
Prevention of *Malassezia*-associated skin disease in dogs

When predisposing factors cannot be identified or controlled in a dog suffering from recurrent *Malassezia* infections, regular topical or pulsed oral antifungal therapy has been recommended to minimise the frequency of infection relapses.

1. Regular shampoo bathing therapy 2% chlorhexidine and 2% miconazole (twice a week).
2. Itraconazole 5 mg/kg q24h PO (two consecutive days per week).

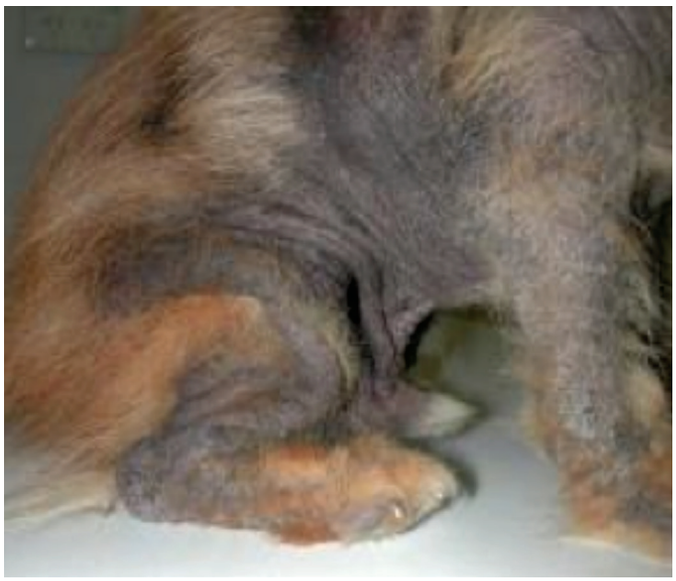


Mild interdigital erythema.

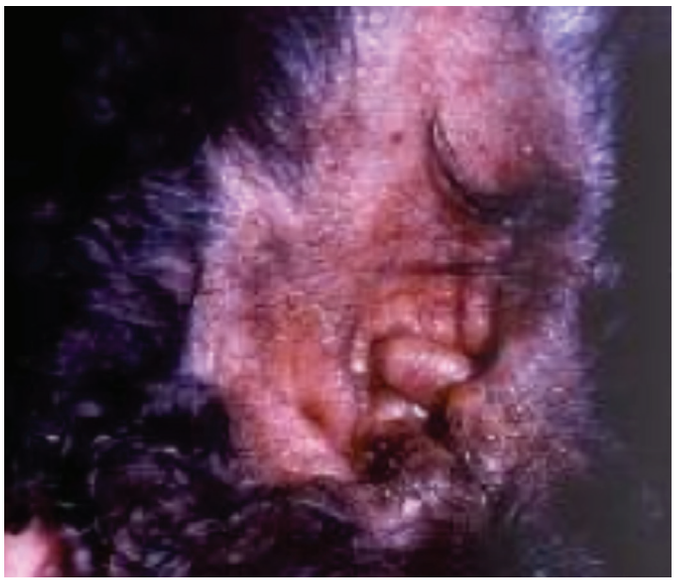


Surface cytology showing heavy *Malassezia* infection collected from the foot (on the left).

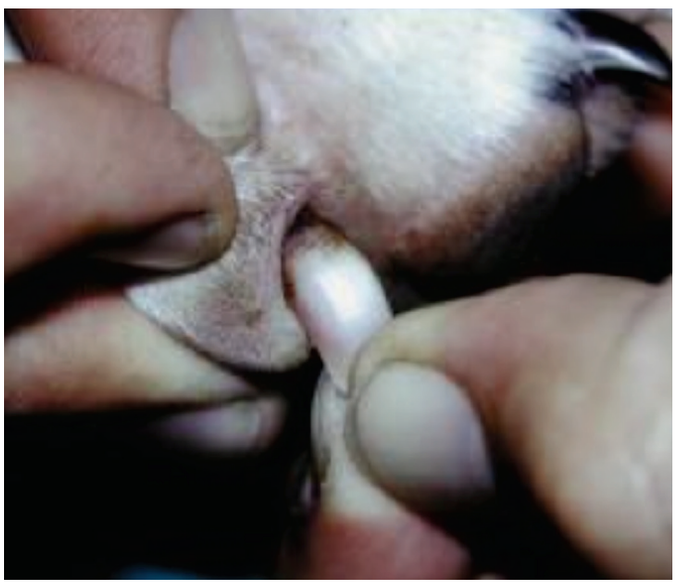
CONDITION: SUPERFICIAL YEAST (*MALASSEZIA*) INFECTIONS OF THE SKIN (NOT INCLUDING EARS)



Alopecia, lichenification, greasiness, scale formation characteristic of yeast infection.



Erythema, brown waxy otitis commonly seen in *Malassezia* otitis.



Discolouration of base of nail commonly seen in association with paronychia.

Photos courtesy of Dr Mandy Burrows & Dr Mike Shipstone.

Key references:

1. Bond R, Morris DO, Guillot J, et al. Veterinary Dermatology. 2020; 31(1): 28-74.
2. Ianiri G, Heitman J, Scheynius A. J. Invest. Dermatol. 2018; 138: 1026–1029. doi: 10.1016/j.jid.2018.01.008.

CONDITION: OTITIS EXTERNA (UNCOMPLICATED, FIRST EPISODE AND COMPLICATED, RECURRENT)

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

The external ear canal of most normal dogs harbours low numbers of a variety of commensal and potentially pathogenic bacteria with between 2 and 52% of healthy ears having bacteria identified on culture.

The normal flora is generally Gram-positive, with higher bacterial counts retrieved from the vertical external ear canal than the horizontal ear canal. Commensal and pathogenic bacteria rapidly colonise the external ear canal where changes in the microclimate following inflammation modify the environment. The microbial proliferation exacerbates and perpetuates the inflammatory response within the ear canal. Once inflamed, there is a shift towards increased bacterial numbers, initially coagulase positive staphylococci and with more chronic inflammation, Gram-negative bacteria.

Because potential pathogens can be recovered in the absence of disease (as they can from the skin surface), it is assumed that they are unable to initiate disease in the ear. However, once the ear becomes inflamed or macerated, proliferation may occur, therefore bacteria are considered perpetuating rather than primary or predisposing factors in otitis externa.

In dogs, coagulase positive *Staphylococcus* spp. (*S. pseudintermedius*, *S. schleiferi*) are mostly isolated in acute otitis and as a single organism. *Streptococcus* spp., *Pseudomonas aeruginosa*, *Proteus mirabilis, E. coli, Corynebacterium* spp., *Klebsiella* spp. are also frequently identified. Pseudomonad organisms are frequently identified in chronic recurrent cases or those cases that have had long term antimicrobial treatment.

In cats, *Staphylococcus pseudintermedius* and *Pasteurella multocida* are mostly isolated from otitis externa cases. *Malassezia* are relatively more important and have been found in more than 95% of cases of otitis externa.

CONDITION: OTITIS EXTERNA (UNCOMPLICATED, FIRST EPISODE AND COMPLICATED, RECURRENT)

TESTS FOR DIAGNOSIS

In many cases of otitis, a single organism can be isolated on bacterial culture of exudate, but in others, multiple potentially pathogenic organisms are identified. It is of critical importance to also conduct cytological examination of the otic discharge when a C&S test is performed. This allows determination of the dominant population of bacteria evident, the presence of leukocytes, and the presence of phagocytosed bacteria.

Cytology is the first step. It is mandatory in ALL cases of otitis externa and should be repeated at each visit. Studies have shown that cytology is more sensitive than C&S testing in identifying the presence of bacteria or yeast. For example, sensitivity of cytology for detection of Gram-positive cocci, Gram-negative rods, and yeasts was 84%, 100% and 100%, respectively. The sensitivity of culture for detection of these organisms was 59%, 69% and 50%, respectively.

Normal cerumen does not have high stain uptake because of the high lipid content. Outlines of occasional squames may be seen. Inflammation leads to increased numbers of squames (some of which may be nucleated indicting faster epithelial turnover with incomplete keratinisation before desquamation). As the severity of inflammation increases, inflammatory cells may be seen along with increasing numbers of organisms. Higher cellular content of cerumen may also be appreciated by increasing stain uptake on the stained slide (before microscopic examination is even started).

The number of organisms and inflammation should be assessed on a scale of 1 to 4. Normal ears may have a few yeast and Gram-positive cocci per oil field but **not** rods. The finding of yeast or cocci should be correlated with the findings of the otoscopic examination. Some animals may have few organisms yet show marked inflammation and exudation, whilst others seem to be able to tolerate quite large numbers without any pathologic changes. Repeating the cytology at each revisit allows accurate assessment of response to therapy. Medical treatment should continue until otoscopic and cytologic examinations demonstrate no pathologic change.

KEY STEPS

Otic examination alone is not sufficient, and the following minimum database is necessary to identify both the nature and type of the otitis as well as any underlying primary or predisposing factors:

01 Thorough dermatological history.

02 Complete physical examination of all areas of integument.

03 Thorough otic examination (may require sedation/anaesthesia – particularly cats).

04 Otic cytology.

05 Implement topical antimicrobial therapy based on cytological findings.

06 Systemic antibiotic therapy is not indicated for otitis externa.

CONDITION: OTITIS EXTERNA (UNCOMPLICATED, FIRST EPISODE AND COMPLICATED, RECURRENT)

TREATMENT

Topical therapy is the key to successful resolution of the majority of cases of otitis externa which is essentially a surface infection. Essential to this therapy is the successful removal of exudate. If the medication cannot penetrate the full length of the ear canal, then treatment is likely to fail. The choice of appropriate active ingredients and vehicles for treatment of otitis externa is usually made empirically based on cytological examination of ear canal exudates and otoscopic examination of the inflamed ear canals.

Most commercially produced topical products contain one or more antibacterial, antifungal, and anti-inflammatory agents in various combinations as well as vehicles, solubilisers, stabilisers and surfactants.

Clients need to be shown how to administer medications correctly. Failure to do this is a significant cause for treatment failure. An adequate volume of medication must be delivered to line the entire canal. Getting clients to count drops increases the time for administration and fundamentally means that the nozzle of the bottle is not in the canal, reducing penetration of the medication. Putting the nozzle of the bottle in the canal and telling clients to use a "squeeze" means that both under and overdosing are risked because the amount to medication is not measured out.

A graduated syringe is recommended to accurately measure ear medications and dispense them into the ear canal.

A broad guideline depending on the length and diameter of the ear canal would be:

1. 0.15-0.2 mL for a cat or a Shih tzu.
2. 0.6 mL for a Labrador.
3. 1 mL for a German Shepherd or very large dog.
4. Twice daily dosing may require slightly smaller volumes to avoid overdosing.
5. It is important to remember that the bulla of cats is divided by an incomplete bony septum. This septum is rooved by a sympathetic nerve plexus that can be easily damaged causing Horner's syndrome. Therefore, products should be used with caution if the tympanum is ruptured.

Duration of therapy

For acute disease a minimum of 5 to 14 days therapy depending on the degree of inflammatory change (oedema, hyperplasia, and erosion, ulceration) is to be expected. Rechecks every one to two weeks are necessary to ensure that ears are cytologically and otoscopically resolved prior to cessation of therapy. It is not uncommon to have a dog clinically resolved with otoscopically normal ears because of anti-inflammatory medications, where cytology is still not normal.

CONDITION: OTITIS EXTERNA (UNCOMPLICATED, FIRST EPISODE AND COMPLICATED, RECURRENT)

Antimicrobial therapy for ears with mainly cocci on cytology

Coccoid organisms will be *Staphylococcus* spp. or *Streptococcus* spp. The challenge for empirical therapy for cocci is the relative resistance of streptococci to some of the routine antibiotics, which otherwise tend to have reasonable activity for most *Staphylococcus* spp. Products containing antibiotics with good efficacy against both bacteria are desirable. Enrofloxacin, marbofloxacin or chloramphenicol are reasonable choices. The use of florfenicol (Osurnia®) should be limited to dogs with evidence of MRSP on culture and susceptibility testing.

When the tympanic membrane is ruptured, enrofloxacin is preferred although its activity against streptococcal infections is not always reliable. If this inadequate, then options include the use of systemic antibiotics based on C&S testing and ear wicks impregnated with more effective (but not middle ear safe) ointments.

Systemic antibiotics are used if there is significant involvement of the pinna, if a methicillin-resistant staphylococcal infection is identified on C&S testing or if otitis media is evident. They are *unreliable* in our experience used as a sole therapy of otitis externa.

Antimicrobial therapy for ears with mainly rods on cytology

Rods are rarely found in healthy ears. In Australia, most rods identified on culture are *Pseudomonas aeruginosa* with *Proteus* and *E. coli* identified in about 11% to 20% of the otitis ears. Less common rods include *Corynebacterium* spp. and *Klebsiella. Corynebacterium* is usually found as part of a mixed culture and is probably of minimal significance unless isolated in pure growth.

EDTA (Otoflush®) acts as a chelating agent and enhances activity of topical antibiotics against otic pathogens by decreasing stability and increasing permeability of the cell wall. The ear canal should be filled with the solution 15 to 30 minutes before a topical antibiotic is applied every 12 hours. First line antibiotic therapy includes enrofloxacin compounded 1.5% with dexamethasone. Once the tympanic membrane is intact and the inflammation controlled then products containing gentamicin (Otomax® q12h and Mometamax® q24h) can be used if the ear is clean. C&S testing is indicated if the infection fails to respond. Marbofloxacin (Aurizon®) q12h or ciprofloxacin (Cipro HC®) can be used topically as a second-line antibiotic. Systemic antibiotics are only used if there is significant involvement of the pinna or if otitis media is evident. They are unreliable in our experience used as a sole therapy of otitis externa.

CONDITION: OTITIS EXTERNA (UNCOMPLICATED, FIRST EPISODE AND COMPLICATED, RECURRENT)

Antimicrobial therapy for ears with mainly yeast on cytology

Malassezia can be retrieved from up to 80% of otitis ears in dogs. Dogs with atopy can produce IgE to *Malassezia* which means that the degree of inflammation depends on the host response rather than the numbers of yeast. *Malassezia* can be retrieved from up to 95% of otitis ears in cats. In some cases, the inflammation seen is disproportionately large compared with the number of organisms seen on cytology.

Disinfectants are useful as sole therapy where there are low numbers of yeast and minimal inflammation or occasionally in cases apparently resistant to other antifungal ear medications. The only one with any proven efficacy against *Malassezia* is Epiotic®. This is not a good ceruminolytic so penetration is an issue where there is significant exudate. Alpha Ear Cleaner® has good activity against yeast and is a good ceruminolytic. None of these products are middle ear safe.

A new product (Sonotix®) containing tromethamine, isopropyl alcohol, ethoxydiglycol, capric glycerides is an effective cerumenolytic.

Most of the major commercial combination ear products (except Baytril Otic®) are reliable in the therapy of an uncomplicated yeast otitis. Surolan® q12h or Otomax® q12h / Mometamax® q24h containing miconazole and clotrimazole, are useful first line treatments. None of these products are middle ear safe.

Systemic use of antifungal medication is a consideration where there is a fungal otitis media and for sole or adjunctive therapy where topical medications are not possible or there are severe proliferative changes in the ear canal.

Secondary changes are sequelae that occur due to acute and chronic inflammation of the external ear canal that when present will increase the likelihood of relapse of otitis externa irrespective of whether the trigger factor has been controlled. Sequelae secondary to otitis externa include epidermal or glandular hyperplasia, inflammatory polyps, fibrosis, stenosis, calcification, ceruminoliths, otitis media and complete occlusion of the external ear canals.

CONDITION: OTITIS EXTERNA (UNCOMPLICATED, FIRST EPISODE AND COMPLICATED, RECURRENT)

AIDAP TOP TIPS

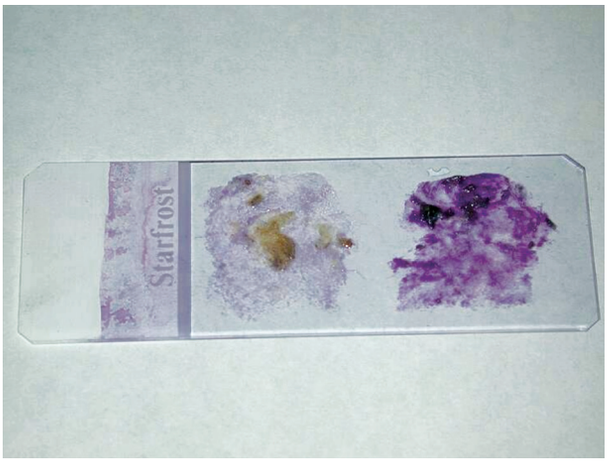
Bacterial C+S testing

The commonly accepted practice is that a bacterial C+S testing should be performed if:

- Rods are seen on cytology
- Ulceration of the epithelium is present
- The condition is recurrent
- There is no response to appropriate treatment
- Otitis media is present.

However there have been several studies raising doubts as to the usefulness and accuracy of culture results (Graham-Minze and Rosser 2004). It has been suggested that the culture may identify organisms from the external ear canal that are low in number and possibly irrelevant in the pathogenesis of the disease state. As such the initial cytology may be a better indicator of the relative importance of the different organisms present.

Robson (2008) has proposed the following:
"That bacterial C+S testing should be performed when cytology shows a uniform or near uniform pattern of bacteria AND when appropriate empirical therapy has failed AND all other causes of failure of therapy have been ruled out as well as causes of otitis media".



Sample on right showing marked stain uptake due to presence of neutrophils.

Photo courtesy of Dr Mandy Burrows & Dr Mike Shipstone.

Key references:

1. Sharma VD and Rhodes HE. J. Sm An Pract. 1975; 156: 241–247.
2. McCarthy G and Kelly WR. Irish V J. 1983; 36: 53–56.
3. Colombini S, Merchant SR, Hosgood G. Vet Dermatol. 2000; 196: 84–90.
4. Graham-Minze CA and Rosser EJ J. Am An Hosp Assoc. 2004; 40(2): 102–108.

CONDITION: SUBCUTANEOUS ABSCESS/CELLULITIS

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Infection will often heal spontaneously if there is sufficient oxygenation of the wound and drainage of purulent and necrotic debris

Cat fight abscesses are polymicrobial infections (mostly anaerobic) that usually result from inoculation of the microbial biofilm from the gingival cleft deep into a bite wound or from the inoculation of soil saprophytes via the claws. Most frequently, the subcutaneous tissues and underlying muscle are damaged by the crushing action of the long canine teeth of the feline assailant, releasing myoglobin and hence iron, but leave little connection to the atmosphere due to the lack of ripping action seen in dog bite wounds. The reduced oxygen environment sets the scene for the development of an **anaerobic infection.** This starts as a cellulitis and will usually evolve into an abscess that may eventually 'point' and burst, discharging pus to the skin surface. Once this occurs, the infection will often heal spontaneously if there is sufficient oxygenation of the wound and drainage of purulent and necrotic debris, especially if the location of the abscess is amenable to licking and the debriding action of the victim's tongue.

The anaerobic nature of the infection results in a characteristic foetid odour (due to volatile fatty acids and other fermentation products of the anaerobic bacteria). **Infections are typically polymicrobial,** involving variable combinations of a variety of obligate anaerobic organisms *(Bacteriodes* spp.*, Porphyromonas* spp.*, Fusobacterium* spp.*, Prevotella* spp.*, Peptostreptococcus* spp.*)* with facultative anaerobic organisms, such as *Pasteurella multocida* and *Streptococcus* spp.

Infrequent organisms:
Nocardia spp. (especially *N. nova*), rapidly growing mycobacteria, *Corynebacterium* spp., *Rhodococcus equi* or other soil or environmental organisms. These are usually inoculated from the claws of the assailant cat.

CONDITION: SUBCUTANEOUS ABSCESS/CELLULITIS

TESTS FOR DIAGNOSIS

Infection is readily confirmed by making smears of the purulent exudate with subsequent Gram staining (or Diff-Quik® staining) which shows **multiple bacterial morphotypes** particularly Gram-negative, fusiform rods, and Gram-positive cocci, and filamentous forms.

C&S is not necessary in most cases. Although theoretically of great interest, it requires anaerobic collection and processing of specimens. This is rarely done except in research settings. The susceptibility pattern of key bacteria is predictable and has not appeared to change over the last 30 years. Cases caused by one of the infrequent organisms listed may require C&S to determine susceptibility.

KEY ISSUES

01 Deep puncture wounds.

02 Crushing results in haemorrhage and myonecrosis.

03 Absence of exposure to air permits an anaerobic environment to develop.

04 Toxin elaborated by organisms may cause further tissue necrosis and signs of sepsis (fever, malaise, etc.).

05 Variable fibrosis occurs to localise the infection.

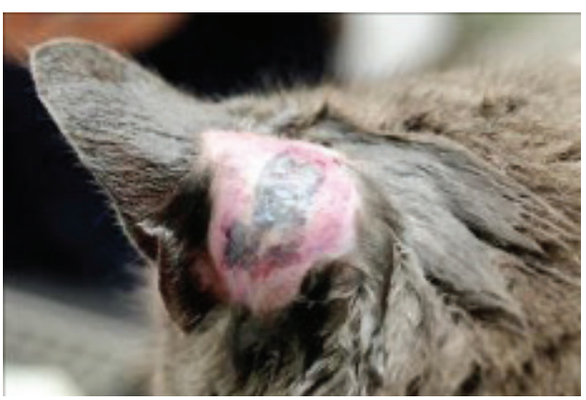
06 Death of overlying skin results in the abscess discharging pus, which can lead to spontaneous resolution.

CONDITION: SUBCUTANEOUS ABSCESS/CELLULITIS

TREATMENT

1. Surgical debridement, abscess drainage and lavage are the first lines of treatment. They allow aeration of the wound, and removal of organisms, purulent debris, and devitalised tissue all of which facilitates wound healing. Antimicrobial therapy may be considered depending on the level of complication, depth of the wound, ability of the open wound to drain (with or without a latex drain) and proximity to tendons, joints, and other vital structures. If the wound is clean and/or granulation tissue is well established, open wound management without antibiotics is feasible.
2. Intravenous (during anaesthesia) and/or subcutaneous fluid therapy may be required to re-establish and maintain hydration.
3. Opioid analgesia may be indicated in some cases.
4. The practitioner should be wary of using any NSAID in a potentially dehydrated cat, subjected to sepsis and general anaesthesia. These drugs are much more safely given from day 2 onwards, once the cat has resumed eating and drinking normally.

Example of a fulminant cat fight abscess.



Dermal necrosis associated with a cat fight abscess.
Photographs courtesy of Dr Anne Quain.

CONDITION: SUBCUTANEOUS ABSCESS/CELLULITIS

RECOMMENDED ANTIBIOTICS

First line:

Amoxicillin
(22 mg/kg q12h PO; 7.5 mg/kg q24h SC or IM)
Amoxicillin-clavulanate
(20 mg/kg q12h PO)
Cephalexin
(30 mg/kg q12h PO)
Doxycycline monohydrate
(5 mg/kg q12h PO†)
Clindamycin
(5.5 mg/kg q12h PO)
Metronidazole
(10 mg/kg q12h PO)

Second line:

Cases that fail to respond to standard therapy should be sampled appropriately (usually via fine needle aspiration from deep within the lesion) to permit cytology (Diff-Quik®), Gram staining, and C&S. This is often needed for the infrequent pathogens listed previously.

Third line:

Cefovecin (8 mg/kg SC) is suitable for cases where there are concerns of compliance, or there are difficulties with oral dosing.

†Ensure doxycycline is given with food and water bowl provided.

USAGE RECOMMENDATION

There is no evidence-base regarding treatment duration. The panel recommends that 4-5 days of therapy is probably adequate in simple uncomplicated cases. In more complicated cases 7-10 days may be required.

Where injectable formulations are available (for example, amoxicillin, amoxicillin-clavulanate), treatment initially by injection (SC or IM), and subsequently PO may be warranted (for example, cellulitis with accompanying pyrexia).

Currently registered veterinary fluoroquinolones are INAPPROPRIATE in this setting as most fluoroquinolones apart from pradofloxacin have no activity against anaerobes.

CONDITION: SUBCUTANEOUS ABSCESS/CELLULITIS

AIDAP TOP TIPS

Adequate drainage, debridement, copious lavage with saline and the resulting aeration of the wound is the cornerstone of therapy, although AIDAP recommends adjunctive antimicrobial therapy in instances where the cat is systemically unwell (fever, lack of appetite, etc.) or where the infection has a substantial cellulitis component extending to involve contiguous tissues.

Key references:

1. Roy J, Messier S, Labrecque O, Cox WR. Canadian Veterinary Journal. 2007; 48: 607-611.
2. Love DN, Malik R, Norris JM. Vet Microbiol. 2000; 74: 179–193.
3. Norris JM and Love DN. Vet Microbiol. 1999; 65: 115–122.
4. Dendle C and Looke D. Aust Fam Physician. 2009; 38(11): 868–874.

CONDITION: USE OF ANTIBIOTICS IN DENTAL PROPHYLAXIS (PERIODONTAL THERAPY)

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Most bacteria found in the mouths of dogs and cats are similar to those recovered from bite wounds.

Cats appear to have a more diverse oral microbiota when compared to dogs. Certain bacterial species are associated with periodontal disease (PD) in dogs and cats. Gram-negative anaerobes such as *Porphyromonas* spp., *Prevotella* spp., *Fusobacterium* spp. and spirochaetes (*Treponema* spp.) comprise most bacterial species found in mild to severe forms of PD in dogs and cats. However, Gram-positive anaerobes such as *Peptostreptococcus* also feature in significant numbers in PD in dogs and cats, unlike in humans, where Gram-positive anaerobes do not feature in the pathogenesis of periodontal disease. *Pasteurella multocida* and anaerobic Gram-negative rods including *Capnocytophaga* are frequently involved, and these are all susceptible to ß-lactam antibiotics including penicillin, amoxicillin, amoxicillin-clavulanate and first-generation cephalosporins.

Numerous studies have shown that a transient bacteraemia can occur in both humans and dogs after dental procedures. This bacteraemia nearly always occurs following tooth extraction(s). In a healthy dog, the bacteraemia is cleared in about 30 minutes, however, in animals with diseases (such as renal, cardiac or hepatic dysfunction), compromised immune systems, poorly controlled diabetes mellitus or other serious endocrinopathies, it is recommended that antimicrobial therapy be given prophylactically at the time of surgery. Perioperative antibiotics may also be indicated in patients with less common systemic risk factors such as subaortic stenosis and orthopaedic implants placed in the last 12–18 months. If unsure, it is recommended to consult with the cardiologist or surgeon prior to commencing.

CONDITION: USE OF ANTIBIOTICS IN DENTAL PROPHYLAXIS (PERIODONTAL THERAPY)

If the trabecular bone and the outer cortical plates of the jaws are invaded by bacteria and their toxins, as well as by host inflammatory mediators, osteomyelitis can occur, which then requires an extended course of antimicrobials.

Pre-wiping the teeth and gingivae with chlorhexidene digluconate 0.12% is also helpful prior to performing a dental cleaning or tooth extraction. It has been shown to reduce the number of aerosolised bacteria during these procedures, reducing the risk of inhalation by both the operator and the assistant.

Most dental procedures in companion animals are classified as clean-contaminated procedures, meaning that following tooth extractions systemic antibiotics are usually not indicated unless there is marked oral inflammation or purulent discharge associated with the affected teeth.

Periodontal Disease stage 4 (PD stage 4) is where there is >50% attachment loss or class 3 furcation, which is evident on intraoral radiographs. Preoperative antibiotics commenced several days before surgery may be administered in cases of PD stage 4 for the purpose of reducing the bacterial load in the oral cavity and making tissues more amenable to surgical handling. This should also be the case when dealing with gingivostomatitis cases where selective or whole mouth extractions are planned.

CONDITION: USE OF ANTIBIOTICS IN DENTAL PROPHYLAXIS (PERIODONTAL THERAPY)

TESTS FOR DIAGNOSIS

- Diagnostic tests for PD include visual and tactile inspection, periodontal probing, and intraoral radiographs. These tests are all included in an extra- and intra-oral examination or the recently termed, comprehensive oral health assessment and treatment (COHAT), which is performed under general anaesthesia. Especially in cats (and sometimes in dogs), the detection of tooth resorptions at the cervical margin is performed both with tactile examination (using an explorer probe) as well as the use of intraoral radiographs.

  Any localised or asymmetrical lesion in the oral cavity should be biopsied to rule out other oral pathology.

  Because of the large number of bacterial species in the mouth, C&S testing is usually unrewarding.

TREATMENT

A dental prophylaxis (or the preferred term, periodontal therapy) will include a COHAT, followed by dental cleaning including removal of plaque and calculus above and below the gums (supra and subgingival debridement). Periodontal therapy will include extraction of poor to hopeless prognosis teeth (furcation 3 and/or >50 % attachment loss around the tooth).

The use of 10% povidone-iodine disinfectant following mechanical periodontal therapy has been shown in human dentistry to reduce periodontal pocket depth (at least in deeper pockets >5 mm) when used as a final subgingival irrigation (needs up to 5 minutes contact time within the pocket).

Normally, antibiotics are not required for extractions unless dealing with a surgical extraction (where a mucoperiosteal flap is raised and bone removed), or multiple extractions associated with severe periodontitis.

KEY ISSUES

- Prophylactic antibiotics (if required) are best administered prior to the procedure. For example, procaine penicillin, amoxicillin or amoxicillin-clavulanate can be administered SC or IM at the time of premedication or shorter acting benzyl penicillin or first-generation cephalosporin (cefazolin) can be given IV immediately after anaesthetic induction. These would cover the great majority of potential pathogens in this setting.

CONDITION: USE OF ANTIBIOTICS IN DENTAL PROPHYLAXIS (PERIODONTAL THERAPY)

RECOMMENDED ANTIBIOTICS

First line:

Penicillin
(Procaine penicillin 30 mg/kg q24h IM or SC dogs, SC cats)

Amoxicillin
(22 mg/kg q12h PO; 7.5 mg/Kg q24h SC or IM)

Second line:

Amoxicillin-clavulanate
(20 mg/kg q12h PO; 8.75 mg/kg q24h SC or IM)

Clindamycin
(5.5 mg/kg q12h PO)

Doxycycline monohydrate
(5 mg/kg q12h PO†)

Third line:

Cefovecin (8 mg/kg SC) is suitable for cases where there are concerns of compliance, or there are difficulties with oral dosing.

†Ensure doxycycline is given with food and access to drinking water.

USAGE RECOMMENDATION

Penicillin or amoxicillin can be given prior to the dental procedure and may be continued q12h PO only in those situations of moderate to severe periodontitis, multiple extractions, or extensive oral surgery or those with moderate co-morbidities.

A perioperative dose of amoxicillin or amoxicillin-clavulanate can be given at the time of premedication (30 to 60 minutes prior to procedure) when performing surgical extractions. Where the patient has severe periodontitis and multiple extractions are anticipated, a preoperative and postoperative course of antibiotics may be required (determined on a case-by-case basis).

CONDITION: USE OF ANTIBIOTICS IN DENTAL PROPHYLAXIS (PERIODONTAL THERAPY)

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CONDITION: CHRONIC GINGIVOSTOMATITIS

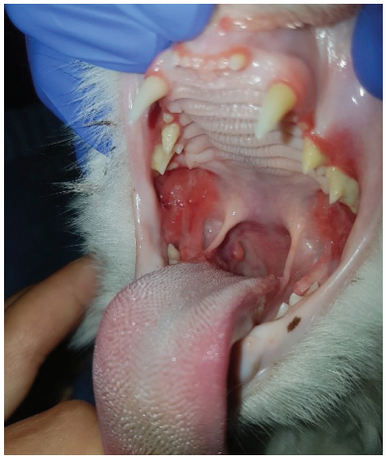
BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Feline chronic gingivostomatitis (FCGS) is a severe inflammatory disease involving various areas of the oral cavity, including the gingivae, oral mucosa, and oropharyngeal region (lateral to the palatoglossal folds).

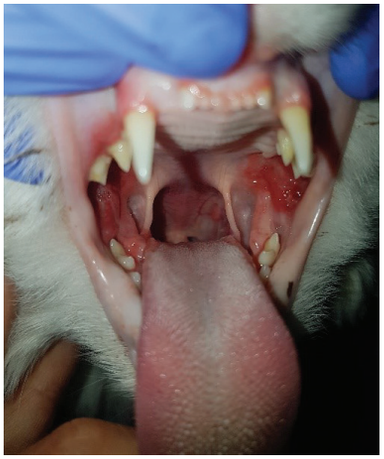
FCGS causes significant pain to the point of inappetence and food aversion. The disease affects a wide range of ages and can affect any breed of cat. There appears to be no sex predilection. The disease involves marked inflammation, proliferation and/or ulceration of the gingival tissues which spreads to the rest of the mouth and can include the buccal mucosa, lip commissures and the oropharyngeal region (caudal stomatitis with inflammation lateral to the palatoglossal folds).

Chronic gingivostomatitis signifies a syndrome in which the inflammatory response in the gingivae and contiguous tissues is disproportionate to the amount of plaque and tartar. It has led some to question whether the disease process is more akin to an immune-mediated disease. The aetiology of gingivostomatitis remains uncertain, but it may be multifactorial, involving an aberrant inflammatory response, possibly to bacterial pathogens and/or viruses and other non-infectious antigens (food allergens).

Clinical signs are associated with pain. The affected cat will often be reluctant to eat or have difficulty with prehending food, have an unkempt coat due to lack of grooming, lose weight and not socialise within the household.



Feline chronic gingivostomatitis.



Feline chronic gingivostomatitis with caudal lesions.

Photographs courtesy of Dr Anthony Caiafa.

CONDITION: CHRONIC GINGIVOSTOMATITIS

TESTS FOR DIAGNOSIS

1. Examination of the oral cavity under general anaesthesia is necessary due to the level of pain in the awake animal.
2. A COHAT including periodontal probing and dental radiographs are essential to formulate a management plan and to rule out other diseases such as tooth resorptions.
3. FIV/FeLV testing should be performed.
4. Multiple gingival biopsies are helpful to determine the underlying disease process(es) and to rule out other pathology, especially if the inflammation is localised within the mouth. Consider PCR on fresh gingival biopsy samples or oropharyngeal mucosal swabs for feline calicivirus and feline herpesvirus.
5. Radiographs are required post-extractions to confirm complete tooth removal and rule out any remaining bony sequestra or bony protrusions.

KEY ISSUES

01 Chronic gingivostomatitis may be associated with severe periodontal disease.

02 Other infectious agents such as viruses, including feline calicivirus, feline herpesvirus (type 1), feline immunodeficiency virus and feline leukaemia virus, and bacteria such as *Pasteurella multocida* and *Bartonella henselae* have been incriminated in this disease by association.

03 At present, the causal agent(s) has not been determined.

04 It has been shown to be more prevalent in shared households.

05 Caudal stomatitis can be significant and tends to carry a poorer prognosis. The finding of caudal stomatitis can lead to a refractory state, even after surgical management of this disease. It has also been suggested that subclinical inflammation elsewhere in the gastrointestinal tract can be concurrently associated with the oral disease process.

CONDITION: CHRONIC GINGIVOSTOMATITIS

TREATMENT

1. The aim of FCGS management is to eliminate or control inflammation/pain without always achieving complete remission.

2. Stomatitis Disease Activity Index (SDAI) has been introduced for the subjective assessment of pain and inflammation before, during and after treatment.

3. Remove plaque and calculus by scaling and polishing the teeth. If the disease is mild, based on periodontal probing and intraoral radiographs, introduce an intensive active homecare program, consisting of either daily toothbrushing or the use of chlorhexidine gluconate (<0.2%) wiped onto the teeth daily. Professional cleans every 3 to 6 months may also be required as a part of the management of the disease.

4. At least, extract teeth with poor or hopeless prognosis (stage 3 or 4 periodontally compromised teeth as well as teeth with stage 2 or 3 furcation involvement).

5. **Based on clinical judgment, consider administration of a preoperative course of antimicrobials 2-3 days prior to any extractions, followed by a 5-7 day postoperative course. Antimicrobials with good activity against obligate anaerobes involved in periodontal disease, should be considered.**

6. Further treatment options for affected cats, based on current evidence, include premolar/molar tooth extractions (partial/selective mouth extractions or SME), even when periodontal disease staging is considered mild, but there is significant soft tissue inflammation. Where significant mucosal inflammation involves the canine and incisor teeth, then full mouth extractions (FME) are considered appropriate management. All tooth extractions should include confirmed extraction of the tooth (pre and postoperative dental radiographs), curettage of the periodontal ligament followed by alveolectomy, or remodelling of the crestal bone and wound closure.

7. Other treatment options include the use of antiviral agents such as famciclovir or the use of immunomodulatory agents such as cyclosporine or feline recombinant interferon omega, either as a trial medication prior to tooth extractions, or more often, as a postoperative treatment where clinical improvement has been poor following SME or FME (refractory cases often with non-responsive caudal stomatitis).

CONDITION: CHRONIC GINGIVOSTOMATITIS

RECOMMENDED ANTIBIOTICS

First line:

Doxycycline monohydrate
(5 mg/kg q12h PO†)

Amoxicillin
(22 mg/kg q 12h PO; 7.5 mg/kg q24h SC or IM)

Amoxicillin-clavulanate
(20 mg/kg q12h PO; 8.75 mg/kg q24h SC or IM)

Second line:

Clindamycin (5.5 mg/kg q12h PO)
Metronidazole (10 mg/kg q12h PO)

Third line:

Cefovecin (8 mg/kg SC) is suitable for cases where there are concerns of compliance, or there are difficulties with oral dosing.

Other treatment options:

Consider topical, intra-lesional or subcutaneous feline recombinant interferon omega, or oral cyclosporine, especially in refractory cases post SME or FME extractions.

†Ensure doxycycline is given with food and water bowl provided.

USAGE RECOMMENDATION

Typically for 5-7 days, but longer in certain circumstances.

CONDITION: CHRONIC GINGIVOSTOMATITIS

AIDAP TOP TIPS

Further therapy is directed at changing the diet to include more chewing.

If partial/selective mouth extractions (SME) have been performed, then further therapy is directed at active home care to minimise plaque build-up. Adjunctive products such as chews, and diets formulated for dental care may be useful. C&S testing is of little value in this disease. The bacteria involved are fastidious obligate anaerobes and it is difficult to collect a meaningful specimen and transport it to the laboratory in a timely manner, that would permit the causal organisms to be cultured. Susceptibility testing for anaerobes is not available routinely in any private or institutional veterinary laboratories in Australia currently.

Other techniques such as qPCR or immunohistology for FCV or FHV on biopsy samples, are routinely available via specialised veterinary diagnostic laboratories in Australia and can be useful in a general practice setting.

Unfortunately, in some affected cats, oral inflammation may not completely resolve with the management methods described above, but the main goal of having a cat who is comfortable and eating, whilst still having some oral inflammation, is achievable.

CONDITION: CHRONIC GINGIVOSTOMATITIS

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CONDITION: FELINE UPPER RESPIRATORY TRACT DISEASE

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Acute upper respiratory tract disease (URTD) describes any acute illness of ⩽10 days duration characterised by sneezing, nasal discharge (serous to mucopurulent) and/or conjunctivitis.

Most cats with acute URTD have an upper respiratory tract infection (URTI) caused by feline herpesvirus (FHV-1) or feline calicivirus (FCV). Influenza viruses very occasionally cause feline URTI in some areas of the world. Primary bacterial URT pathogens include *Bordetella bronchiseptica* and *Chlamydia felis.* Members of the URT microbiome including *Pasteurella multocida, Staphylococcus* spp., *Streptococcus* spp., *Escherichia coli* and anaerobes can cause secondary bacterial infections. *Mycoplasma* spp. and *Streptococcus equi* subsp. *zooepidemicus* can be primary pathogens, or secondary opportunists. The most common bacteria detected in the nasal cavity of cats with acute URTD are *C. felis, Staphylococcus, Pasteurella, Streptococcus* and *Moraxella* spp.

Cats with URTI may have single-agent infections or, more frequently, are co-infected with viruses or bacteria.

Clinical signs may be mixed, or nasal or ocular signs may predominate, depending on the infectious agents, as well as host-factors such as age. For example, some FHV-1 infections may be characterised by severe rhinitis with nasal turbinate destruction, while others present with only ocular signs (for example, corneal ulceration). Ocular signs often predominate in *C. felis* infections (for example, conjunctival oedema (chemosis) or hyperaemia).

If nasal discharge is serous, a primary or secondary bacterial infection is less likely and antimicrobial therapy is not indicated.

CONDITION: FELINE UPPER RESPIRATORY TRACT DISEASE

TESTS FOR DIAGNOSIS

1. Nasal swab bacterial C&S testing is not indicated in cats with acute URTD (see above).
2. Results of molecular (PCR) respiratory panels are of low value (see above).
3. In multi-cat environments with outbreaks of acute URTD, respiratory PCR panels may be useful to identify the causative agents and guide therapeutic recommendations. Several cats should be sampled to increase the chance of a definitive diagnosis.
4. Radiography, endoscopy, or advanced diagnostic imaging (CT or MRI) is not warranted in acute URTD unless a foreign body is suspected.

KEY ISSUES

01 FHV-1 and FCV are the most common causes of acute URTD in cats.

02 Some bacteria are primary pathogens and cause acute URTD, for example, *Chlamydia felis*.

03 Most bacterial infections are secondary, involving members of the URT microbiome.

04 Co-infections involving ≥1 virus and/or bacteria are common in acute URTD.

05 Nasal swab bacterial C&S testing is not indicated in cats with acute URTD since non-causative commensals are frequently cultured and primary pathogens such as *Mycoplasma* spp. and *C. felis* do not grow on standard laboratory media.

06 Results of molecular (PCR) respiratory panels are of low value in individual cats since FHV-1, FCV, *Mycoplasma* spp. and *C. felis* can be amplified in both healthy and diseased cats and in cats that have been recently vaccinated using attenuated pathogen strains.

07 Antimicrobial therapy is only recommended in cats with purulent or mucopurulent nasal discharge that are febrile, anorexic, or lethargic.

CONDITION: FELINE UPPER RESPIRATORY TRACT DISEASE

TREATMENT

1. **Antimicrobial therapy is only recommended in cats with purulent or mucopurulent nasal discharge that are febrile, anorexic, or lethargic. In most cats with URTI, disease is self-limiting within 10 days, with or without antimicrobial therapy.**

2. Supportive therapy is important for cats with acute URTD including maintenance of hydration (for example, subcutaneous fluid therapy), appetite stimulants such as mirtazapine, assisted grooming (brushing) and saline nebulisation where congestion is severe.

3. Antiviral therapy (famciclovir) is recommended where FHV-1 infection is considered likely, such as in the presence of corneal ulcers.

4. Empirical administration of famciclovir should be considered in cats with acute URTD in multi-cat environments, although the results of recent studies by Reinhard *et al* and Lucyshyn *et al* were conflicting.

5. If signs persist after administration of an antimicrobial for 7-10 days, further diagnostic investigation is recommended, for example a molecular respiratory PCR panel, before further antimicrobial therapy is considered.

CONDITION: FELINE UPPER RESPIRATORY TRACT DISEASE

RECOMMENDED ANTIBIOTICS

First line:

Doxycycline monohydrate (5 mg/kg q12h PO†) or (10 mg/kg q24h PO†)

Second-line:

Amoxicillin (22 mg/kg q12h PO)
Amoxicillin-clavulanate (20 mg/kg q12h PO)

Third line:

Azithromycin (5–10 mg/kg q12h PO on day 1, then q3 days)

Additional treatments:

Where severe disease due to FHV-1 is suspected, administer famciclovir (90 mg/kg q12h)

†Ensure doxycycline is given with food and the animal has access to drinking water.

USAGE RECOMMENDATION

Duration of therapy for all drugs listed: 7-10 days

Doxycycline generally has *in vitro* and/or in *vivo* efficacy against *C. felis*, *Mycoplasma* spp. and *B. bronchiseptica* and has activity against many opportunistic bacterial species.

Tooth enamel discoloration with doxycycline may occur in kittens <4 weeks of age. Alternative antibiotics should be considered.

Amoxicillin and amoxycillin-clavulanate have poor activity against *B. bronchiseptica, Mycoplasma* spp. and *C. felis* but have good efficacy against secondary bacterial infections.

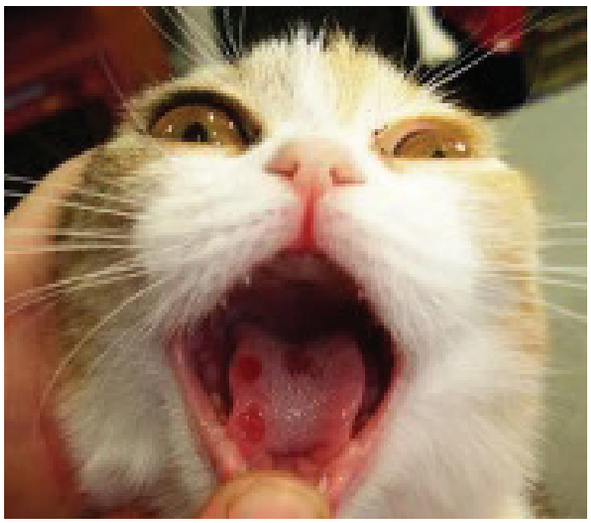
Azithromycin is less effective against *C. felis* than doxycycline and is not registered for use in animals. It should not be used off-label except in exceptional circumstances for individual animals.

AIDAP TOP TIPS

If signs persist after administration of an antimicrobial for 7-10 days, further diagnostic investigation is recommended, for example a molecular respiratory PCR panel, before further antimicrobial therapy is considered.

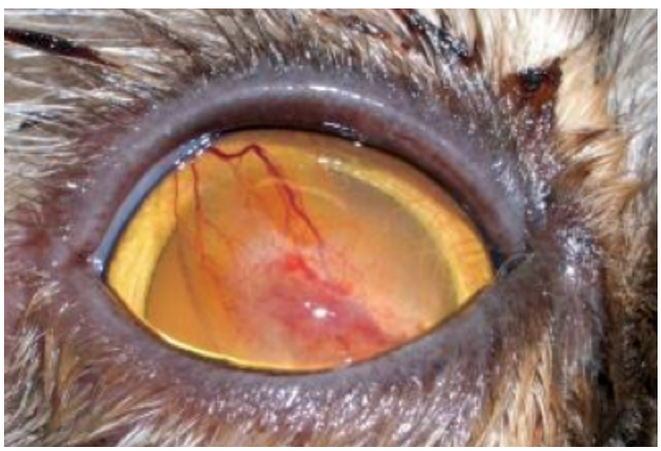
CONDITION: FELINE UPPER RESPIRATORY TRACT DISEASE

Ocular and nasal discharges in a cat with severe, acute URTD.
Photographs courtesy of Prof Vanessa Barrs.



Ocular discharge and oral ulcers in a kitten examined during veterinary screening at a cat show. Oral ulcers are most frequently associated with acute FCV infection.

FHV-1 infection in a kitten.
Secondary bacterial infections are common.
Photos courtesy of Dr Richard Malik.



Chronic feline herpes keratitis.

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CONDITION: CHRONIC RHINOSINUSITIS (CRS)

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Chronic rhinosinusitis (CRS a.k.a chronic rhinitis, idiopathic rhinitis or 'chronic snuffler'), is the second most common cause of chronic nasal discharge and sneezing (>4 weeks) in cats after neoplasia.

Previous or recurrent FHV-1 infection is implicated, but not definitively proven, in the pathogenesis of CRS. Severe early FHV-1 infection, viral persistence within nasal mucosal epithelium, or chronic reactivation of FHV-1 in nasal tissues is thought to cause inflammation, necrosis, and impaired drainage of secretions as well as dysbiosis and secondary infection by commensals (for example, *Pasteurella, Mycoplasma* spp., *E. coli, Streptococcus* spp., *Staphylococcus* spp., anaerobes), resulting in destruction of nasal turbinates.

Disease occurs at any age, but mostly in young to middle-aged cats. Some may have a history of "cat flu" as a kitten. Diagnosis is one of exclusion by ruling out other infectious agents, especially fungal, and other common causes of disease (see tests for diagnosis).

Bilateral nasal discharge in a cat with a nasopharyngeal polyp – a differential diagnosis for chronic rhinosinusitis in cats.
Photo courtesy of Dr Vanessa Barrs.

CONDITION: CHRONIC RHINOSINUSITIS (CRS)

TESTS FOR DIAGNOSIS

1. Since CRS is a diagnosis of exclusion, other diseases need to be ruled out, including infectious agents - especially fungal (in Australia cryptococcosis or aspergillosis), primary bacterial infection (rare) (for example, *C. felis, B. bronchiseptica, Mycoplasma* spp.), secondary bacterial infection due to tooth root disease or an oronasal fistula, parasitic (rare, such as lungworm), neoplasia (lymphoma or adenocarcinoma), other inflammatory diseases (for example, nasopharyngeal polyps, nasal polyps, nasopharyngeal stenosis), foreign bodies, or congenital anomalies such as choanal atresia.
2. Serum antigen tests for *Cryptococcus* spp. (latex agglutination or lateral flow assay) have high sensitivity to rule out cryptococcosis.
3. Other diagnostic techniques performed under general anaesthesia include dental probing and imaging, advanced diagnostic imaging (CT or MRI), endoscopic evaluation of the nasal cavity and nasopharynx, deep nasal lavage, biopsy, and histopathology.
4. Culture of deep nasal lavage fluid may identify secondary chronic bacterial infections and determine antimicrobial susceptibility. However, cultured bacteria may be commensals.
5. Histopathology of nasal mucosal biopsies in CRS, unless neoplastic, is often non-specific such as neutrophilic and/or lymphoplasmacytic infiltrates. Eosinophilic infiltrates might suggest FHV-1 infection or fungal infection and special stains for fungi should be requested.

KEY ISSUES

01 CRS is thought to result from previous severe or recurrent FHV-1 infection, including necrosis of nasal turbinates, accumulation of nasal secretions and secondary bacterial infection by commensals of the nasal cavity.

02 Clinical signs of CRS overlap with other common causes of nasal disease in cats including fungal infections, tooth root abscesses, inflammatory diseases such as nasopharyngeal polyps, and neoplasia.

03 Since CRS diagnosis is by exclusion, other common diseases need to be ruled out and referral to a specialist is recommended if advanced imaging or endoscopy is not available.

04 **Nasal swab bacterial C&S testing is not indicated in cats with CRS since non-causative commensals will often be cultured and primary pathogens can be isolated from healthy or diseased cats.**

CONDITION: CHRONIC RHINOSINUSITIS (CRS)

TREATMENT

- There is no definitive treatment for CRS.
- Since FHV-1 is implicated, it is possible that some cats may benefit from famciclovir, although evidence-based trials to support this are lacking.
- Antimicrobials should be reserved for cats with severe clinical signs.
- Supportive treatments such as saline nebulisation, saline nasal drops, mucolytics such as bromhexine (1 mg/kg q24h PO) or decongestants (for example dimenhydrinate (4 mg/cat q8h PO) or nasal drops (ephedrine hydrochloride (0.5%) or phenylephrine hydrochloride 2.5% 1 drop per nostril q24h for a maximum of 3 days)) may help control signs, but any benefit is anecdotal, and no evidence-based trials have been performed. In severe cases therapeutic saline irrigation under general anaesthesia may provide a period of relief from clinical signs.
- If *Pseudomonas aeruginosa* is isolated and implicated to be the cause of severe secondary infection, extensive saline irrigation of the nasal cavity under anaesthesia may be beneficial to break down and flush out loculated secretions.
- Recurrence of clinical signs is common. When recurrences occur, in a cat that has been diagnosed with CRS and where other causes of disease have been ruled out, repeat antimicrobial therapy is indicated. See usage recommendation.

CONDITION: CHRONIC RHINOSINUSITIS (CRS)

RECOMMENDED ANTIBIOTICS

In most circumstances no antimicrobials are required unless there is bacterial pneumonia or marked co-morbidities.

First line:

Doxycycline monohydrate (5 mg/kg q12h or 10 mg/kg q24h PO†)

Second line:

Clindamycin (5.5 mg/kg q12h PO)
Amoxicillin (22 mg/kg q12h PO)

Third line:

Azithromycin (5–10 mg/kg q12h PO on day 1, then q3 days)

Antiviral therapy for FHV-1 famciclovir (90 mg/kg q12h PO) may be of benefit in some cases (see treatment).

†Ensure doxycycline is given with food and the animal has access to drinking water.

USAGE RECOMMENDATION

Duration of therapy:

In the absence of data, it is recommended to treat for a minimum of 7 days, then assess the response to therapy. In cases where antimicrobial therapy has resulted in clinical improvement, treatment should be continued until one week beyond the resolution of clinical signs, or one week beyond when there is no further improvement in clinical signs.

Fluoroquinolones and third generation cephalosporins should not be prescribed unless results of C&S testing indicate that first and second-line antimicrobial agents are unlikely to be effective.

Clindamycin has activity against many anaerobic bacteria, many Gram-positive bacteria, and some mycoplasmas, but is not effective for most Gram-negative bacteria, including *Pasteurella* spp.

Azithromycin is not registered for use in animals. It should not be used off-label except in exceptional circumstances for individual animals.

When clinical signs recur, antimicrobial therapy should be restarted using the same, previously effective drug. If there is no improvement after 48 hours, change to another antimicrobial class or a more active drug within the same class. If there is no response, repeat the culture of nasal lavage fluid.

CONDITION: CHRONIC RHINOSINUSITIS (CRS)

AIDAP TOP TIPS

1. Rule out dental-associated disease and neoplasia, especially in older cats.
2. Antigen tests on serum are important to rule out cryptococcosis because Australia is a "hot-spot" for this disease. Where advanced diagnostic imaging and endoscopy is not possible, histopathology and fungal culture of nasal mucosal biopsies collected "blind" under anaesthesia using cup-biopsy forceps are useful to rule out neoplasia and aspergillosis.

Key references:

1. Quimby J and Lappin M. Compend Contin Educ Vet. 2010; 32(1): E1-E10.
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CONDITION: LOWER RESPIRATORY TRACT INFECTION (LRTI)

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

The most common bacteria to cause LRTI in cats are *Bordetella bronchiseptica*, *Pasteurella* spp., *Mycoplasma* spp., *Streptococcus* spp. and *E. coli.*

Less common bacteria include mycobacteria, *Salmonella typhimurium, Pseudomonas* spp. and obligate anaerobes. *Pasteurella* spp. and obligate anaerobes are commensals of the oropharynx and micro-aspiration of these bacteria is the most likely route of infection, for example after viral URTI or other disease resulting in impairment of muco-ciliary clearance.

B. bronchiseptica is a primary pathogen that can cause acute or chronic LRT disease. Risk factors include high population density such as in catteries or shelters, poor hygiene, young age/immunosuppression, and exposure to dogs with respiratory disease. Clinical disease is most likely in kittens less than 10 weeks of age and signs range from sneezing, ocular discharge and mild cough to severe dyspnoea and respiratory distress, which can be fatal.

Mycoplasmas are also an important cause of LRTI in cats. *M. felis*, *M. gateae*, or *M. feliminutum* can be detected by culture or PCR in up to 23% of cats with LRT disease. They can cause bronchopneumonia, focal pulmonary abscessation and pyothorax.

CONDITION: LOWER RESPIRATORY TRACT INFECTION (LRTI)

TESTS FOR DIAGNOSIS

1. Rule out non-infectious respiratory diseases such as congestive heart failure.
2. Thoracic imaging (radiography first, then possibly CT where available).
3. Haematology (full-blood count) and serum biochemistry.
4. Consider further testing for non-bacterial causes of LRT disease including faecal flotation, faecal sedimentation, heartworm serology, toxoplasmosis serology and rarely for viral or fungal causes.
5. BAL samples (for cytology and C&S if indicated) obtained via bronchoscopy, or unguided BAL.
6. PCR of BAL fluid for specific pathogens such as *Mycoplasma* spp. and *B. bronchiseptica* in some settings. *B. bronchiseptica* can also be detected by PCR of oropharyngeal swabs.

KEY ISSUES

01 Although infectious LRT disease occurs in cats, allergic lower airway disease is more common, and is a diagnosis of exclusion.

02 Mycoplasmas may cause primary LRTI or be a complication of "feline asthma".

03 ***B. bronchiseptica* is a primary respiratory pathogen and clinical disease is most common in kittens.**

04 Because of the difficulty of determining whether bacterial involvement is primary or secondary in cases with feline bronchial disease, antimicrobials are often used as a component of therapy, even when the primary aetiology is thought to be allergic.

CONDITION: LOWER RESPIRATORY TRACT INFECTION (LRTI)

TREATMENT

- Therapy should be based on antimicrobial susceptibility testing where available.
- *Mycoplasma* spp. are generally susceptible to doxycycline, macrolides, and fluoroquinolones.
- *B. bronchiseptica* is usually susceptible to doxycycline and fluoroquinolones. Resistance has often been detected to amoxicillin and trimethoprim. Amoxicillin-clavulanate is not recommended, even if susceptibility is documented, due to poor distribution into respiratory secretions. Early treatment of URT infections caused by *B. bronchiseptica* is recommended to prevent LRT involvement.
- For empirical therapy pending C&S results, or where the aetiological agent is suspected to be bacterial, doxycycline is the recommended first-choice antimicrobial and has good efficacy against *B. bronchiseptica, Pasteurella* spp., mycoplasmas, and anaerobes.

RECOMMENDED ANTIBIOTICS

Acute life-threatening bronchopneumonia
Ampicillin empirically (20 mg/kg q8h IV) and Gentamicin (5-6 mg/kg q24h IV)

together with IV fluid therapy, saline nebulisation, and thoracic percussion and coupage.

Chronic LRTI
Doxycycline monohydrate empirically (5 mg/kg q12h PO†)

Re-assess drug choices in the light of C&S data and the response to therapy.

†Ensure doxycycline given with food or water bowl provided.

USAGE RECOMMENDATION

Minimum treatment durations:

For LRTI caused by mycoplasmas or *B. bronchiseptica*, in the absence of data, six weeks therapy is recommended. For other bacterial LRT infections three weeks may be adequate.

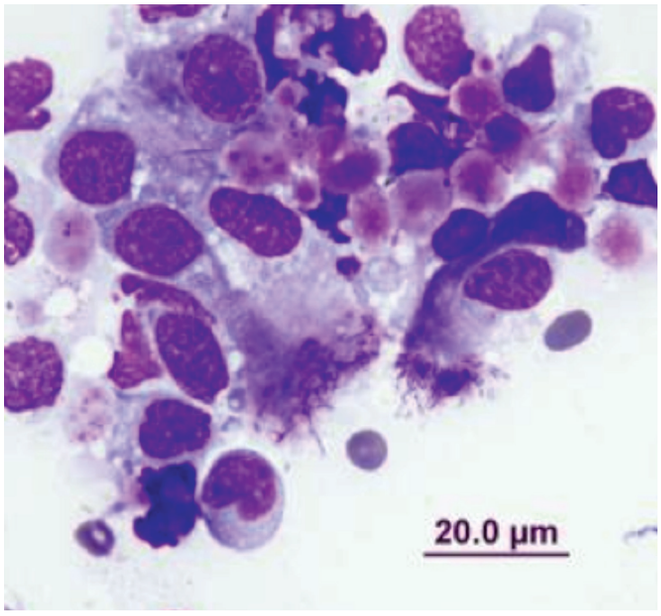
As mycoplasmas lack a peptidoglycan cell wall, they are resistant to all ß-lactam drugs.

CONDITION: LOWER RESPIRATORY TRACT INFECTION (LRTI)

AIDAP TOP TIPS

1. As well as systemic antimicrobials, nebulisation with oxygen and saline, combined with thoracic physiotherapy can be helpful in cats with severe LRTI.

2. Maintain a high index of suspicion for pulmonary toxoplasmosis in cats receiving combination therapy using prednisolone and either cyclosporine or cytotoxic drug therapy.



Diff-Quik® stained smear of bronchial lavage fluid from a cat with a *Bordetella bronchiseptica* lower respiratory tract infection. Clusters of rods are attached to ciliated respiratory epithelial cells.

Photo courtesy of Dr Patricia Martin.

Key references:

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2. Lappin MR, Blondeau J, Boothe D, Breitschwerdt EB, et al. J Vet Int Med. 2017; 31:279-294.
3. Foster SF and Martin P. J Feline Med Surg. 2011; 13(5): 313–332.
4. Bart M, Guscetti F, Zurbriggen A, et al. Vet J. 2000; 159(3): 220–230.
5. Macdonald ES and Norris CR. J Am Vet Med Assoc. 2003; 223(8): 1142–1150.
6. Foster SF, Martin P, Allan GS, et al. J Feline Med Surg. 2004; 6(3): 167–180.
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CONDITION: PYOTHORAX

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Most cases of pyothorax in cats are polymicrobial infections caused by obligate and facultative anaerobic bacteria derived from the feline oral cavity, similar to cat bite abscesses.

Isolates include Bacteroidaceae (*Bacteroides* spp., *Porphyromonas* spp., *Prevotella* spp.), *Fusobacterium* spp., *Peptostreptococcus* spp., *Actinomyces* spp., *Eubacterium* spp., *Propionibacterium* spp., *Filifactor villosus, Pasteurella multocida, Streptococcus* spp. and *Mycoplasma* spp.

Less than 20% of cases are caused by non-oropharyngeal microbiota such as *Staphylococcus* spp., *Rhodococcus equi, Nocardia* spp., enteric Gram-negative organisms *(E. coli, Salmonella spp., Klebsiella* spp., *Proteus* spp.), non-enteric Gram-negative organisms (*Pseudomonas* spp.) and protozoa (*Toxoplasma gondii*).

In contrast to dogs, infection with Enterobacteriales is uncommon, with *E. coli* only being isolated in 0–7% of cases.

Fungal causes are rare and include *Cryptococcus* spp. and *Candida albicans*.

Pyothorax occurs at any age but is mostly a disease of young cats (4–6 years). There is no breed or gender predisposition.

Possible routes of infection include extension from an adjacent structure (bronchopneumonia, para-pneumonic spread, oesophageal rupture, mediastinitis or sub-phrenic infection), direct inoculation (penetrating trauma, migrating foreign body, thoracocentesis or thoracic surgery) or haematogenous or lymphatic spread from a distant site (systemic sepsis).

Aspiration of oropharyngeal microbiota, subsequent colonisation of the LRT and direct extension of infection from the bronchi and lungs is the most common cause of feline pyothorax, as it is in human pyothorax and equine pleuropneumonia that also often involve obligate anaerobes. Viral URTI can impair muco-ciliary clearance of respiratory secretions and predispose to accumulation of aspirated oropharyngeal secretions, resulting in colonisation of the LRT then pleuropneumonia.

CONDITION: PYOTHORAX

TESTS FOR DIAGNOSIS

1. Pleural effusion can be confirmed by ultrasonography (U/S) of the thorax, followed by U/S guided thoracocentesis using a 23-gauge butterfly needle. Pleural fluid is often echogenic, and fibrinous strands may be seen between the pleural margins. Note the odour of the discharge, which is usually foul-smelling in infections with an anaerobic component.

2. If U/S is not available and respiratory distress is severe, perform thoracic radiography using only a dorsoventral view to confirm the presence of pleural effusion without causing respiratory decompensation.

3. Where U/S is not available, thoracocentesis can be performed safely at the ventral third of the 6th, 7th or 8th intercostal space with the cat standing or in ventral recumbency. Avoid the intercostal vessels and nerves located near the caudal rib margin.

4. Post-drainage radiographs (three-view) may provide evidence of underlying pulmonary disease. Thoracic CT is useful for confirmation of pulmonary disease such as abscessation.

5. Pleural fluid cytology should be performed. In-house smears can be stained with Diff-Quik® and Burke's modification of the Gram stain (if available).

6. Both aerobic and anaerobic culture of pleural fluid should be performed. Anaerobic culture requires the exclusion of air from the diagnostic sample. Inoculation into a commercial anaerobic specimen collector (anaerobic blood culture bottle) is ideal.

KEY ISSUES

01 **Most cats with pyothorax have polymicrobial infections dominated by anaerobic bacteria of oropharyngeal origin.**

02 Most cases are thought to begin in the lung after micro-aspiration of oropharyngeal microbiota with secondary spread to the pleura and pleural space. This can occur as a complication of a previous viral URTI, which may not have been noticed by the owner. Rarely, infection occurs after penetrating cat bite injuries to the thoracic wall.

03 Unlike dogs, grass seeds are rarely the cause of pyothorax in cats.

CONDITION: PYOTHORAX

TREATMENT

1. Successful treatment requires pleural drainage and lavage plus antimicrobial therapy.
2. Mortality rates are considerably higher in cats treated with daily thoracocentesis compared with indwelling chest tubes.
3. Before anaesthesia or sedation for chest tube placement, the pleural effusion should be drained as completely as possible using needle thoracocentesis to minimise anaesthetic complications. Small bore wire-guided chest drains (for example 14-gauge MILA International, Inc) are generally well tolerated.
4. If the pleural effusion is bilateral, bilateral indwelling thoracostomy tubes should be placed, with twice daily aspiration and lavage until pleural fluid formation is <2 mL/kg/day together with resolution of infection on pleural fluid cytology and on radiographic evaluation.

RECOMMENDED ANTIBIOTICS

Empiric treatment of polymicrobial feline pyothorax:

Penicillin G (benzylpenicillin potassium or sodium) (20,000-40,000 IU/kg q6h IV)
or
Ampicillin (20-40 mg/kg q6-8h IV)
or
Amoxicillin (10-20 mg/kg q12h IV)

alone or in combination with metronidazole (10 mg/kg q12h IV)

Amoxicillin-clavulanate (20 mg/kg q 12h PO)

These agents are effective against both ß-lactamase producing anaerobes and *Pasteurella* spp. Additional targeted antimicrobial therapy can be administered if supported by C&S testing, or if Gram-negative rods only are seen in smears of pleural fluid.

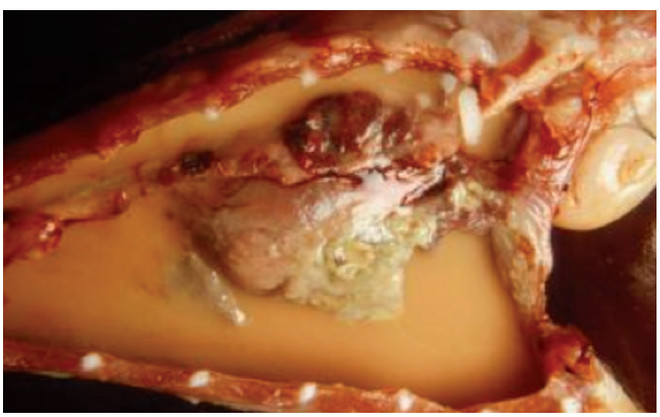
USAGE RECOMMENDATION

- Thoracic instillation of antimicrobials via thoracostomy tubes is not recommended because data to support efficacy is lacking.
- For ongoing treatment, once clinical improvement is seen and the patient is eating well, oral antibiotics may be substituted for IV agents.
- Duration of therapy: Repeat thoracic radiographs 10-14 days after being discharged from hospital on oral antimicrobials. Extend therapy as required.
- Previously, antimicrobial therapy for 3-6 weeks was recommended. Evidence to recommend a minimum period of administration is lacking and serial monitoring is recommended.

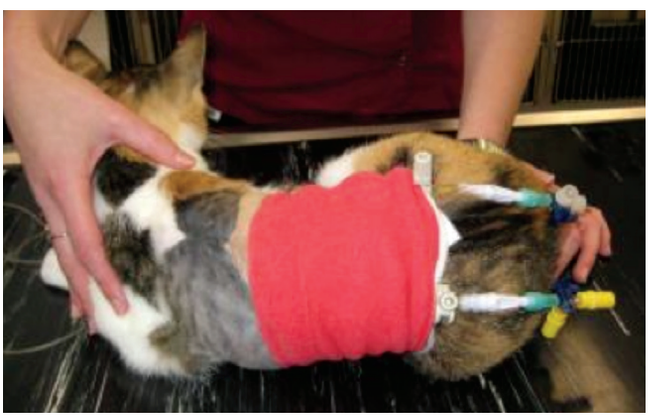
CONDITION: PYOTHORAX

AIDAP TOP TIPS

1. Pyothorax in cats is commonly a complication of upper respiratory tract infection with micro-aspiration of oropharyngeal flora and spread of aspirated bacteria from the lung to the pleural space.
2. An ultrasound scan for pleural effusion is a cost-effective test in cats with dyspnoea and fever of unknown origin, especially if respiration is restrictive (rapid and shallow).
3. Of all the causes of pleural effusion in the cat, bacterial pyothorax has the best long-term prognosis.



Cat with pyothorax at necropsy.
Photos courtesy of Dr Vanessa Barrs.



Placement of bilateral chest tubes is recommended where effusions are bilateral.

Key references:

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CONDITION: ACUTE LOWER UTI SPORADIC CYSTITIS (FIRST OCCURRENCE)

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

The most common cause of lower urinary tract (LUT) signs in cats is feline idiopathic cystitis, which does not involve bacteria or fungi.

Bacterial urinary tract infections are much less frequent in cats than in dogs. The International Society for Companion Animal Infectious Diseases (ISCAID) guidelines for the diagnosis and management of bacterial urinary tract infections in dogs and cats defines 'sporadic bacterial cystitis' as a sporadic bacterial infection of the urinary bladder with compatible lower urinary tract signs, and may include animals with known comorbidities that have not had more than three episodes of UTI in the last 12 months. The rationale for this is that there is no evidence that the treatment of UTI in cats with comorbidities is more complex to manage.

The clinical presentation for LUT disease in cats is common, including dysuria, haematuria, stranguria, pollakiuria and periuria (inappropriate urination). The major differential diagnosis for cats presenting with LUT signs (up to 55% of cases) is feline idiopathic cystitis (FIC, previously known as interstitial cystitis, feline lower urinary tract disease, or feline urological syndrome). The prevalence of bacterial UTI in cats ranges from 2% to 19% depending on study design, with the prevalence increasing with age-related comorbidities such as renal disease and endocrinopathies. Urethral obstruction in male cats is more frequent in cats with FIC than with UTI, while less common causes include urolithiasis, anxiety disorder (where inappropriate urination is the presenting sign) and neoplasia.

In recent published and unpublished Australian studies, the most common cause of sporadic bacterial cystitis in cats is *E. coli* (up to 70%), followed by *Enterococcus faecalis, Staphylococcus* species (mostly *S. felis* but some *S. pseudintermedius)* and *Proteus*, which collectively represent 95% of isolates cultured. Less frequently isolated bacteria include *Enterobacter* spp., *Klebsiella* spp., and *Pseudomonas aeruginosa*. Most *E. coli* (>80%), *E. faecalis* (>95%) and *S. felis* (100%) or *S. pseudintermedius* (>90%) remain susceptible to amoxicillin.

Amoxicillin has been suggested as an appropriate first-choice empiric antimicrobial for sporadic bacterial cystitis in cats by ISCAID. Trimethoprim-sulphonamide has also been recommended for first-line therapy of sporadic bacterial cystitis in cats however many cats do not tolerate this well. For cats that cannot be orally medicated with amoxicillin, empirical treatment with doxycycline may be an alternative with most Australian isolates susceptible to this agent, which reaches appropriate concentrations in urine. Some Gram-negative pathogens (for example *Proteus* spp.) are intrinsically resistant to doxycycline.

CONDITION: ACUTE LOWER UTI
SPORADIC CYSTITIS (FIRST OCCURRENCE)

KEY ISSUES

01 The majority of acute LUT cases in cats are sterile and due to feline idiopathic cystitis.

02 UTIs are uncommon in young cats but the prevalence increases with age due to the presence of co-morbidities such as chronic kidney disease and endocrinopathies.

03 *E. coli* is the most common Gram-negative bacterium while *Enterococcus* spp., and *S. felis* are the most common Gram-positive bacteria to cause sporadic bacterial UTIs in Australian cats.

04 Considering both antimicrobial susceptibility and compliance issues, amoxicillin (or if not available amoxicillin-clavulanate), or doxycycline are appropriate empiric choices for treating sporadic bacterial cystitis in cats.

05 Collection of urine by cystocentesis for full urinalysis including sediment examination can be difficult on an out-patient basis in some cases due to LUT signs resulting in an empty bladder at first presentation. Examination of the urine via wet preparation only requires a few drops and for the purposes of determining FIC vs UTI is very helpful. Consider admitting the cat, if required, to obtain a urine sample by cystocentesis.

06 For cats with urethral obstruction and indwelling urinary tract catheters, prophylactic antimicrobials should never be given during the period of catheterisation. Culture of urine from urine-collection bags or urinary catheter tips after removal is contraindicated as they do not represent a diagnostic sample indicative of clinical infection. A biofilm can form on these structures that has no correlation with clinical infection. Post-catheter UTI should be diagnosed on the presence of clinical signs and cytological evidence of inflammation and infection using a urine sample collected by cystocentesis.

CONDITION: ACUTE LOWER UTI
SPORADIC CYSTITIS (FIRST OCCURRENCE)

TESTS FOR DIAGNOSIS

1. A full urinalysis is recommended for all cats presenting with LUT signs, using a urine sample collected by cystocentesis. In cases where the bladder is empty, examination of a few drops under the microscope as a 'wet preparation' is valuable in differentiating FIC from UTI.

2. Diagnosis of sporadic bacterial UTI requires LUT signs with supporting evidence of UTI (epithelial cells, RBC, WBC, and bacteria) on urinalysis including examination of the urine sediment (wet preparation +/- Diff-Quik® +/- Gram stain) and/or examination by a urine analysis machine. Urine collected for urinalysis should also be submitted for C&S testing.

3. Free-catch urine samples are inferior and should generally be avoided for culture but can be of some use for urine analysis.

4. Where subclinical bacteriuria is identified (positive urine culture or cytological evidence of bacteruria in the absence of clinical signs or pyuria) antimicrobial treatment is generally not recommended.

CONDITION: ACUTE LOWER UTI
SPORADIC CYSTITIS (FIRST OCCURRENCE)

TREATMENT

1. Empiric therapy is appropriate while waiting for urine culture results. Depending on the results of urine sediment exam this should include muscle relaxants such as diazepam or analgesics/anti-inflammatories such as meloxicam. If bacteria have been visualised on urine sediment exam, empiric use of antibiotics can commence.

2. Recommended first-line choice for empiric therapy in cats with supportive evidence (urine sediment and clinical signs) of bacterial infection and inflammation include amoxicillin (or if not available amoxycillin-clavulanate). Doxycycline can be considered as an alternative for non-compliant cats or cats that vomit on amoxicillin.

3. The recommended treatment duration is 3-5 days for sporadic bacterial cystitis.

RECOMMENDED ANTIBIOTICS

First line:

Amoxicillin (11-15 mg/kg q8-12h PO)

Doxycycline (5 mg/kg q12h PO†)

Second line:

See section on recurrent bacterial cystitis. Requires C&S testing.

†Ensure doxycycline given with a small bolus of water, or a small piece of food or margarine to ensure rapid oesophageal passage.

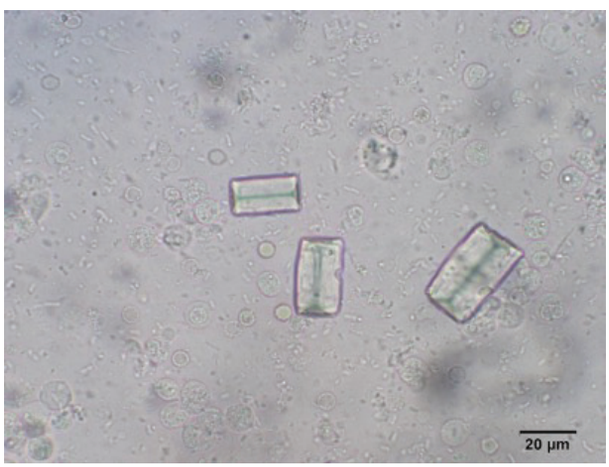
USAGE RECOMMENDATION

Recommended duration of therapy is 3-5 days.

CONDITION: ACUTE LOWER UTI
SPORADIC CYSTITIS (FIRST OCCURRENCE)

AIDAP TOP TIPS

1. Feline idiopathic cystitis is the most common cause of lower urinary tract signs in cats.
2. Consider bacterial UTI in cats with underlying risk factors such as age, renal impairment, or metabolic disease. Perform a full diagnostic workup including C&S testing.



Examination of a few drops of urine sediment is a valuable tool in cats for differentiating FIC from bacterial UTI. The microscope needs to have the lights turned down and the condenser lowered, allowing the differentiation between red blood cells, white blood cells, bacteria, and crystals. While a sample via cystocentesis is ideal, a voided sample is adequate for this initial examination, but not suitable for culture. Staining of the sediment is not required in most cases. This technique is available via video AMR Vet Collective | Continuing Education.

Photograph courtesy of Jacqui Norris.

Key references:

1. Weese, et al. The Veterinary Journal. 2019; 247: 8–25.
2. Scarborough R, Bailey K, Galgut B, et al. Antibiotics. 2020; 9: 924. doi:10.3390/antibiotics9120924.
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CONDITION: RECURRENT BACTERIAL CYSTITIS

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Recurrent bacterial cystitis is defined as three or more episodes in the preceding 12 months or two episodes in the preceding six months.

It may be the result of persistent infection or reinfection. It may be associated with an unresolved or unidentified co-morbidity such as kidney disease, endocrinopathies such as hyperthyroidism or diabetes mellitus, urolithiasis, congenital or acquired conformational abnormalities, bladder neoplasia or other disorders of growth, or urinary retention. Therefore, a diagnostic plan that identifies likely potential co-morbidities is required for successful resolution. Equally it may be the result of inadequate or inappropriate treatment of an acute bacterial cystitis or the presence of a resistant organism. While there have been instances of multidrug-resistant (MDR) *E. coli, Enterococcus faecalis, Corynebacterium urealyticum, Staphylococcus* spp. and *Enterobacter* spp. isolated from feline urine in Australian veterinary diagnostic laboratories, they remain infrequent even in referral medicine settings.

CONDITION: RECURRENT BACTERIAL CYSTITIS

TESTS FOR DIAGNOSIS

1. A broader diagnostic plan may include full serum biochemistry, haematology, endocrine testing, diagnostic imaging, neurological exam, etc. as appropriate to the patient's clinical signs.
2. A full urinalysis is recommended for all cats presenting with recurrent cystitis, using a urine sample collected by cystocentesis where possible.
3. Diagnosis of recurrent bacterial cystitis requires an appropriate history of repeated UTI together with LUT signs with supporting evidence of UTI (epithelial cells, RBC, WBC, and bacteria) on sediment examination by wet preparation (see instructions on 'Wet Prep"), or examination by a urine analysis machine, and/or stained urine sediment (Gram or Diff-Quik®).
4. Urine C&S testing is very important for the correct identification of the causative bacterium and the appropriate choice of antimicrobial agent.

KEY ISSUES

01 Look for markers of underlying disease or bladder dysfunction/ abnormalities (for example, urolithiasis, kidney disease, PU/PD, hyperthyroidism, diabetes mellitus, urine retention) so that complications predisposing to infection can be addressed, reducing the risk of recurrent cystitis.

02 The presence of a co-morbidity does not categorise the infection as recurrent but failure.

TREATMENT

1. Empiric antimicrobial therapy is reasonable pending urine C&S results although evidence in human medicine suggests the use of anti-inflammatory/pain relief achieves similar results in the first 24-48 hours.
2. The recommended first-line choices for empiric therapy for recurrent bacterial cystitis are amoxicillin (or if not available, amoxicillin-clavulanate) due to high susceptibility of most causative bacteria in Australian cats.
3. Urine C&S testing from an aseptically collected sample is important in recurrent bacterial cystitis as is the identification of complicating co-morbidities.
4. Treatment duration may be short (3-5 days) or longer (7-14 days) depending on the results of the broader diagnostic plan.

CONDITION: RECURRENT BACTERIAL CYSTITIS

RECOMMENDED ANTIBIOTICS

First line:

(until C&S available)
Amoxicillin (11–15 mg/kg q8-12h PO)

Second line:

Based on C&S testing

USAGE RECOMMENDATION

Short duration courses (3-5 days) may be considered for reinfection.

Longer courses (7-14 days) may be considered when persistent infections are likely.

Ensure duration, dose and choice of antimicrobial is reviewed following C&S and further diagnostic tests to ensure good penetration of an appropriate antimicrobial.

AIDAP TOP TIPS

Identification of unresolved co-morbidity and/or antimicrobial-resistant bacteria is critical for the successful resolution of recurrent bacterial cystitis.

Key references:

1. Weese, et al. The Veterinary Journal. 2019; 247: 8–25.
2. Scarborough R, Bailey K, Galgut B, et al. Antibiotics. 2020; 9, 924; doi:10.3390/antibiotics9120924.
3. Dorsch R, Teichmann-Knorrn S, Sjetne Lund H. Journal of Feline Medicine and Surgery. 2019; 21: 1023–1038.

CONDITION: PYELONEPHRITIS

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

The bacteria involved in pyelonephritis are the same as for bacterial cystitis, with *Escherichia coli* being the most frequent bacterium involved.

Pyelonephritis is defined as inflammation of the renal pelvis and parenchyma, most commonly the result of ascending bacterial infection or less frequently bacteraemia. Ascending infection can occur with or without overt clinical signs of lower urinary tract disease. Medullary infection extends from the renal pelvis due to poor blood supply and other factors.

Pyelonephritis can be acute or chronic and there is notably overlap in clinical signs. In acute pyelonephritis, fever is the most consistent clinical sign. Other frequent clinicopathological signs include pain on renal/abdominal palpation, variation in kidney size, anorexia, lethargy, vomiting, diarrhoea, uraemia, azotaemia, leucocytosis, pyuria, cylinduria, and/or sepsis, but clinicopathologic signs can be much less overt and require a high index of suspicion. Cats may range from polyuric to oliguric or anuric.

Chronic pyelonephritis is likely underdiagnosed as it has greater variation in clinical signs but may lead to changes in kidney shape due to chronic inflammation and fibrosis. The bacteria involved in pyelonephritis are the same as for bacterial cystitis, with *Escherichia coli* being the most frequent bacterium involved.

CONDITION: PYELONEPHRITIS

TESTS FOR DIAGNOSIS

1. A diagnostic plan should include full serum biochemistry, haematology, endocrine testing, diagnostic imaging, as appropriate to the patient's clinical signs.
2. A full urinalysis is recommended for all cats presenting with pyelonephritis, using a urine sample collected by cystocentesis or if possible, pyelocentesis.
3. Urine C&S testing is very important to guide the appropriate choice of antimicrobial agent.

KEY ISSUES

01 Look for markers of underlying disease such as urolithiasis, kidney disease, diabetes mellitus, as possible predisposing factors to infection, and address the impact of infection rapidly.

02 Definitive diagnosis of both acute and chronic pyelonephritis can be challenging, with clinical signs varying from overt to covert, requiring thorough diagnostic investigation.

TREATMENT

1. Empirical treatment should commence immediately pending C&S testing given the serious potential consequences of delayed treatment.
2. The recommended first-line choice for empiric therapy for pyelonephritis in cats is marbofloxacin, due to its lipophilic qualities, good penetration into renal interstitial tissue, absence of the side effects seen with enrofloxacin, and susceptibility of the major pathogens.
3. The identification of complicating co-morbidities is very important.
4. Treatment duration should be 10-14 days depending on the results of the broader diagnostic plan.
5. Re-examination and evaluation of the patient, including physical examination, serum biochemical assessment of renal analytes, and aerobic urine culture is highly recommended one week after the end of the antimicrobial course.

CONDITION: PYELONEPHRITIS

RECOMMENDED ANTIBIOTICS

First line:

(until C&S available)
Marbofloxacin (5.5 mg/kg PO q24h)

Second line:

Based on C&S testing

USAGE RECOMMENDATION

Treatment duration should be 10-14 days depending on the results of the broader diagnostic plan.

Re-examination and evaluation of the patient including physical examination, serum biochemical assessment of renal analytes, and aerobic urine culture is highly recommended one week after the end of the antimicrobial course.

Ensure duration, dose and choice of antimicrobial is reviewed following C&S testing and further diagnostic tests.

AIDAP TOP TIPS

Pyelonephritis has a wide range of clinical signs with fever being the most consistent. A high index of suspicion is required to correctly diagnose pyelonephritis in more subtle presentations.

Key references:

1. Weese, et al. The Veterinary Journal. 2019; 247: 8–25.
2. Dorsch R, Teichmann-Knorrn S, Sjetne Lund H. Journal of Feline Medicine and Surgery. 2019; 21: 1023–1038.

CONDITION: ACUTE FEBRILE ILLNESS

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Many cats with acute febrile illness have an underlying infectious cause.

Common causes may be bacterial such as cat fight cellulitis, or viral such as feline infectious peritonitis, FHV-1, or FCV.

In a retrospective study of 106 cats presenting for fever the most common cause of pyrexia was feline infectious peritonitis (20.8%) and the largest disease category was infectious (38.7%). Inflammatory conditions were found in (17.9%), neoplasia (12.3%), miscellaneous causes (10.4%) and immune-mediated disease in (5.7%). No diagnosis was reached in (15.0%) cats.

Thus, it is uncommon to have immune-mediated disease in cats when compared with dogs, although sterile polyarthropathies can mimic infection-related fever.

Pasteurella, *Staphylococcus pseudintermedius*, obligate anaerobic species and *Streptococcus* spp. can be involved commonly in feline infectious disease.

CONDITION: ACUTE FEBRILE ILLNESS

TESTS FOR DIAGNOSIS

01 History and thorough clinical examination remain the cornerstone. Was the cat boarded recently? Are mouth ulcers present? Is there oculo-nasal discharge. Does the cat have outside access? Was the cat recently in a fight?

02 Systematic palpation for regions of hyperaesthesia.

03 Consider thoracic radiographs and ultrasound examination of the thoracic and abdomen for fluid.

04 Consider echocardiography to determine the likelihood of bacterial endocarditis.

05 Consider blood culture, haematology, and serum biochemistry if there is an index of suspicion for sepsis, defined as bacteraemia with systemic inflammatory response syndrome (SIRS).

06 Where joint effusions are detected, imaging and analysis of the joint fluid (cytology and culture) are recommended.

KEY ISSUES

01 A key element of any acute febrile disease is making a diagnosis as early as possible. A thorough physical examination is required, looking especially for cat bite cellulitis lesions.

02 A high creatine kinase activity in a serum biochemistry profile in a cat with acute fever can suggest myonecrosis secondary to undetected cat bite wounds or other trauma.

03 Acute upper respiratory viral disease is a common cause of fever in the cat.

04 Early pyothorax can present with fever and little else, as there is insufficient fluid to impair ventilation. Thoracic radiographs or thoracic sonography may reveal a small amount of septic pleural exudate in this case.

05 Palpate joints carefully for thickening joint capsules, effusion, and pain.

CONDITION: ACUTE FEBRILE ILLNESS

TREATMENT

- The main differentials are catfight wounds and viral infections.
- Investigating and reaching a diagnosis is very important, but empirical antibiotics may be used if there is a strong suspicion of a bacterial infection, for example cat fight cellulitis.

RECOMMENDED ANTIBIOTICS

First line:

Amoxicillin-clavulanate (20 mg/kg q12h PO)
Doxycycline monohydrate (5 mg/kg q12h PO†)

†Ensure doxycycline given with a small bolus of water, or a small dab of margarine.

USAGE RECOMMENDATION

An initial injection of amoxicillin-clavulanate subcutaneously followed by administration of oral therapy as required for 3 days. Modifications of this pending result of further diagnostic testing.

Doxycycline† may be useful in situations where paste or a small pill will improve compliance.

CONDITION: ACUTE FEBRILE ILLNESS

AIDAP TOP TIPS

1. Although there is a strong compassionate desire to use an NSAID to reduce the fever and make the patient feel more comfortable, this is only recommended when the diagnosis is known – for example detection of cat puncture wounds, but no abscess. In other situations – it is more prudent to use antibiotics alone – and let the response to therapy (resolution of fever, resumption of eating) confirm that there is likely an underlying bacterial aetiology.

2. If there is a favourable response to antimicrobial therapy – consider a longer course, rather than a shorter course, in case there is undetected significant disease such as pneumonia or purulent pleurisy.

Key references:

1. Spencer, S.E. J Feline Med Surg. 2017; 11: 1123-1130.

CONDITION: GASTROINTESTINAL INFECTIONS

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Gastrointestinal disease in cats is a frequent problem. In most instances the presenting clinical signs are either vomiting, diarrhoea or both.

Infectious agents are an uncommon cause of vomiting that is not accompanied by diarrhoea. The possible exception being gastric *Helicobacter* spp. infection although their normal habitation of the gastric mucosa is a confounding issue.

Dietary causes, gastrointestinal infections, inflammation, neoplasia, or extra-gastrointestinal disease may cause vomiting/diarrhoea and infectious diseases are only one group of possible aetiologies. Additionally, the asymptomatic carriage of infectious agents may complicate diagnosis.

Several potential pathogens have been isolated from cats with diarrhoea and these include helminths, bacterial, viral, protozoal, and other parasitic organisms. Some of these pathogens are also present in cats that are non-diarrhoeic. Similar isolation rates for putative bacterial entero-pathogens in animals with and without diarrhoea are reported, leading to the overuse of antibiotics for diarrhoea in small animals. Most bacterial entero-pathogens are associated with self-limiting diarrhoea, and the inappropriate administration of antimicrobials could be more harmful than beneficial by removing the normal flora. Co-infections are possible and can have clinical consequences, for example, *Cryptosporidium* spp. has been associated with an increased severity of diarrhoea in *Tritrichomonas foetus* positive cats. A higher incidence of infectious disease is seen in young cats, purebred cats, and cats from multi-cat environments (particularly breeding establishments).

CONDITION: GASTROINTESTINAL INFECTIONS

The following infectious agents have been implicated as causing gastrointestinal disease in the cat:

Viral
Feline parvovirus (panleukopenia), feline enteric coronavirus.

Bacterial
Salmonella spp., *Campylobacter jejuni*, *Clostridium perfringens*, *Clostridium difficile*, some *E. coli* (enteropathogenic, enteroinvasive, enterotoxigenic, enterohaemorrhagic biotypes), *Helicobacter felis*, and *Helicobacter heilmannii*.

Protozoal
Giardia spp., *Cryptosporidium*, *Tritrichomonas foetus*.

Parasitic helminths
Toxocara cati, *Ancylostoma tubaeforme/braziliense*.

SPECIFIC INDICATIONS FOR THE USE OF ANTIMICROBIALS IN DIARRHOEA:

1. Specific identification of a bacterial agent listed below via C&S or PCR.
2. Diarrhoea characterised as severe with likely compromise of the epithelium to the extent that bacterial translocation is likely. For example, in feline parvovirus, or suspected severe Salmonellosis.
3. Diarrhoea with marked neutropenia (less than 1 x 10^9/L).
4. Acute haemorrhagic diarrhoea with dehydration.

CONDITION: GASTROINTESTINAL INFECTIONS

TESTS FOR DIAGNOSIS

1. Faeces placed in a sterile container for specific culture of a pathogen on specified media. C&S testing is often required for correct choice of antimicrobial.
2. PCR testing panels for multiple parasites.
3. Antigen testing of faeces for feline parvovirus (canine parvovirus kits are often used).
4. Wet slide preparation of faecal material and microscopic evaluation for *Giardia* and *Tritrichomonas foetus* (TTF) trophozoites. Culture pouch for TTF.
5. Faecal flotation for identification of helminths, *Giardia* cysts, *Cryptosporidium* and *Coccidia*.
6. Enteric biopsy may identify *Cryptosporidium* in some infrequent cases of chronic diarrhoea.

KEY ISSUES

01 **Most cases of acute diarrhoea in cats are self-limiting and do not require antimicrobial therapy.**

02 If antibiotic therapy is to be used, diagnostic samples should be collected prior to starting therapy.

03 Diarrhoea with marked neutropenia or potential for translocation of bacteria across severely damaged intestinal epithelium may require prophylaxis for bacterial sepsis, but studies indicating improved patient survival have a low evidence base.

TREATMENT

Where dehydration is evident with diarrhoea, hospitalisation for treatment with intravenous fluids is strongly advised.

CONDITION: GASTROINTESTINAL INFECTIONS

RECOMMENDED ANTIBIOTICS

Acute non-life-threatening diarrhoea with no evidence of patient dehydration or haematochezia:

No antibiotic is required.

Acute diarrhoea with marked neutropenia or strong evidence of sepsis:

First line:
Amoxicillin (20 mg/kg q8h IV) and
Marbofloxacin (5.5 mg/kg q24h PO) or
Pradofloxacin (7.5 mg/kg q24h PO)

Second line:
Amoxicillin (20 mg/kg q8h IV) and
Gentamicin (5-8 mg/kg q24h SC*)

Specific infectious agents if conclusively identified:

Salmonella:
Based on C&S but the above first and/or second choice combinations should be effective against most strains.

Campylobacter:
Most cases are mild and self-limiting and rely on normal microbiota to assist in recovery, so treatment is only used in severe cases or when issues of zoonosis in immune-compromised households arise.

First line: (based on C&S)
Marbofloxacin (5.5 mg/kg PO q24h)
Pradofloxacin (7.5 mg/kg PO q24h)

Second line:
Erythromycin (5-10 mg/kg q8h)

Third line:
Azithromycin (5-10 mg/kg q12h)
Clarithromycin (7.5 mg/kg q12h)

***Should be accompanied by intravenous fluid therapy**

USAGE RECOMMENDATION

There is no evidence-base regarding treatment duration, but the panel recommends at least 4 days, and ideally 7 days.

There is no conclusive evidence for an advantage of one antibiotic over another.

Veterinary fluroquinolones can be used as Australian strains rarely exhibit resistance.
Treatment duration 3-5 days.

Azithromycin and Clarithromycin are not registered for use in animals. They should not be used off-label except in exceptional circumstances for individual animals.

CONDITION: GASTROINTESTINAL INFECTIONS

RECOMMENDED ANTIBIOTICS

Helicobacter:

If the clinician is convinced that *Helicobacter* is the cause of chronic vomiting. This would require biopsies of the gastric mucosa that show fibrosis, lymphoid inflammation that is associated with large numbers of *Helicobacter*.

First line:
Amoxicillin-clavulanate (20 mg/kg q12h PO) and Metronidazole (10 mg/kg q12h PO)

Second line:
Clarithromycin (7.5 mg/kg q12h PO) and Metronidazole (15 mg/kg q12h PO)

Tritrichomonas:

First line:
Ronidazole (30 mg/kg q24h PO)

Giardia:

First line:
Fenbendazole (50 mg/kg q24h PO)

Second line:
Metronidazole (25 mg/kg q12h PO)

Cryptosporidium:

Most cases are mild and self-limiting.

The following drugs have been used with some success in cases where animals have persistent diarrhoea with oocyst shedding:

Azithromycin (7-15 mg/kg q24h PO for 5-7 days)

Nitazoxanide (100 mg q12h PO for 5 days in animals 24 to 47 months old and 200 mg q12h PO for 5 days in animals 4 to 11 years old)

USAGE RECOMMENDATION

For Helicobacter:
Add acid inhibition with omeprazole 10 mg per cat q24h PO for 14 days.

Ronidazole may need to be compounded. 2 weeks therapy is recommended. Neurotoxicity has been reported at this and higher doses.

Giardia treatment duration is 5 days.

Metronidazole: Higher doses (50 mg/kg) carry an increased risk of side effects, including weakness, ataxia, disorientation, and seizures and are best avoided. In multi-cat households, consideration of treatment or all animals should be made. Great care with cleaning of faeces from the environment is part of eradication programs.

CONDITION: GASTROINTESTINAL INFECTIONS

AIDAP TOP TIPS

1. Always take samples for diagnostic testing prior to treating with antimicrobials.
2. Antibiotics should be reserved for animals definitively diagnosed with a specific bacterial pathogen and those that are at risk of life-threatening sepsis.
3. Liquid antibiotic medications are often easier to administer for clients.

Key references:

1. Paris JK, Wills S, Balzer H. et al. BMC Vet Res. 2014; 10: 13. https://doi.org/10.1186/1746-6148-10-13.
2. Marks SL, Rankin SC, Byrne BA, Weese JS. J Vet Intern Med. 2011; 25(6): 1195-208.
3. Giardia treatment in cats. http://www.abcdcatsvets.org/giardiasis/.
4. Tritrichomonas treatment in cats. http://www.abcdcatsvets.org/tritrichomoniasis/.
5. ASTAG - Importance ratings and Summary of Antibacterial Uses in Human and Animal Health in Australia. 2018. ISBN: 978-1-76007-369-5.

CONDITION: USE OF ANTIMICROBIALS IN SURGERY

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

In surgical practice, antimicrobials may be used for treatment of a pre-existing infection, or for prevention of an infection resulting from the procedure, known as antimicrobial prophylaxis.

For pre-existing infections, C&S should be performed prior to the administration of any antimicrobial.

In the case of a potential break in sterile technique, the surgeon must weigh the risk of an infection against the risk of antimicrobial resistance development.

Regardless of the timing of infection, surgical site infections (SSI) represent significant cost, morbidity and even mortality in veterinary medicine. The rate of SSI depends upon the type of surgery but may be reduced by following the principles of surgical asepsis and meticulous use of aseptic techniques.

When antimicrobials are used appropriately in surgical prophylaxis, the antimicrobial chosen is often empirical, however the minimum inhibitory concentration (MIC) of the drug for the most likely contaminant/s should be reached at the surgery site prior to the first incision and should remain at or above the MIC for the duration of surgery.

Surgery is commonly classified as:

Clean - no infection present and not likely to become contaminated.

Clean-contaminated - surgery of the respiratory, gastrointestinal, genitourinary tracts without likely contamination.

Contaminated/dirty - infection already present at surgical site or contamination likely during the procedure.

The reported rates for SSI are approximately 5% for clean surgeries, 12% for clean-contaminated surgeries and 10% for contaminated/dirty surgeries.

CONDITION: USE OF ANTIMICROBIALS IN SURGERY

Clean surgical procedures (for example, routine desexing or neurological procedures of short duration) do not require antimicrobial prophylaxis in animals without risk factors; antisepsis and aseptic technique reduces the risk of infection to negligible levels.

Appropriately administered antimicrobials can reduce the risk of SSI by 6-7 fold. Improper selection of antimicrobials or incorrect administration results in increased SSI and the development of antimicrobial-resistant organisms.

Clean surgical procedures (for example, routine desexing or neurological procedures of short duration) do not require antimicrobial prophylaxis in animals without risk factors; antisepsis and aseptic technique reduces the risk of infection to negligible levels.

Antimicrobial prophylaxis during the procedure and for up to 24 hours following the surgery is indicated in animals undergoing the following:

Clean surgical procedures with risk factors such as longer duration surgery time or American Society of Anesthesiologists (ASA) patient classifications 3, 4, or 5.

Clean-contaminated surgeries with implant placement.

Contaminated/dirty surgeries.

Generally, in cases with the following: increased numbers of people present in the operating room; prolonged anaesthesia time; presence of a drain; or administration of propofol.

This reduces SSI to acceptable levels of 4.2-6.3%. Even in surgeries where SSI rates have been reported as high as 21%, no definitive evidence has been found to support the use of antimicrobials beyond 24 hours postoperatively. Risk factors indicating postoperative use of antimicrobials beyond 24 hours postoperatively include a dirty surgical site, increasing body weight (dogs >50 kg), duration of postoperative intensive care unit stay, extremes of age, morbid obesity, removal of hair greater than 4 hours preoperatively, hypothermia, and hypotension.

Risk factors for development of SSI with multiple drug resistance include the type of bacteria present (Enterobacteriales, *Salmonella*, methicillin-resistant *Staphylococcus pseudintermedius*), feeding a homemade diet or feeding raw food to the animal.

CONDITION: USE OF ANTIMICROBIALS IN SURGERY

Even in surgeries where SSI rates have been reported as high as 21%, no definitive evidence has been found to support the use of antimicrobials beyond 24 hours postoperatively.

When empirically choosing an antimicrobial, a narrow spectrum of activity is recommended to preserve the animal's normal microbiota and reduce the development of drug resistance.

The drug levels at the surgical site must be maintained at greater than MIC for the expected pathogens throughout the surgery, therefore repeat administration at intervals of 1-2 times the elimination half-life of the drug is recommended. The antimicrobial must be present in the tissue prior to commencement of surgery, and therefore should be administered at least 30 minutes prior to surgical incision, but to reduce development of MDR and SSI, no earlier than 60 minutes prior to surgical incision. This means there is a 30-minute window of appropriate timing for antimicrobial administration.

SCENARIO BASED DECISIONS ON ANTIBIOTIC USE:

Antimicrobial prophylaxis is not a substitute for aseptic preparation of the patient, staff, facilities, or equipment.

In animals at risk of carrying MDR organisms such as those living in a household with humans carrying resistant organisms, animals fed home cooked or raw diets, animals with distant infections or recent antibiotic exposure, prior culture of nasal or aural tissues is warranted since such animals are at increased risk of SSI following surgery.

Selection of antimicrobials to be used prophylactically should be based upon the procedure, potential pathogens, and risk factors of the animal and surgical environment, such as the number of people in the operating room.

CONDITION: USE OF ANTIMICROBIALS IN SURGERY

TIMING OF ANTIMICROBIAL PROPHYLAXIS

The following recommendations are the standard of care in humans (National Surgical Infection Prevention Project):

1. Parenteral antimicrobials administered 30-60 minutes prior to surgical incision.
2. Repeat administration every 1–2 elimination half-lives during the procedure.
3. Discontinuation of prophylactic antimicrobials is recommended less than 24 hours following conclusion of surgery unless patient or surgical risk factors determine postoperative use is indicated.

KEY ISSUES

Numerous studies have been conducted on the use of antibiotics in surgery using animal models for human medical purposes. Studies specific to dogs and cats in veterinary practice recommend the following:

01 The success of antimicrobial prophylaxis at reducing surgical site infections is affected by the surgical site, likely pathogens present, pathogens to most likely cause a surgical site infection, risk factors present in the animal, and adherence to strict aseptic preparation and technique.

02 Antimicrobials administered following surgery are ineffective at reducing the risk of SSI and may increase the risk of multidrug-resistant (MDR) infections.

03 The risk of SSI doubles approximately every 70 minutes of surgery time and increases by 30% for each additional hour of anaesthesia time. Minimally invasive surgical procedures reduce the risk of SSI.

04 The likelihood of SSI increases with the number of people present in the operating theatre, increased hospitalisation postoperatively, and with increasing contamination of the surgical site.

CONDITION: USE OF ANTIMICROBIALS IN SURGERY

ANTIBIOTICS RECOMMENDED

Clean surgical procedures:

No antimicrobial is required.

Surgical procedures where contamination is associated with severe consequences, but infection is not clearly established:

Elective Orthopaedic surgery:
Cefazolin (30 mg/kg IV given every 90 minutes until wound closure)

Gastric, urogenital, and small intestinal surgery:
Cefazolin (30 mg/kg IV)

Large intestinal surgery:
Cefazolin (30 mg/kg IV)
Cefoxitin (30 mg/kg IV)

Pyometra (contained, no leakage):
Cefazolin (30 mg/kg IV)
Amoxicillin (20 mg/kg IV)

Abdominal surgery:
Cefazolin (30 mg/kg IV)
Amoxicillin (20 mg/kg IV)

USAGE RECOMMENDATIONS

Clean surgical procedures for which antimicrobial use is unnecessary:

1. Ovariohysterectomy.
2. Castration.
3. Removal of skin masses (lipomas for example).
4. Dental procedures (scaling and polishing) with minimal periodontal disease or risk of bacteraemia (see Dental prophylaxis chapter).
5. Clean surgical procedures without implant placement and procedures <70 minutes duration such as some neurological surgeries.

In all cases antibiotics are administered 30-60 minutes prior to surgery and repeated at 1-2 elimination half-lives during the procedure.

For elective orthopaedic surgery (total hip replacement, cruciate ligament surgery TPLO, TTA, others that involve cutting bone, or use of surgical implants), the anticipated pathogen is *Staphylococcus* spp. First generation cephalosporins can be continued parenterally every 8 hours for up to 24 hours postoperatively.

For upper gastrointestinal surgery, anticipated pathogens are Gram-positive cocci and Gram-negative bacilli.

For lower gastrointestinal surgery, anticipated pathogens are Gram-negative bacilli, enterococci, anaerobes.

If there is a ruptured gastrointestinal tract prior to surgery, use an extended Gram-negative antibiotic treatment in addition to ampicillin, such as a fluoroquinolone or gentamicin.

For pyometra (contained at surgery), anticipated pathogens are *Escherichia coli, Streptococcus* spp. and anaerobes.

For abdominal surgery (splenectomy, liver lobectomy) without contamination, anticipated pathogen is *Staphylococcus* spp.

CONDITION: USE OF ANTIMICROBIALS IN SURGERY

RECOMMENDED ANTIBIOTICS

Contaminated surgery where infection is already apparent or likely to be present:

Abscesses, hepatobiliary surgery, removal of organs, marsupialisation and drain insertions.

INITIAL THERAPY:

First line:

Cefazolin (30 mg/kg IV) or
Amoxicillin (20 mg/kg IV) and
Gentamicin (6 mg/kg SC)

Second line:

Cefoxitin (30 mg/kg IV)

ONGOING THERAPY:

Based on C&S. Narrow spectrum if possible.

USAGE RECOMMENDATIONS

Gentamicin is added and given prior to surgery if enteric or Gram-negative bacteria possibly involved.

Cefoxitin is suggested if anaerobic infection likely and possibly resistant to amoxicillin or cefazolin. For example, in hepatobiliary, urogenital, or lower GI surgery.

For head and neck surgery, clindamycin or cefazolin are appropriate choices, and anticipated pathogens are *Staphylococcus* spp., *Streptococcus* spp., anaerobes.

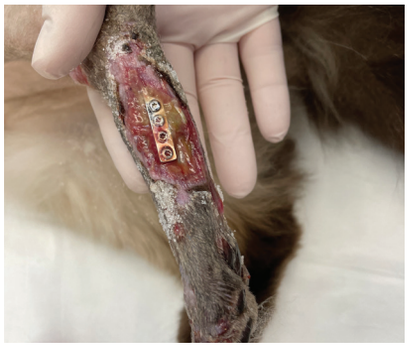
For abscesses such as cat fight abscess and anal sac abscess where drained refer to the chapters on soft tissue infections elsewhere in this guideline.

For hepatobiliary, anticipated pathogens are *Clostridium* spp., Gram-negative bacilli, anaerobes.

CONDITION: USE OF ANTIMICROBIALS IN SURGERY

AIDAP TOP TIPS

1. The risk of SSI must be weighed against the risk of MDR organism establishment when determining whether to administer prophylactic antimicrobials to surgical patients.
2. Knowledge of the surgery type, the most common organisms from SSI following the surgery, and the risk factors for SSI development will determine what type of antimicrobial prophylaxis should be administered.
3. Strict surgical personnel, facilities, and equipment aseptic preparation as well as adherence to aseptic technique in the surgery reduce the risk of SSI and the requirement for antimicrobial prophylaxis in many surgeries.
4. In surgical procedures where antimicrobial prophylaxis is used, timing of administration between 30 and 60 minutes prior to procedure commencement is vital.



Infection following tarsal arthrodesis.

Photo courtesy of Dr Wendy Baltzer.

Key references:

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2. https://vetantibiotics.fvas.unimelb.edu.au/surgery-guidelines/.
3. Hardefelt, L.Y. et.al. Aust Vet J. 2019 316-322.
4. Whittem, T.L et al. J Am Vet Med Assoc. 1999;215(2):212-6.
5. Ferrel, et al. Vet Surg. 2019;48(5):694-699.
6. Budsberg SC, Torres ST, Sandberg GS. Vet Surg. 2021. DOI: 10.1111/vsu.13603.
7. Verwilghen D and Singh A. Vet Clin Small Anim. 2015:45;243-276.

CONDITION: MYCOBACTERIA AND NOCARDIA AS CAUSES OF DEEP DRAINING SINUS TRACTS

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Mycobacteria cause two major types of disease affecting the skin and subcutis:

(i) Infections of the subcutaneous panniculus generally with rapidly growing mycobacteria and

(ii) Granulomatous or pyogranulomatous masses of the skin and subcutis (generally due to non-cultivable mycobacteria such as feline leprosy-like syndromes, leproid granulomas, and sometimes *Mycobacterium avium* complex infections). These infections will not be discussed further here but generally combination therapy is required using rifampicin, clarithromycin, pradofloxacin and clofazimine in various combinations.

The taxonomy of these organisms continues to evolve, and currently they are divided into complexes:

a. *M. smegmatis* complex (including *M. goodii*) – drugs of choice are doxycycline, pradofloxacin, (gentamicin, amikacin).

b. *M. fortuitum* complex– drugs of choice are clarithromycin, pradofloxacin, (gentamicin, amikacin).

c. *M. chelonae/abscessus* complex– drugs of choice are clarithromycin and linezolid, otherwise depends on susceptibility testing.

In general, they are all resistant to rifampicin, and all susceptible to clofazimine (which is hard to source but good for some refractory cases).

Draining sinus tracts should alert the practitioner to the presence of saprophytic pathogens such as mycobacteria, *Nocardia* spp. and fungi.

Involvement of the inguinal panniculus is suggestive of mycobacterial and nocardial disease.

Preliminary cytology stained with Diff-Quik® can be very helpful in cases where *Nocardia* and fungi are involved, whereas culture on routine media is far more expedient a way to diagnose mycobacterial infections caused by rapidly growing saprophytic mycobacteria.

CONDITION: MYCOBACTERIA AND NOCARDIA AS CAUSES OF DEEP DRAINING SINUS TRACTS

KEY ISSUES

a. Rapidly growing mycobacteria, *Nocardia* spp. and fungi can all give rise to deep draining tracts that discharge to the skin surface.

b. Rapidly growing mycobacteria (and to a lesser extent *Nocardia nova*) have a predilection for the fatty subcutaneous panniculus, especially in the inguinal region.

TREATMENT

These infections require months to years of antimicrobial therapy, and in some cases, surgery is required to debulk lesions to enable a clinical cure to be achieved. The timing of surgery is a value judgement and ideally it should occur after preliminary medical therapy, so the lesion is smaller and blood levels of effective drugs are present at the time of surgery and during the healing phase.

Topical therapy is not useful in the management of these infections as the disease process is situated in the subcutis and involves the skin secondarily.

TESTS FOR DIAGNOSIS

1. The cornerstone of therapy is obtaining a positive culture.
2. This is obtained by aspirating purulent exudate present in the subcutis through intact skin, after preparation of the skin with 70% ethanol (and allowing time for drying).
3. Primary isolation can be done in a veterinary laboratory, although it important to keep the plates for the 4-5 days it takes for the colonies to appear.
4. Positive cultures should be forwarded to a human mycobacteria reference laboratory for species identification and C&S testing.

REFERENCE LABORATORIES MANAGING CULTURE AND PCR OF MYCOBACTERIA AND NOCARDIA:

NSW

Charlotte Webster
Concord Hospital
Charlotte.Webster@health.nsw.gov.au

Institute of Clinical Pathology and Medical Research
Westmead Hospital (via Vetnostics)

WESTERN AUSTRALIA

Dr Ammie Higgins
PathWest Laboratory Medicine WA
Ammie.higgins@health.wa.gov.au

QEII Medical Centre, Nedlands
Western Australia

CONDITION: MYCOBACTERIA AND NOCARDIA AS CAUSES OF DEEP DRAINING SINUS TRACTS

RECOMMENDED ANTIBIOTICS

C&S is strongly advised in these cases

Mycobacteria:

First line:

Doxycycline (5 mg/kg q12h PO†) and

Pradofloxacin (5-8 mg/kg q24h PO) for *M. smegmatis* complex infections.

Clarithromycin (5-15 mg/kg q12h PO) and

Pradofloxacin (5-8 mg/kg q24h PO) for other *M. fortuitum*.

Clarithromycin/linezolid for *M. chelonae/abscessus*.

Second line:

Clofazimine (4-10 mg/kg q24h PO compounded for cats) and

Amikacin (10-15 mg/kg q24h IV/IM/SC)

***Nocardia* spp.**

First line:

Trimethoprim-sulphonamide (12.5 to 30 mg/kg q12h PO)

Second line:

Amoxicillin (20 mg/kg BID PO) for *N. nova* (not amoxicillin-clavulanate)

Third line:

Clarithromycin (5-15 mg/kg q12h PO)

Pradofloxacin (5-8 mg/kg q24h PO)

†Ensure doxycycline given with food or a water bolus.

USAGE RECOMMENDATIONS

Clarithromycin, amikacin and linezolid are not registered for use in animals and should not be used off-label except in exceptional circumstances for individual animals.

In some refractory cases linezolid can be lifesaving but is expensive. When using linezolid, best to determine peak and trough levels to optimise efficacy and avoid toxicity.

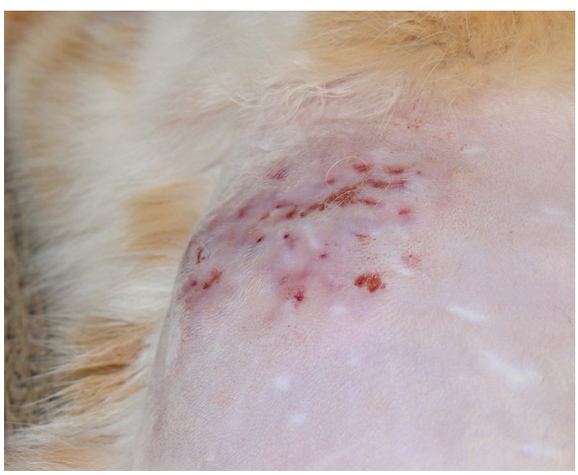
When using trimethoprim-sulphonamide combinations, do not split or otherwise divide the coated tablet. If coated tablets are unavailable give inside a gelatine capsule coated with margarine) combined with a second drug depending on C+S testing. Beware keratoconjunctivitis sicca during therapy.

CONDITION: MYCOBACTERIA AND NOCARDIA AS CAUSES OF DEEP DRAINING SINUS TRACTS

AIDAP TOP TIPS

1. Getting susceptibility data and species identification is expensive in the short term but good value in the long term. Compliance and diligent owners are important as long courses of therapy are required.

2. Richard Malik is available to provide free advice on optimal management of these infections – Richard.Malik@sydney.edu.au.



Close-up showing 'pepper pot' draining sinus tracts.
Photos courtesy of Dr Richard Malik.

Cat with rapidly growing mycobacterial infection.
Photo courtesy Dr Alison Stuart.

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CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

Microsporum canis is responsible for 70 to 95% of canine infections and 94 to 99% of feline infections

Dermatophytoses are superficial fungal infections that involve the skin, hair, and claws.

Microsporum canis is responsible for 70 to 95% of canine infections and 94 to 99% of feline infections.

Microsporum gypseum and *Trichophyton mentagrophytes* account for most of the remaining cases. Infections with unusual species such as *Microsporum persicolor* have been reported; it is uncertain how common these infections occur, but prevalence maybe related to local climate and environmental factors.

Skin lesions: There are many clinical presentations of feline dermatophytosis. Pruritus is variable and can range from nil to severe. In kittens, irregular, annular to circular patches of alopecia with scale, crust and erythema affecting face, ears and forelegs are common. In adult cats, focal, multifocal, or generalised patchy alopecia with or without scale occurs frequently, especially in long haired cats.

Dermatophyte infections are not common in dogs. They are most common in young dogs, dogs from animal shelters or dogs that are debilitated. Circular patches of alopecia with scale, crust, central hyperpigmentation, and follicular papules on the periphery affecting the face, pinnae, paws, and tail is the most frequent presentation in the dog.

Less commonly dermatophytosis can present with folliculitis that may be localised, regional (facial) or generalised; often with furunculosis. Nodular (kerion) lesions on face and legs are exudative, circumscribed type of furunculosis with multiple draining tracts usually associated with *M. gypseum* or *T. mentagrophytes*.

Onychomycosis is a rare, chronic subungual fold inflammation with or without footpad involvement, paronychia, claw deformity and fragility in the dog.

CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON

TESTS FOR DIAGNOSIS

No single test has been identified as the "gold standard".

Surface cytology: acetate tape preparations or firm impression smears may identify fungal hyphae and arthrospores in the stratum corneum.

Trichograms: examine the follicular debris of anagen follicles for the presence of ectothrix fungal elements. This may be aided by clearing agents, such as chlorphenolac.

Wood's lamp examination: positive in most cases of *M. canis* dermatophytosis. Fluorescing hairs are most often detected in untreated infections; fluorescence may be difficult to find in treated cats.

Fungal culture: a soft bristle, sterile toothbrush technique is the recommended method for collecting hair samples. An alternate method of equal sensitivity is to collect the sample using a 4 cm length of acetate tape attached to the end of a glass slide for submission.

Histopathology: a biopsy can be useful.

PCR: a positive test does not always indicate active infection, as DNA from inactivated organisms and non-infected fomite carriers can be detected. A negative PCR in a treated cat is compatible with cure.

KEY STEPS

01 Perform a trichogram to evaluate for ectothrix arthrospores.

02 Wood's lamp examination for *Microsporum canis* infections only.

03 Collect hair and scale for fungal culture using haemostat (dogs).

04 Avoid 'spot' therapy with topical antifungal ointments.

05 Implement topical/systemic/environmental treatment.

CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON

TREATMENT

In most healthy animals, dermatophytosis is a self-curing disease, with full resolution of disease in 10–16 weeks without therapy.

The best treatment protocol is a combination of three modalities:

1. **Topical treatment:** to kill infective material and prevent its dissemination into the environment.
2. **Systemic treatment:** to shorten the time of infection in the individual animal.
3. **Environmental treatment:** to help prevent recurrence of infection or spread to other animals or people in the household.

TOPICAL THERAPY:

i. Total body treatment

Topical therapy inactivates fungal spores and mycelia on and within hair shafts reducing environmental contagion and results in a faster cure than systemic therapy alone. Shampoo therapy, dipping or rinsing with topical antifungal agents is preferred. Twice weekly application of miconazole/chlorhexidine shampoo and/or 2% enilconazole rinse are currently recommended. Chlorhexidine as monotherapy is poorly effective and is not recommended.

ii. Localised (treating only the spots) or whole-body topical therapy

Focal lesions in difficult-to-treat locations such as the face and ears should receive additional specific topical localised therapy with 2% miconazole cream. In animals, not all the lesions may be visible due to the long hair coat. It is almost certain that there are infective spores in non-lesional areas. Therefore 'spot treatment' alone with topical drugs is not recommended.

SYSTEMIC THERAPY:

Systemic antifungal therapy targets the active site of fungal infection and proliferation on the infected animal. Until the infection is eliminated in this site, the infected animal is at risk for further spread of lesions on its body, continued seeding of the hair coat with infective spores, and being a source of infection for other animals and people. Systemic therapy is the treatment of choice for dermatophytosis. It is important to remember that systemic treatment does not rapidly reduce contagion and should be used in conjunction with topical antifungal therapy.

Itraconazole (non-compounded) and terbinafine are the most effective and safe treatments for dermatophytosis.

CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON

ITRACONAZOLE (SPORANOX®)

Itraconazole persists in the skin and nails for weeks to months after dosing and is frequently prescribed for skin infections or onychomycosis.

Itraconazole is generally well tolerated; reported side effects include vomiting and/or anorexia in cats. Signs of dose related hepatotoxicosis have been reported rarely in cats and an idiosyncratic cutaneous vasculitis has been reported in dogs. Itraconazole is reportedly not teratogenic when used at a dose of 5 mg/kg. Compounded or reformulated itraconazole should not be used as studies in both dogs and cats have shown poor bioavailability.

TERBINAFINE

There is limited data on the use of terbinafine in dogs and cats and this drug appears to offer no advantages over itraconazole.

Terbinafine is generally well tolerated; reported adverse effects include vomiting and asymptomatic elevation in liver enzymes. Idiosyncratic acute hepatotoxicity has been reported occasionally. No teratogenicity has been reported. The drug reaches very high concentrations in sebum and stratum corneum and fungicidal concentrations persist in the skin for several weeks after administration in humans and cats, but not in dogs.

FLUCONAZOLE

Fluconazole has poor antifungal efficacy against dermatophytes. It has the highest MIC compared to itraconazole, terbinafine, ketoconazole and griseofulvin for both *Microsporum* spp. and *Trichophyton*. It is not recommended for the treatment of dermatophytosis.

MONITORING AND CURE

Monitoring of response to therapy includes clinical improvement, Wood's lamp evaluation and fungal culture. Clinical cure will precede mycological cure. A lack of resolution of clinical signs and/or development of new lesions indicates a treatment problem or misdiagnosis. Wood's lamp examination can be used to monitor cats for resolution of *M. canis* infections.

In most cats, systemic antifungal treatment is needed for four to eight weeks until clinical resolution of lesions. Topical antifungal therapy should be continued until mycological cure. There is no scientifically established definition, but a negative PCR test is compatible with mycological cure. For *M. canis* infections, a negative fungal culture and a negative Wood's lamp examination (except for glowing tips) are compatible with mycological cure.

ENVIRONMENTAL TREATMENT:

The two reasons for environmental disinfection are:

1. To minimise the risk of disease transmission to people and other animals.
2. To minimise fomite carriage causing false positive fungal culture or PCR results.

Infection from the environment alone is rare. Contact with a contaminated environment alone in the absence of concurrent microtrauma is an exceedingly rare source of infection in both people and animals. Infected owners are found only in households containing cats and owner infection is mostly associated with direct contact with a cat (or kittens). The primary mode of dermatophyte transmission is animal to animal contact even in the presence of a contaminated environment.

CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON

Minimising contamination can be achieved via clipping of affected lesions, topical therapy, and routine cleaning.

CLIPPING THE HAIR COAT

Clipping the hair coat is not necessary in every case of dermatophytosis. In most cases, extensive clipping requires sedation to minimise patient injury and fear. Clipping the hair coat removes fragile hairs that will fracture and release spores into the environment. The owner should be warned that a temporary exacerbation of lesions may occur after clipping.

If the animal is to be clipped in the clinic all debris produced is infectious with zoonotic potential and so rigorous infection control measures should be observed, such as covering table surfaces with disposable drapes, using gowns and gloves, collecting all materials and double bagging, thoroughly disinfecting the room and all equipment used with an appropriate antifungal agent.

TOPICAL THERAPY

The major owner actions that minimise confinement and decrease risk of infection to susceptible people are compliance with oral antifungal therapy and use of topical therapy twice a week.

ROUTINE CLEANING

Twice weekly cleaning/disinfection is recommended. This includes mechanical removal of hair, washing and disinfection of target areas. Daily removal of pet hair from the room/area where the pet is being confined using mechanical removal is recommended (dust cloths, flat mops, sweeping). Use of a daily one-step cleaner can be used on days between more thorough cleaning.

Infective material is easily removed from the environment; if it can be washed, it can be decontaminated.

ANTIFUNGAL DISINFECTANTS

1. **Accelerated hydrogen peroxide (AHP)** is currently available in concentrates, ready-to-use formulations, and over-the-counter products. Its antifungal efficacy against *M. canis* and *Trichophyton* spp. have been shown in several studies.
2. **Sodium hypochlorite (household bleach)** is an effective disinfectant at concentrations ranging from 1:10 to 1:100 even with short contact times. Household bleach diluted at 1:100 and not stored in a brown opaque container retains only 40 to 50% of chlorine after 30 days. If household bleach is used it should be prepared at least once weekly and stored in a dark opaque container. There are many reasons not to use bleach and these include a lack of detergency which is a critical factor for disinfection, potential to react with other chemicals to create toxic gases, unpleasant odour, damage to hard surfaces, discolouration of fibres and coloured surfaces, damage to floor finishes, rapid loss of efficacy once diluted and human health concerns. The product is an irritant to both animals and people.

DISINFECTION OF NONPOROUS SURFACES

Disinfection of nonporous surfaces involves three steps. The first is the mechanical removal of all debris using vacuuming or sweeping as disinfectants will not work in the presence of organic debris.

CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON

The second is the washing of the target surface with a detergent to remove debris. Detergents must be rinsed from the target surface because some may inactivate disinfectants. These two steps are the most important and will often decontaminate a surface. The final step is the application of a disinfectant to kill any residual spores.

DISINFECTION OF LAUNDRY

Fabrics contaminated with infective spores and hairs can be washed in any water temperature without the addition of sodium hypochlorite. Two washings on the longest wash cycle are effective. Spray the surface of the washing machine and the dryer with accelerated hydrogen peroxide after use.

DISINFECTION OF CARPETS AND WOOD FLOORS

Vacuuming alone does not decontaminate carpets exposed to infective *M. canis* hairs and spores but is recommended to remove debris including infective hairs. Disinfect the vacuum with AHP spray and/or wipes.

Wash exposed carpeting twice with a carpet shampooer with detergent or use a steam cleaner. Steam cleaning has the fastest drying time and no discolouration. Pre-treat heavily contaminated carpets with disinfectant and then wash with a beater brush carpet shampooer. Disinfectants (chlorhexidine and sodium hypochlorite) may discolour carpets.

There are no safe surface disinfectants for wooden floors. Wooden floors can be decontaminated via daily removal of hair and dust using commercial disposable cleaning cloths designed for dry mopping and washed twice weekly with a wood oil soap.

CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON

ANTIFUNGAL AGENTS USED

First line:

Dogs: Itraconazole (5–10 mg/kg q24h PO)

Cats: Itraconazole (5 mg/kg q24hr PO)

Second line:

Terbinafine (30-40 mg/kg q24h PO)

USAGE RECOMMENDATIONS

Significant duration: 6–10 weeks.

Treat until cured.

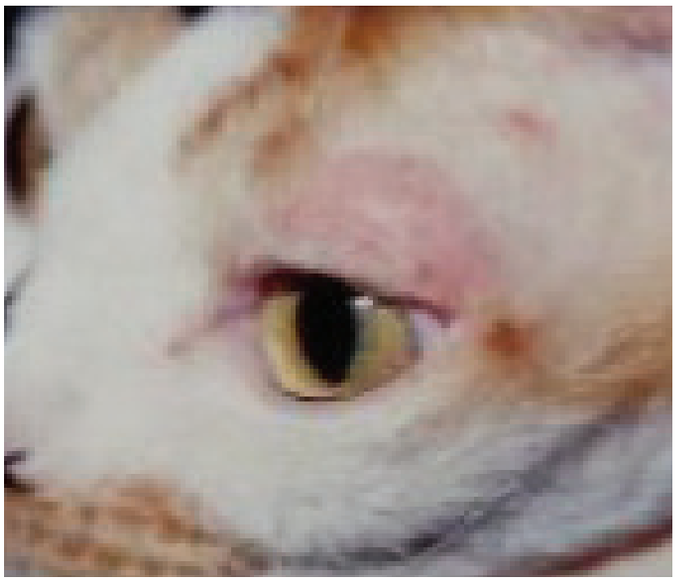
For itraconazole in cats a regime of 5 mg/kg q24h PO for three one week-on and one week-off cycles is recommended.

AIDAP TOP TIPS

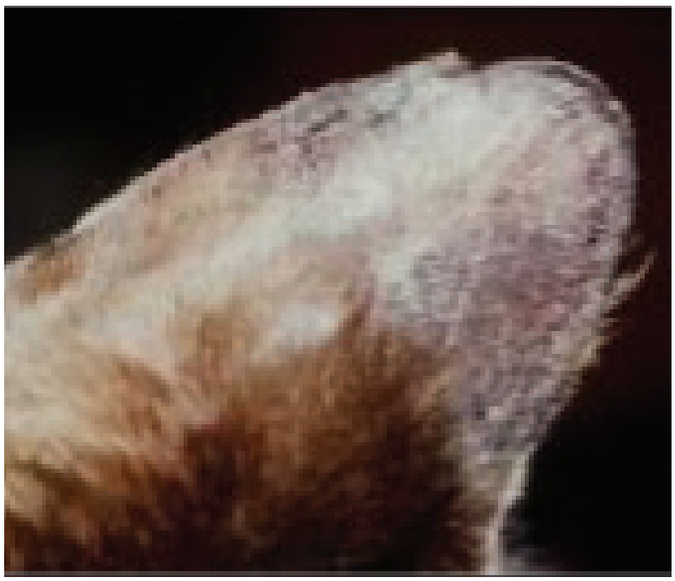
Our treatment recommendations for dermatophytosis for dogs and cats:

1. 2% miconazole, 2% chlorhexidine shampoo baths twice a week.
2. 0.2% enilconazole (Imaverol®) rinse twice a week (not registered for use in cats).
3. Environmental decontamination: important but zoonotic infection unlikely from just environmental exposure.

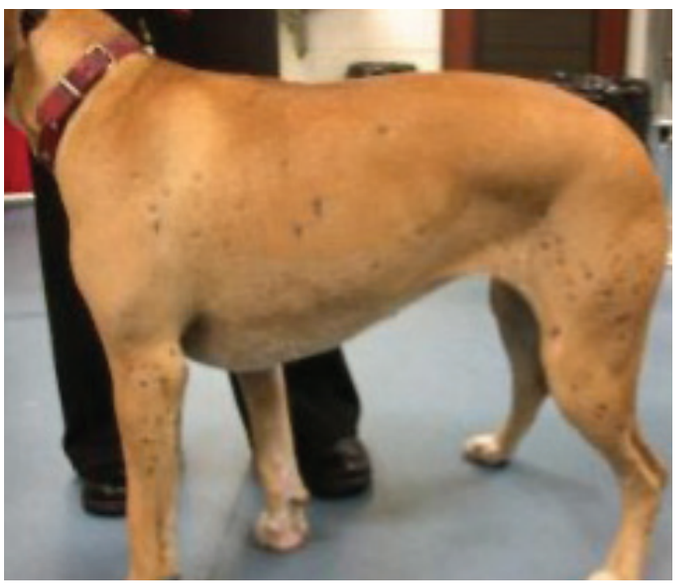
CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON



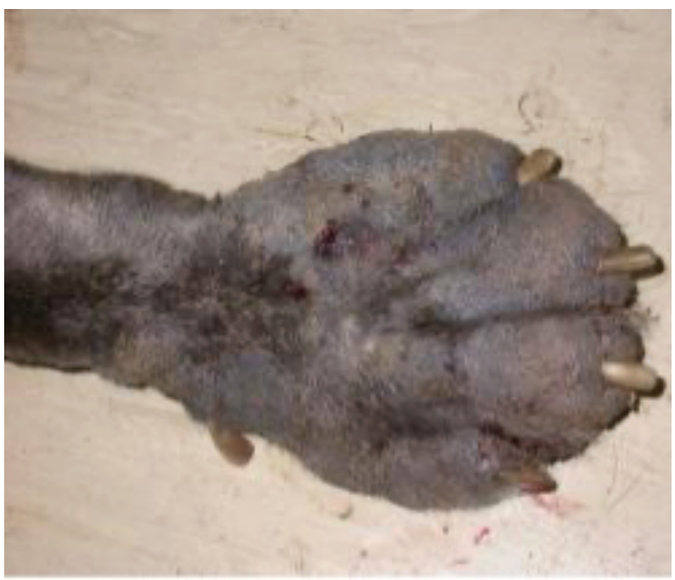
M. canis infection on preauricular skin showing mild inflammation.



M. canis infection of pinnal tip producing alopecia with minimal skin changes.



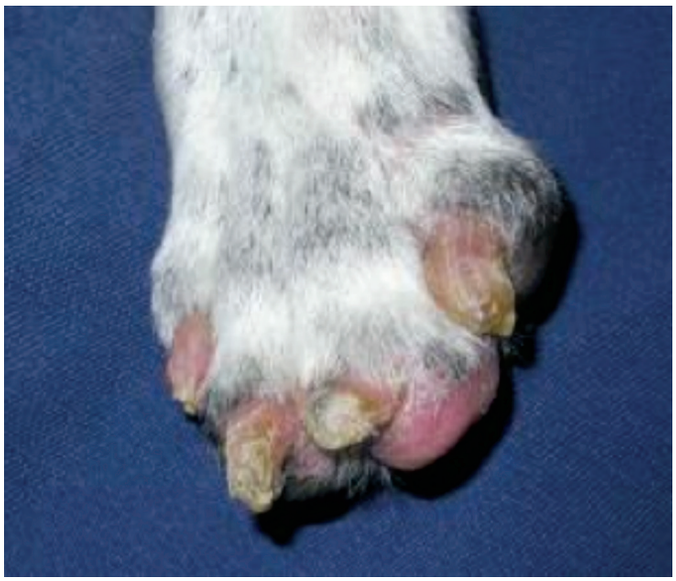
Multifocal alopecia due to *Trichophyton* infection inflammation.



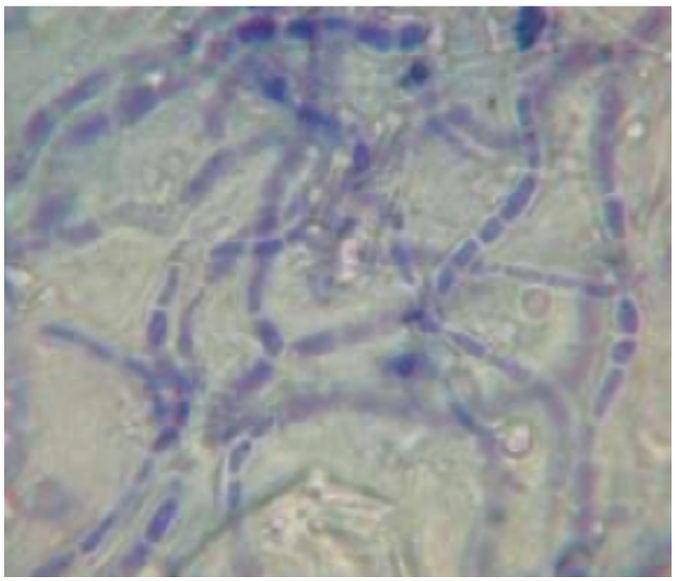
Severe dermatophytosis causing soft tissue swelling.

Photos courtesy of Dr Mandy Burrows & Dr Mike Shipstone.

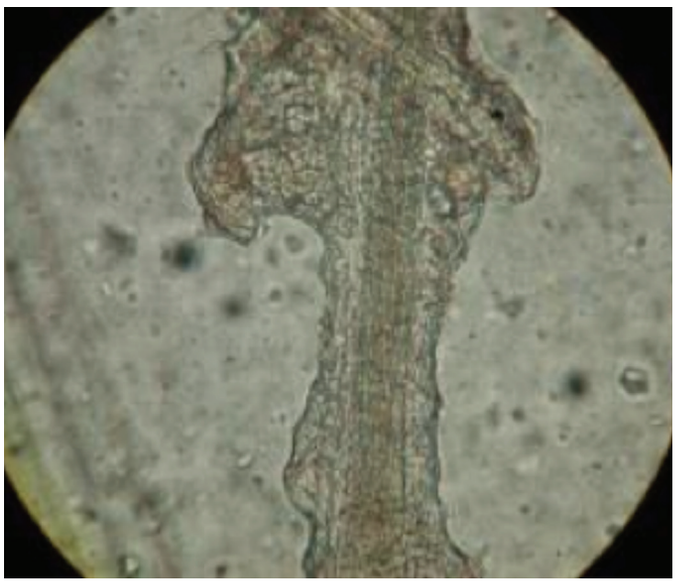
CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON



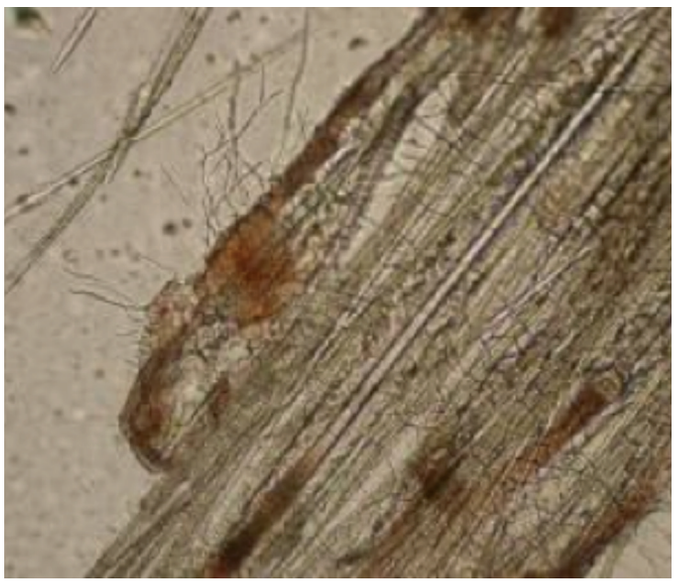
Onychomycosis causing nail deformation.



Fungal hyphae on surface cytology.



Fungal hyphae surrounding the hair shaft.



Fungal hyphae surrounding the hair shaft.

Photos courtesy of Dr Mandy Burrows & Dr Mike Shipstone.

CONDITION: DERMATOPHYTE INFECTIONS - MICROSPORUM OR TRICHOPHYTON

Dermatophyte lesions of *M.canis* affecting a child. Photo courtesy of Dr Richard Malik.

Key references:

Moriello KA, Coyner K, Paterson S, Mignon B. Veterinary Dermatology. 2017; 28(3): 266-e68.

CONDITION: OTITIS EXTERNA (UNCOMPLICATED, FIRST EPISODE AND COMPLICATED, RECURRENT)

BACKGROUND/NATURE OF INFECTION/ ORGANISMS INVOLVED

In cats, *Staphylococcus pseudintermedius* and *Pasteurella multocida* are mostly isolated from otitis externa cases. *Malassezia* are relatively more important and have been found in more than 95% of cases of otitis externa.

The normal flora is generally Gram-positive, with higher bacterial counts retrieved from the vertical external ear canal than the horizontal ear canal. Commensal and pathogenic bacteria rapidly colonise the external ear canal where changes in the microclimate following inflammation modify the environment. The microbial proliferation exacerbates and perpetuates the inflammatory response within the ear canal. Once inflamed, there is a shift towards increased bacterial numbers, initially coagulase positive staphylococci and with more chronic inflammation, Gram-negative bacteria.

Because potential pathogens can be recovered in the absence of disease (as they can from the skin surface), it is assumed that they are unable to initiate disease in the ear. However, once the ear becomes inflamed or macerated, proliferation may occur, therefore bacteria are considered perpetuating rather than primary or predisposing factors in otitis externa.

In dogs, coagulase positive *Staphylococcus* spp. (*S. pseudintermedius*, *S. schleiferi*) are mostly isolated in acute otitis and as a single organism. *Streptococcus* spp., *Pseudomonas aeruginosa*, *Proteus mirabilis, E. coli, Corynebacterium* spp., *Klebsiella* spp. are also frequently identified. *Pseudomonas* organisms are frequently identified in chronic recurrent cases or those cases that have had long term antimicrobial treatment.

In cats, *Staphylococcus pseudintermedius* and *Pasteurella multocida* are mostly isolated from otitis externa cases. *Malassezia* are relatively more important and have been found in more than 95% of cases of otitis externa.

CONDITION: OTITIS EXTERNA (UNCOMPLICATED, FIRST EPISODE AND COMPLICATED, RECURRENT)

TESTS FOR DIAGNOSIS

In many cases of otitis, a single organism can be isolated on bacterial culture of exudate, but in others, multiple potentially pathogenic organisms are identified. It is of critical importance to also conduct cytological examination of the otic discharge when a C&S test is performed. This allows determination of the dominant population of bacteria evident, the presence of leukocytes, and the presence of phagocytosed bacteria.

Cytology is the first step. It is mandatory in ALL cases of otitis externa and should be repeated at each visit. Studies have shown that cytology is more sensitive than C&S testing in identifying the presence of bacteria or yeast. For example, sensitivity of cytology for detection of Gram-positive cocci, Gram-negative rods, and yeasts was 84%, 100% and 100%, respectively. The sensitivity of culture for detection of these organisms was 59%, 69% and 50%, respectively.

Normal cerumen does not have high stain uptake because of the high lipid content. Outlines of occasional squames may be seen. Inflammation leads to increased numbers of squames (some of which may be nucleated indicting faster epithelial turnover with incomplete keratinisation before desquamation). As the severity of inflammation increases, inflammatory cells may be seen along with increasing numbers of organisms. Higher cellular content of cerumen may also be appreciated by increasing stain uptake on the stained slide (before microscopic examination is even started).

The number of organisms and inflammation should be assessed on a scale of 1 to 4. Normal ears may have a few yeast and Gram-positive cocci per oil field but **not** rods. The finding of yeast or cocci should be correlated with the findings of the otoscopic examination. Some animals may have few organisms yet show marked inflammation and exudation, whilst others seem to be able to tolerate quite large numbers without any pathologic changes. Repeating the cytology at each revisit allows accurate assessment of response to therapy. Medical treatment should continue until otoscopic and cytologic examinations demonstrate no pathologic change.

KEY STEPS

Otic examination alone is not sufficient, and the following minimum database is necessary to identify both the nature and type of the otitis as well as any underlying primary or predisposing factors:

01 Thorough dermatological history.

02 Complete physical examination of all areas of integument.

03 Thorough otic examination (may require sedation/anaesthesia – particularly cats).

04 Otic cytology.

05 Implement topical antimicrobial therapy based on cytological findings.

06 Systemic antibiotic therapy is not indicated for otitis externa.

CONDITION: OTITIS EXTERNA (UNCOMPLICATED, FIRST EPISODE AND COMPLICATED, RECURRENT)

TREATMENT

Topical therapy is the key to successful resolution of the majority of cases of otitis externa which is essentially a surface infection. Essential to this therapy is the successful removal of exudate. If the medication cannot penetrate the full length of the ear canal, then treatment is likely to fail. The choice of appropriate active ingredients and vehicles for treatment of otitis externa is usually made empirically based on cytological examination of ear canal exudates and otoscopic examination of the inflamed ear canals.

Most commercially produced topical products contain one or more antibacterial, antifungal, and anti-inflammatory agents in various combinations as well as vehicles, solubilisers, stabilisers and surfactants.

Clients need to be shown how to administer medications correctly. Failure to do this is a significant cause for treatment failure. An adequate volume of medication must be delivered to line the entire canal. Getting clients to count drops increases the time for administration and fundamentally means that the nozzle of the bottle is not in the canal, reducing penetration of the medication. Putting the nozzle of the bottle in the canal and telling clients to use a "squeeze" means that both under and overdosing are risked because the amount to medication is not measured out.

A graduated syringe is recommended to accurately measure ear medications and dispense them into the ear canal.

A broad guideline depending on the length and diameter of the ear canal would be:

1. 0.15-0.2 mL for a cat or a Shih Tzu.
2. 0.6 mL for a Labrador.
3. 1 mL for a German Shepherd or very large dog.
4. Twice daily dosing may require slightly smaller volumes to avoid overdosing.
5. It is important to remember that the bulla of cats is divided by an incomplete bony septum. This septum is rooved by a sympathetic nerve plexus that can be easily damaged causing Horner's syndrome. Therefore, products should be used with caution if the tympanum is ruptured.

Duration of therapy

For acute disease a minimum of 5 to 14 days therapy depending on the degree of inflammatory change (oedema, hyperplasia, and erosion, ulceration) is to be expected. Rechecks every one to two weeks are necessary to ensure that ears are cytologically and otoscopically resolved prior to cessation of therapy. It is not uncommon to have a dog clinically resolved with otoscopically normal ears because of anti-inflammatory medications, where cytology is still not normal.

CONDITION: OTITIS EXTERNA (UNCOMPLICATED, FIRST EPISODE AND COMPLICATED, RECURRENT)

Antimicrobial therapy for ears with mainly cocci on cytology

Coccoid organisms will be *Staphylococcus* spp. or *Streptococcus* spp. The challenge for empirical therapy for cocci is the relative resistance of streptococci to some of the routine antibiotics, which otherwise tend to have reasonable activity for most *Staphylococcus* spp. Products containing antibiotics with good efficacy against both bacteria are desirable. Enrofloxacin, marbofloxacin or chloramphenicol are reasonable choices. The use of florfenicol (Osurnia®) should be limited to dogs with evidence of MRSP on culture and susceptibility testing.

When the tympanic membrane is ruptured, enrofloxacin is preferred although its activity against streptococcal infections is not always reliable. If this inadequate, then options include the use of systemic antibiotics based on C&S testing and ear wicks impregnated with more effective (but not middle ear safe) ointments.

Systemic antibiotics are used if there is significant involvement of the pinna, if a methicillin-resistant staphylococcal infection is identified on C&S testing or if otitis media is evident. They are *unreliable* in our experience used as a sole therapy of otitis externa.

Antimicrobial therapy for ears with mainly rods on cytology

Rods are rarely found in healthy ears. In Australia, most rods identified on culture are *Pseudomonas aeruginosa* with *Proteus* and *E. coli* identified in about 11% to 20% of the otitis ears. Less common rods include *Corynebacterium* spp. and *Klebsiella. Corynebacterium* is usually found as part of a mixed culture and is probably of minimal significance unless isolated in pure growth.

EDTA (Otoflush®) acts as a chelating agent and enhances activity of topical antibiotics against otic pathogens by decreasing stability and increasing permeability of the cell wall. The ear canal should be filled with the solution 15 to 30 min before a topical antibiotic is applied every 12 hours. First line antibiotic therapy includes enrofloxacin compounded 1.5% with dexamethasone. Once the tympanic membrane is intact and the inflammation controlled then products containing gentamicin (Otomax® q12hrs and Mometamax® q24hrs) can be used if the ear is clean. C&S testing is indicated if the infection fails to respond. Marbofloxacin (Aurizon®) q12hrs or ciprofloxacin (Cipro HC®) can be used topically as a second-line antibiotic. Systemic antibiotics are only used if there is significant involvement of the pinna or if otitis media is evident. They are unreliable in our experience used as a sole therapy of otitis externa.

CONDITION: OTITIS EXTERNA (UNCOMPLICATED, FIRST EPISODE AND COMPLICATED, RECURRENT)

Antimicrobial therapy for ears with mainly yeast on cytology

Malassezia can be retrieved from up to 80% of otitis ears in dogs. Dogs with atopy can produce IgE to *Malassezia* which means that the degree of inflammation depends on the host response rather than the numbers of yeast. *Malassezia* can be retrieved from up to 95% of otitis ears in cats. In some cases, the inflammation seen is disproportionately large compared with the number of organisms seen on cytology.

Disinfectants are useful as sole therapy where there are low numbers of yeast and minimal inflammation or occasionally in cases apparently resistant to other antifungal ear medications. The only one with any proven efficacy against *Malassezia* is Epiotic®. This is not a good ceruminolytic so penetration is an issue where there is significant exudate. Alpha Ear Cleaner® has good activity against yeast and is a good ceruminolytic. None of these products are middle ear safe.

A new product (Sonotix®) containing tromethamine, isopropyl alcohol, ethoxydiglycol, capric glycerides is an effective cerumenolytic.

Most of the major commercial combination ear products (except Baytril Otic®) are reliable in the therapy of an uncomplicated yeast otitis. Surolan® q12h or Otomax® q12h / Mometamax® q24h containing miconazole and clotrimazole, are useful first line treatments. None of these products are middle ear safe.

Systemic use of antifungal medication is a consideration where there is a fungal otitis media and for sole or adjunctive therapy where topical medications are not possible or there are severe proliferative changes in the ear canal.

Secondary changes are sequelae that occur due to acute and chronic inflammation of the external ear canal that when present will increase the likelihood of relapse of otitis externa irrespective of whether the trigger factor has been controlled. Sequelae secondary to otitis externa include epidermal or glandular hyperplasia, inflammatory polyps, fibrosis, stenosis, calcification, ceruminoliths, otitis media and complete occlusion of the external ear canals.

CONDITION: OTITIS EXTERNA (UNCOMPLICATED, FIRST EPISODE AND COMPLICATED, RECURRENT)

AIDAP TOP TIPS

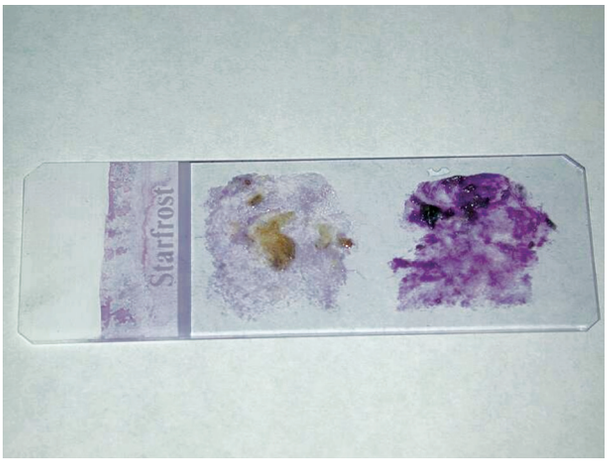
Bacterial C+S testing

The commonly accepted practice is that a bacterial C+S testing should be performed if:

- Rods are seen on cytology
- Ulceration of the epithelium is present
- The condition is recurrent
- There is no response to appropriate treatment
- Otitis media is present.

However there have been several recent studies raising doubts as to the usefulness and accuracy of culture results (Graham-Minze and Rosser 2004). It has been suggested that the culture may identify organisms from the external ear canal that are low in number and possibly irrelevant in the pathogenesis of the disease state. As such the initial cytology may be a better indicator of the relative importance of the different organisms present.

Robson (2008) has proposed the following:
"That bacterial C+S testing should be performed when cytology shows a uniform or near uniform pattern of bacteria AND when appropriate empirical therapy has failed AND all other causes of failure of therapy have been ruled out as well as causes of otitis media".



Sample on right showing marked stain uptake due to presence of neutrophils.

Photo courtesy of Dr Mandy Burrows & Dr Mike Shipstone.

Key references:

1. Sharma VD and Rhodes HE. J. Sm An Pract. 1975; 156: 241–247.
2. McCarthy G and Kelly WR. Irish V J. 1983; 36: 53–56.
3. Colombini S, Merchant SR, Hosgood G. Vet Dermatol. 2000; 196: 84–90.
4. Graham-Minze CA and Rosser EJ J. Am An Hosp Assoc. 2004; 40(2): 102–108.

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Zoetis would like to thank the dedicated members of AIDAP for all their hard work and contribution towards these guidelines. AIDAP, the Australasian Infectious Diseases Advisory Panel, is a committee of Specialists with fields in Internal Medicine, Feline Medicine, Dermatology, Microbiology, Dentistry and Surgery. The panel works together with Zoetis to assist with the ongoing understanding of the nature of infectious diseases; the understanding of how to treat infectious diseases; and also the current rationale for the appropriate use of antibiotics.

Please note, these recommendations are based entirely on the decisions made by the AIDAP committee, and some of these recommendations include the "off label" use of certain medications.These off label uses are not endorsed by Zoetis.

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AIDAP AUSTRALASIAN INFECTIOUS DISEASES ADVISORY PANEL

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