

# Cracking the otitis code: practical diagnostics for better outcomes

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Otitis externa (OE) is common, causes considerable distress to the patient and due to difficulties in administering treatments, its often recurrent nature and costs it can be a major concern for dog owners. This affects the owner's relationship with veterinary practices and with their own pet. OE is defined as inflammatory disease between the opening of the external canal and the drum, clients and sometimes veterinary professionals get sidelined into always considering this to be an infection and it is common to hear the term 'ear infection' for all forms of OE.

The signs of primary disease may be subtle, and this often leads to dogs being presented only when there is marked bacterial or yeast overgrowth, smell, discharge (otorrhea), pain or pruritus or worse secondary changes.

The PPP system, initially described by John August (1988) allowed development of the current PSPP system in which the various factors playing a part in the development and continuation of OE are described allowing a better understanding of the aetiopathology and targeted treatment. The factors are Primary diseases (P) - the underlying cause of disease; Secondary infections (S) – complicating infections that may result in a dramatic increase in signs; Perpetuating changes (P), in which changes to the structure and function of the ear cause continuation or rapid return of disease and Predisposing factors (P) that allow more rapid or more severe ear disease to occur (e.g. anatomic differences in the canal or over wetting with ear cleaners or swimming will all cause increased dysbiosis or inflammation through changing barrier function). The components of PSPP are shown in Table 1.

The interrelationship of these factors is complex. Initially inflammation leads to changes in the ear canal – increased secretions and reduced barrier function with consequential dysbiosis. Over time the level of secondary infection develops, ultimately leading to purulent infections. More profound functional and physical changes in the ear result and once the ear is severely affected, the primary reason can resolve but ear disease will carry on effectively 'decoupling' the ear disease from the initial cause. This model of otitis is summarised in Figure 1.

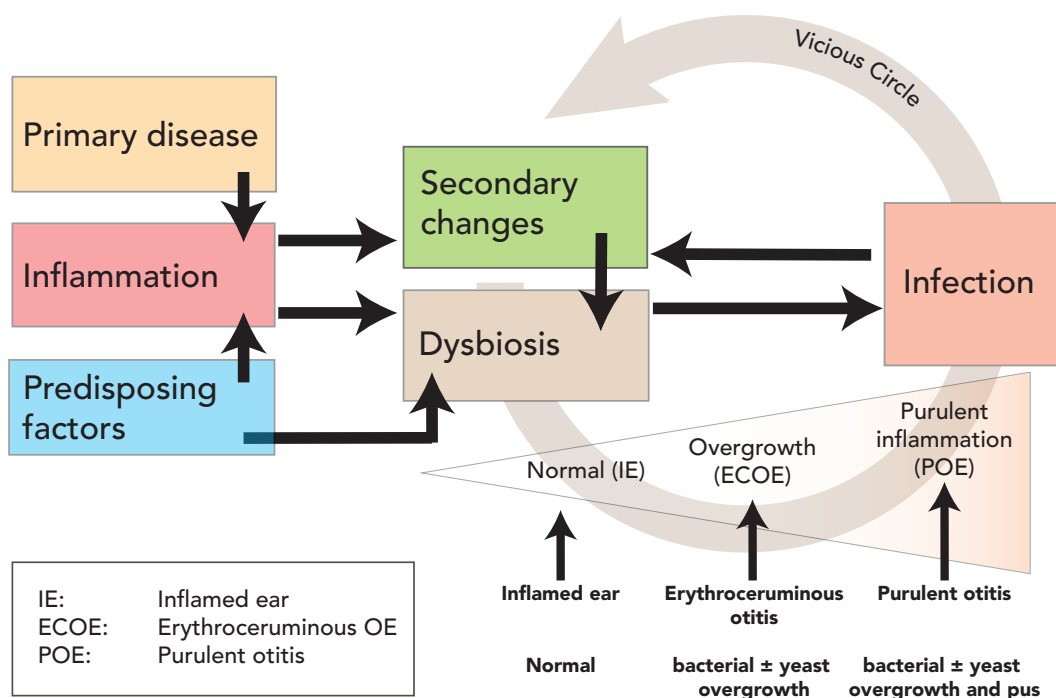
Secondary infections follow a 'typical' course when there is no intervention, initially being seen as a non-inflamed increase in bacteria and/or yeast. Usually *Staphylococcus pseudintermedius* and *Malassezia pachydermatis* predominate. These organisms, part of the normal flora are adapted for skin, but as changes in the ear become more severe, potentially aided by antibiotic or antiseptic pressure, less

skin adapted organisms such as *Pseudomonas aeruginosa*, *Escherichia coli* and other gram-negative rods can invade and overtake the commensals. These bacteria are more often multi-drug resistant and cause more severe clinical signs. The speed of this microbial progression is very variable and the full range of infection is seen at presentation with some presented with acute severe purulent rod-based disease.

### Classification of otitis

This progression allows us to classify the type of otitis. Dogs without overt bacterial or yeast overgrowth and with inflamed ears are described as having uncomplicated **inflammatory OE [IOE]**, whereas otitis externa with overgrowth, but no neutrophilic exudate is termed **erythroceruminous OE (alternatively erythematoeruminous [ECOE])**. Otitis externa in which there is inflammatory exudate present is termed **purulent or suppurative [POE or SOE]**. The majority of OE seen by veterinary surgeons is erythroceruminous (ECOE) evidenced in three studies, in which seventy-five to eighty-five percent of 1678 dogs presented to primary care clinics for OE had ECOE (Carlotti and Le Roy 1997, Bensignor, Legeay et al. 2000, Aymeric-Cuingnart and Bensignor 2018) and allergic skin disease was the most common primary cause identified (Carlotti and Le Roy 1997).

Fig 1. The pathogenesis of otitis externa (the PSPP model)



Primary disease causes inflammation in the ear canal and this results in dysbiosis, followed by overt overgrowth and in the face of high numbers of microorganisms, there is a purulent response. Perpetuating factors may be associated with the initiation or prolongation of disease. As disease progresses, changes within the ear potentially decouple the perpetuating and secondary infection from the primary disease, explaining why ear disease may not resolve with systemic therapeutics or management options aimed at resolving the primary problem (e.g. an elimination diet). For this reason, in many severe cases of OE treating the primary disease is performed concurrently or sometimes after resolving the secondary signs.

Table 1. Components of the PSPP System

Classification	Causes
<b>Primary</b>	<p>Parasites</p> <ul style="list-style-type: none"> <li>» <i>Otodectes cynotis</i></li> <li>» <i>Demodex</i> spp.</li> <li>» Scabies</li> </ul> <p>Keratinisation disorders</p> <ul style="list-style-type: none"> <li>» Primary idiopathic seborrhoea</li> <li>» Hypothyroidism</li> </ul> <p>Foreign bodies</p> <ul style="list-style-type: none"> <li>» Grass awns</li> </ul> <p>Hypersensitivity</p> <ul style="list-style-type: none"> <li>» Atopic dermatitis</li> <li>» Food allergy</li> <li>» Medication adverse drug reactions</li> </ul> <p>Glandular disorders</p> <ul style="list-style-type: none"> <li>» Breed specific excessive wax production (e.g Cocker &amp; English springer spaniels &amp; Labrador retrievers)</li> </ul> <p>Miscellaneous</p> <ul style="list-style-type: none"> <li>» e.g. feline proliferative &amp; necrotising otitis externa</li> </ul>
<b>Secondary</b>	<p>Bacterial and yeast infections</p> <p>Acute: <i>Malassezia pachydermatis</i> and <i>Staphylococci</i></p> <p>Chronic: As above ± <i>Pseudomonas</i> and other environmental</p> <p>Occasionally fungal e.g. Aspergillosis</p>
<b>Perpetuating</b>	<p>Changes in canal wall</p> <ul style="list-style-type: none"> <li>• acute changes <ul style="list-style-type: none"> <li>» Inflammation causing failure of epithelial migration</li> <li>» Oedema, epithelial hyperplasia</li> </ul> </li> <li>• Chronic change <ul style="list-style-type: none"> <li>» Proliferative changes</li> <li>» Canal stenosis</li> <li>» Calcification of peri-cartilaginous</li> <li>» Fibrous tissue</li> </ul> </li> </ul> <p>Changes in glandular tissue</p> <ul style="list-style-type: none"> <li>• Hyperplasia of ceruminous and sebaceous glands, (seen initially as a papular change)</li> <li>• Hidradenitis</li> </ul> <p>Changes in the tympanum</p> <ul style="list-style-type: none"> <li>» Dilation Rupture</li> <li>» Diverticulum (false middle ear – cholesteatoma)</li> </ul> <p>Otitis media</p> <ul style="list-style-type: none"> <li>• Acute <ul style="list-style-type: none"> <li>» Foreign material</li> <li>» Mucopurulent exudate</li> </ul> </li> <li>• Chronic <ul style="list-style-type: none"> <li>» Biofilm formation</li> <li>» Granulation material ± bony change in the bulla</li> </ul> </li> </ul>
<b>Predisposing</b>	<p>Conformation</p> <ul style="list-style-type: none"> <li>» Excessive hair growth in canals (e.g. poodle)</li> <li>» Hairy concave pinna (e.g. cocker spaniel)</li> <li>» Pendulous pinna (e.g. basset hound)</li> <li>» Stenotic canals (e.g. shar pei)</li> </ul> <p>Excessive moisture</p> <ul style="list-style-type: none"> <li>» Environment (heat &amp; high humidity)</li> <li>» Water ('swimmer's ear', grooming, cleaners)</li> </ul> <p>Obstructive ear disease<sup>†</sup></p> <ul style="list-style-type: none"> <li>» Feline apocrine cystadenomatosis</li> <li>» Neoplasia /Polyps</li> </ul> <p>Otitis media</p> <ul style="list-style-type: none"> <li>» Primary otitis media</li> <li>» Primary secretory otitis media in CKCS</li> <li>» Sepsis</li> </ul> <p>Treatment effects</p> <ul style="list-style-type: none"> <li>» Altered normal microflora (e.g. inappropriate cleaner)</li> <li>» Trauma from cleaning or plucking</li> </ul>

<sup>†</sup> Neoplasia and polyps are traditionally considered to be predisposing factors, but that classification fits better with the concept of otitis externa being considered ear infection, and in modern thinking they should likely be described as primary disease.

# Investigation of OE

## History and clinical examination

The signalment and history provide many clues as to the nature of the disease. For instance, young, kennelled dogs are more likely to suffer ear mite infestation and seasonality is often noted in both grass awn penetration and harvest mite infestation. In many dogs with hypersensitivity, the history provides the wider context of the patient's allergic disease. Regardless of the cause, a key question is whether treatments are possible at home. This may explain prior treatment failure and sets the scene for future management.

Clinical examination should consider general health, any other skin disease and then focus on the ears. Information about more generalised skin disease helps in determining the primary cause and may provide additional avenues for treatment. The clinician should consider neurological problems associated with facial nerve disease (e.g. facial paralysis), the sympathetic chain (dry eye and Horner's syndrome) and evaluate hearing or balance abnormalities. Signs of **otitis media (OM)** may be more subtle and poorly localised pain, depression or problems eating may be noted in the consulting room. In dogs, OM often represents an extension of OE, but primary secretory otitis media should be considered in Cavalier King Charles spaniels and other brachycephalic breeds.

To examine the ears, first look at both sides of the pinnae and then at the opening of the canal. Careful palpation of the canal externally can reveal hardening (fibrosis and ossification of the auricular cartilages) giving a clue to severe disease. Assess whether it is possible to perform otoscopic examination. Severe pain, often accompanied by ulceration makes otoscopic examination inappropriate without analgesia  $\pm$  sedation and in such circumstances cytology ( $\pm$  bacteriology) and initial therapy is often the preferred option, even before considering video otoscopy. However, at some point early in the disease otoscopy must be performed.

## Otoscopic examination

In performing otoscopy, endeavour to have the head facing straight ahead with a slightly extended neck and level jaw line. This ensures the canals are not compressed but can be difficult to achieve in a fractious or worried patient. Using a box-style muzzle can be useful to allow more gentle handling than when using the hands alone on the head regardless of the bite risk. Ensure that the speculum (ear cone) has undergone a vigorous cleaning process as Meticillin-resistant *Staphylococci* (MRS) (e.g. *S. pseudintermedius*, *S. aureus* or *S. coagulans*) as well as multi-drug-resistant (MDR) rods (e.g. *Pseudomonas aeruginosa*) may be easily transmitted between patients. The handles and heads of otoscopes should also be cleaned.

## Discharge:

The nature of the discharge, although not always predicting the organisms or inflammation present can often be divided into normal looking wax, variable discharge (most difficult to judge) and frank pus. In addition, by looking at the discharge on the walls of the canal, the movement of normal wax out of the ear by epithelial migration (Tabacca, Cole et al., 2011) can sometimes be assessed. When the discharge has a mucoid or 'snotty' appearance biofilm is likely.

## Canal surface:

The surface of the ear allows assessment of both severity and chronicity of OE. In acute disease, ulceration is a severe manifestation of purulent otitis. In milder cases erythema indicates inflammation. Chronic signs such as glandular hyperplasia, in which the wall acquires a papular (cobblestone pattern) is often accompanied by hair loss in the ear and this indicates chronic disease. In more chronic cases the canal examination can reveal marked stenosis and hyperplasia. This hyperplasia can appear polyp-like and care is needed to decide between hyperplasia and tumour in some cases. Most dogs have sparse hairs over the canal, but in dogs with anagenic hair growth (such as poodles) this can form a matted barrier to further examination. Interestingly in many poodle-crosses dense hair is not found throughout the whole canal and useful otoscopy can often be achieved.

## Tympanic membrane

Observing the ear drum is often impossible due to discharge, swelling and the long length of some ear canals. Where visible, the surface should resemble a flat sloping semi-translucent structure with the 'J' shaped manubrium of the malleus clearly visible. Pus behind the drum may change the colour to yellow or green, there may be capillaries crossing the drum indicating inflammation or repair and the drum may become greyed in chronic disease. Ruptures can be very difficult to see. The pars flaccida of the tympanic membrane sits above the rest of the drum and is variable in size and commonly has some visible small vessels over the surface (Cole 2010).

The integrity of the drum is a major question that affects diagnosis, treatment, and outcome, but in many conscious animals cannot be answered. Acute inflammatory or erythroceruminous otitis externa are relatively unlikely to cause ear drum rupture whereas chronic severe rod-based purulent otitis represents a considerable risk. In this context the clinician can estimate risk and communication with the client in this respect is key as all ear treatments carry a risk of causing ototoxicity. However, potential ototoxicity of treatments must be set against the need to treat infection and inflammation that may also cause deafness and neurological disease. Owners should be asked to report any suspicion of ototoxicity as soon as it occurs, to allow removing potentially offending treatment and investigate the situation.

## Scoring ear disease

Increasingly otitis severity in clinical studies is described using a formal scoring scheme. OTIS3 (Nuttall and Bensignor 2014) can be used to provide clear comparisons between dogs and between individual studies. Adding scores for cytology (see below), pruritus and pain provides a holistic view of the situation for clinical records or studies. See Table 2.

Table 2. OTIS-3 Scoring

Clinical parameter	Minimum Score	Maximum Score
Erythema	0	3
Oedema/Swelling	0	3
Erosion/Ulceration	0	3
Exudate	0	3
<b>Total score</b>	<b>0</b>	<b>12</b>

## Cytology sampling

This is a key technique and should be performed in all cases if possible (Angus 2004, Shaw 2016). Even when otoscopy is not possible, cytology can be performed. Using a gloved finger to collect *superficial* discharge can avoid any apprehension or risks from using a cotton bud, alternatively asking the owner to collect a cotton bud sample may be possible when veterinary intervention is highly resented. Ideally a cotton bud should be placed down the vertical canal to the junction with the horizontal canal, rolled gently and then the material rolled carefully onto a slide. For obviously purulent samples using all three parts of a rapid haematology stain is appropriate, but for more oily or waxy samples using just the eosin and methylene blue stains (2 & 3) avoids the sample being lost into the fixative. There is no value in heat fixing, but warming gently to dry the slide can be useful to speed the procedure (Toma, Cornegliani et al. 2006).

In reporting cytology, three aspects of your findings should be reported in each case. These are **tissue cells** (in this context epithelial cells and most often squames), **inflammatory cells** (most commonly neutrophils and occasionally macrophages) and **micro-organisms**. Red blood cells may be noted when there is ulceration.

Scoring cytology in a relatively reproducible way means that all clinicians involved with a case can appreciate the findings at later date. The recommended scoring scheme described by Budach is shown below in Table 3 (Budach and Mueller 2012).

Table 3. Scoring scheme for cytology findings (e.g. Budach and Müller, 2012)

Classification	Description
0	No bacteria/yeast/inflammatory cells
1+	Occasional bacteria/yeast/inflammatory cells present, but slide must be scanned carefully for detection
2+	Bacteria/yeast/inflammatory cells present in low numbers, but detectable rapidly without difficulties
3+	Bacteria/yeast/inflammatory cells present in larger numbers and detectable rapidly without any difficulties
4+	Massive amounts of bacteria/yeast/inflammatory cells present and detectable rapidly without difficulties

## Epithelial cells

These are more often flat rather than rolled in ear cytology. They may show some signs of degeneration, but are usually easily discernible. **Prominent melanosomes can be confused with bacteria**, and their variable red brown to black colour and refractile nature needs to be recognised.

## Microorganisms

Although bacteria are normal in the canal with a predominance of gram-positive cocci including *Micrococcus* spp., Coagulase negative *Staphylococci*, *Staphylococcus coagulans*, *Staphylococcus pseudintermedius* and *Streptococcus* spp., these are rarely seen on cytology in the normal ear. The gram-positive rod *Corynebacterium* sp. is also present but rarely seen even in OE.

In otitis externa, cocci particularly *Staphylococcus* spp. and *Streptococcus* spp., and rods such as *Pseudomonas aeruginosa* are noted on cytology. Rods may occasionally be seen in the absence of inflammatory cells in ECOE, but a thorough evaluation of samples for purulent material is recommended in this situation.

Bacteria are common in purulent infections, in skin samples (e.g. pustules, sinus tracts) the presence of intracellular bacteria is an important point confirming infection (Udenberg, Griffin et al. 2014). However, in ear cytology, where streaming and degeneration are common, the presence of bacteria is considered significant regardless of this. Cytology often reveals mixed populations of bacteria initially, often accompanied by *Malassezia*, but as disease becomes established a single organism usually predominates.

*Malassezia* (usually *M. pachydermatis*) are common in early infections but are often absent in more severe bacterial infections. Although common in ECOE, *Malassezia* can also be associated with purulent exudate and biofilm, despite no overt bacteria on cytology. This is a more challenging situation to treat. In assessing cytology in the recovery phase of a POE, the reappearance of *Malassezia* is often a positive diagnostic indicator (Juhola, et al., 2025). Examples of cytology are given in Table 5. Note the varying levels of degeneration.

## Culture and susceptibility testing

Not all ear disease needs bacteriological testing, particularly in early or acute disease. Where cytology indicates ECOE, antibiotics are not going to be used and culture is rarely beneficial. However, in purulent disease bacteriology ensures the best antibiotic stewardship. The antibiotic susceptibility of cocci is often predictable, but when there is treatment failure culture can still be valuable. Similarly in cases where meticillin-resistant *Staphylococci* are suspected, identifying carriage of this commensal has implications for general management. For rods, antibiotic susceptibility can be variable and identifying the species and testing in chronic cases can explain treatment failure and direct future therapy. Common reasons for culture are shown in Table 4.

Table 4. Common reasons for using culture and sensitivity testing in otitis externa

<b>Rods</b>
<ul style="list-style-type: none"><li>• In most cases when not financially compromised</li><li>• Commonly available antibiotics in ear creams with activity against rods include fluoroquinolones, aminoglycosides and Polymixin B and may be considered as generic choices whilst waiting for results</li></ul>
<b>Cocci</b>
<ul style="list-style-type: none"><li>• Increased risk of MRS<ul style="list-style-type: none"><li>» frequent veterinary interventions</li><li>» multiple antibiotic courses</li><li>» living in a household with another pet with MRS</li></ul></li><li>• Commonly available antibiotics in ear creams with activity against cocci include fucidic acid, florfenicol, aminoglycosides, and fluoroquinolones. Miconazole has some anti-staphylococcal activity.</li></ul>
<b>Rods and/or cocci</b>
<ul style="list-style-type: none"><li>• Otitis interna (most bacterial causes in dogs and cats originate from otitis externa/media</li><li>• When flushing in the middle ear and may induce otitis interna</li><li>• Treatment failure</li></ul>
Note: Bacteriology results may list antibiotics that will never be useful against the organisms grown e.g. cephalixin for <i>Pseudomonas aeruginosa</i> . The best labs indicate this 'intrinsic resistance' on their reports.

There is a common perception that bacteriology (culture and bacterial antibiotic sensitivity testing [BAST]) are not needed in many cases as the antibiotic in topical medications is at sufficiently high concentration to overcome antibiotic resistance. This is however only true for some resistance mechanisms. For instance, efflux pumps in resistant bacteria may remove all of the antibiotic even at very high dose. Similarly, a genetic change may alter the antibiotic binding site making the concentration irrelevant (e.g. MEC-A). Assumptions such as presuming that rods are all *Pseudomonas* spp. can lead to delays in treatment success and the use of inappropriate antibiotics (Henneveld, Rosychuk et al. 2012, Popa, Iancu et al. 2026). However, it is worth noting that MRS is usually treated effectively with products containing florfenicol or fucidic acid and some labs do not list these antibiotics as standard on BAST reports.

## Inflammatory cells

Neutrophils predominate in ear inflammation. Occasionally eosinophils or macrophages are noted. In the wet environment of an inflamed purulent otitis, neutrophils are rarely healthy and so are

found in various states of degeneration from mildly swollen through to just nuclear streaming. Although in other cytology samples where there is marked degeneration or apparent cell damage, we are very wary of making a diagnosis it would be most unusual to see other cells here and we can use nuclear streaming to confirm a purulent otitis. As previously discussed, the presence of neutrophils is consistent with purulent otitis and is a **key cytological finding**. The cytology decision tree to decide between IOE, ECOE and POE is shown (Fig 2) and cytology is shown in Table 5.

Fig 2. Decision tree for cytology

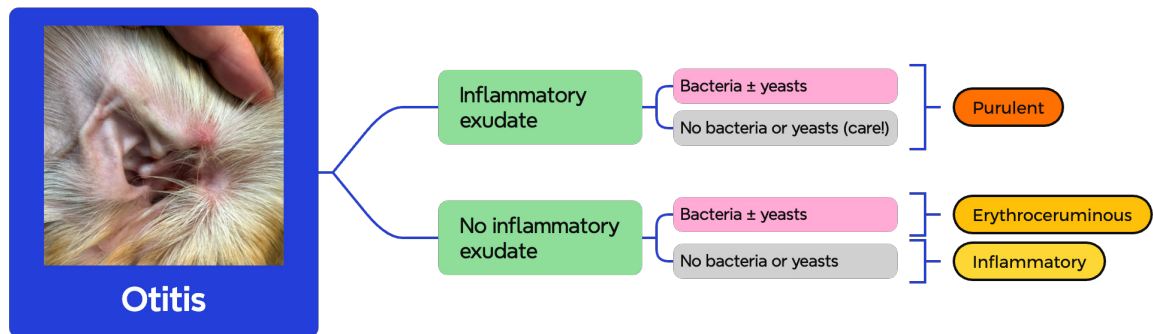
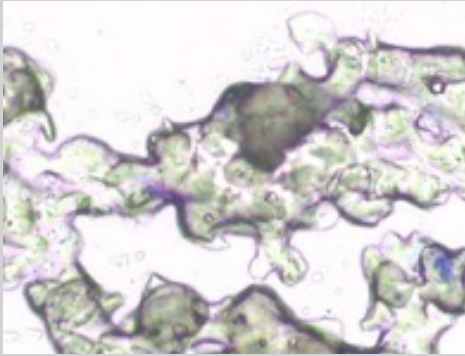
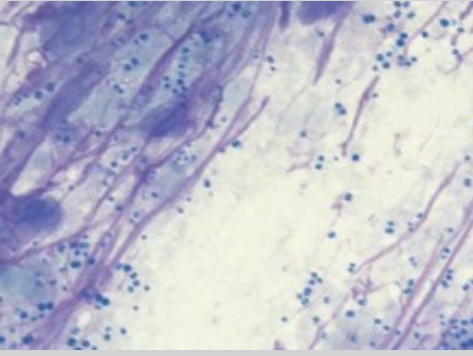
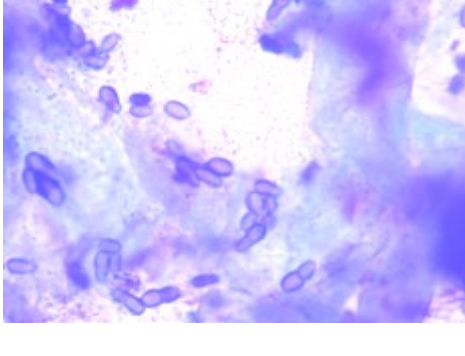
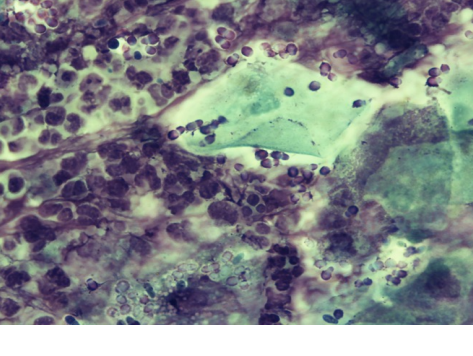


Table 5. Example Cytology cases

	
<p>In a normal ear cytological examination shows minimal microorganisms with squames. These stain variably from pale to purple.</p>	<p>In purulent otitis, bacterial overgrowth is accompanied by neutrophilic exudate. Marked degeneration and nuclear streaming are common.</p>
	
<p>In ECOE otitis, bacteria and/or Malassezia overgrowth is noted. There are no inflammatory cells.</p>	<p>In purulent Malassezia infection, yeasts are often obscured in biofilm and are easily missed</p>



# Otitis treatment strategies: To prescribe or not to prescribe?

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## Treatment

### General principles

Treatment choices should be made based on PSPP. The following steps are needed: 1. Identify and remove/treat the primary cause (and consider potential predisposing factors), 2. Correct the dysbiosis / infection, 3. Reduce swelling, discomfort or pain and identify and treat perpetuating changes and 4. ensure a treatment philosophy aimed at controlling primary disease that seeks to prevent the recurrence of 2° infection and the development of further perpetuating changes. However, whilst it is crucial that you return to dealing with the primary disease and a proactive preventative treatment strategy, infection, inflammation and secondary ear changes are the acute priority in severe disease. Explicit communication to ensure that the owner returns for rechecks and a long-term plan to ensure that 1° disease is treated must not be forgotten.

### ECOE

In erythroceruminous otitis, microbial dysbiosis results from inflammation due to the underlying primary disease. Where possible (e.g. *Otodectes cyanotis* and foreign bodies) these should be removed and the ear treated to clinical cure. In many cases hypersensitivity disease (e.g. canine atopic dermatitis) may need to be managed systemically, but local treatment is often essential to managing the otitis. The choice of a cleaner is decided by the nature and quantity of discharge and this is combined with a topical steroid to reduce the canal inflammation which allowed the overgrowth. Combining cleaner and steroid in homemade mixtures often delivers inadequate doses of steroid and the larger volumes involved risks higher exposure of the carer to steroid containing fluids.

In ECOE maintenance cleaners with both ceruminolytic and antiseptic properties are very useful. Products such as Clorexyderm Oto, Cleanaural, Douxo S3 Care and Epiotic fulfil these criteria and many good alternatives exist. For the steroid component two products are licenced in the United Kingdom. Hydrocortisone aceponate spray (Cortotic®; Virbac) is used for 7-14 days with a success rate in over 90% of dogs (Rigaut, Briantais et al. 2024), this being the same as in the control group treated with a polypharmacy product containing miconazole, prednisolone acetate and polymixin B demonstrating the benefit of a non-antibiotic approach. Hydrocortisone aceponate spray has a very low systemic impact despite steroid potency. Alternatively, triamcinolone acetonide with added

salicylic acid (Recicort®; Dechra) is often used, again for up to 14 days. Not using antimicrobials appears illogical to some in the face of marked overgrowth, but this approach has proved clinically successful and has the potential to significantly reduce the use of antibiotics. Barrier function and dysbiosis is driven by inflammation, and removing inflammation before there is a purulent exudate can prevent the progression of the dysbiosis and reverse the clinical signs. Supporting this, barrier function in an *in vitro* model of atopic skin was recently shown to be largely dependent on pro-inflammatory cytokines (Combarros, Brahma et al. 2025).

The author has seen some cases of ECOE that have not responded to multiple courses of topical steroid treatment. In these cases, chronic unsuccessful treatment has sometimes result in increased difficulty in administering treatments, and a short course of appropriate polypharmacy ear treatment has then been successful. Thus, knowing that if the treatment has been used correctly and you are seeing a poor response, re-evaluation and the use of the lowest possible antibiotic category polypharmacy product is likely appropriate.

## POE

Treatment should be guided by the history, clinical examination and cytology. In most cases there is an obvious exudate and this needs to be removed. Using an aqueous antiseptic product is most useful as these are considered largely otosafe and do not sting when ulceration and erosion are present and may also disrupt biofilm. Steroids remove swelling and reduce pain and pruritus and are an essential part of therapy. Steroids are present in all polypharmacy agents with marketing authorisation for otitis externa in the UK. Most polypharmacy agents have the potential to treat the three most important pathogens (*Malassezia*, *Staphylococcus pseudintermedius* and *Pseudomonas*), but steroid potency is variable. However, products with florfenicol are only suitable for *Malassezia* and cocci and products with terbinafine without antibiotic are only suitable for yeast infection.

As with all modern veterinary treatment, antibiotic stewardship is a key factor in deciding on a chosen therapy. It is important to consider that stewardship does not just involve withholding antibiotics but also making optimum use them when they are needed. When treating otitis externa and many cases of otitis media, topical antiseptics and antimicrobials are used and systemic drugs can be avoided. Local antibiotics can be used at high concentration, unachievable through systemic administration, and have less effect on the skin and gut microbiome and the development of resistance. Moreover systemic fluoroquinolones given before TECABO surgery were found to be at largely inadequate concentrations in ear tissue, risking the rapid development of resistance and treatment failure (Cole, Papich et al. 2009).

## Factors important in the success of therapy.

One of the most crucial things for therapy is owner and dog compliance. Here communication of

the reasons for therapy and the practical ways of delivering treatments as well as ensuring psychological safety to allow the owner to discuss their fears and difficulties with treatment are key. In this context pain relief should be used to make treatment as easy as possible. Longer acting products can be very useful in providing an immediate intervention, but are not suitable for long term proactive use.

Excessive discharge and biofilm may prevent antibiotics from reaching their target and must be removed. Cleaning dilutes the organism and inflammatory mediators and often has antiseptic effects. Polymyxin B and gentamicin-based products may show reduced activity in the presence of pus making ear cleaning an essential part of treatment. In more severe cases, flushing under general anaesthetic (GA) can remove the discharge, allowing much better visualisation of the canal walls and the tympanic membrane. Flushing under GA will benefit many cases, likely reducing the time to remission and bacterial exposure to antibiotics (reducing the risk of acquired resistance), but cost can be an impediment and contextualised care considering the whole situation is essential.

Insufficient anti-inflammatory (steroid) medication is a problem in some cases. Although counterintuitive as we consider glucocorticoids to be immunosuppressive, steroids are an essential part of treatment in the inflamed ear. It is interesting to note that as polypharmacy ear drops have been developed over the last fifty years the steroid potency has increased. When using products containing less potent steroids adding or increasing systemic prednisolone can be useful, but risks increased adverse effects. Products containing mometasone, dexamethasone and hydrocortisone aceponate have high potency whereas prednisolone is at the lower end.

The choice of antibiotic should be grounded in knowledge of the bacterial susceptibility of the 'wild' type of the bacteria noted on cytology. For cocci, particularly the *Staphylococci*, many agents are suitable including fucidic acid, florfenicol, aminoglycosides and potentially fluoroquinolones, but these should be avoided. One product, containing miconazole, polymyxin B and prednisolone has activity against Staphylococci despite predicted intrinsic resistance for polymyxin B by virtue of the antibacterial effect of topical miconazole (Pietschmann, Hoffmann et al. 2009, Pietschmann, Meyer et al. 2013). However, using this class B antibiotic without a specific target should be avoided. For the rods, when *Pseudomonas aeruginosa* is implicated, three classes of antibiotics available in ear ointments are suitable, these are polymyxin B, fluoroquinolones and aminoglycosides. Of these the aminoglycosides have the lowest antibiotic category.

Although most commercial ear products have a broad antibacterial spectrum, Neptra and Osrnia, containing florfenicol are not suitable in rod-based infections and there are currently no depot ear creams with marketing authorisation to treat rods. Cytology should be used to ensure the correct treatment.

Ototoxicity is a common concern, particularly as assessing the tympanic membrane is so difficult in the affected ear and this can be a hurdle to selecting treatment. Deafness, damage to the facial nerve, sympathetic nerves (often seen as Horner's syndrome) and vestibular disease may be seen. The chances of drum rupture in a relatively acute ECOE with *Malassezia* overgrowth is small but grows with increasing numbers of bacteria and in chronic *Pseudomonas* otitis tympanic membrane rupture is more common, although not inevitable. The practicalities are such that a risk-benefit assessment is made based on the history and clinical examination including cytology. This should be communicated to the owner recognising the need for therapy and the potentially ototoxic effects of ear inflammation and bacterial toxins in the untreated animal. **Logistically this means written consent.** Products known to have an increased risk for ototoxicity include gentamicin, polymyxin B, ticarcillin, imipenem, propylene glycol and higher doses of chlorhexidine (Oishi, Talaska et al. 2012, Paterson 2018). However, the number of reports of problems is low for the ear creams with marketing authorisation (e.g. Strain, Merchant et al. 1995). Some clinicians use home-made antibiotic solutions as these are considered less toxic than those in oily suspension/solution, but there is little evidence for this benefit in the literature except for gentamicin. Ototoxicity may also arise from systemic drugs used alone or in combination with ear creams. Commonly quoted examples include frusemide, loop diuretics, cis-platin, erythromycin and NSAIDs.

The time to control a severe ear infection is often underestimated and licenced products are commonly recommended for shorter periods than required. An aggressive approach to the introduction of cleaning and using adequate steroids will minimise the time taken, but for more severe POE this may be 3-4 weeks. After this, if there is poor progress, the approach should be reviewed.

## Ear cleaners and flush products

### General principals

There are vast numbers of ear cleaners. For some there is scientific evidence for their use published in peer-reviewed journals, but for many others information is limited. *In vitro* experiments predominate and no cleaner has marketing authorisation as a POM-V at this time. Owners may obtain cleaners from many non-veterinary sources and regardless of their efficacy these are sometimes used excessively to try to prevent or treat otitis and this represents a common predisposing factor. It is useful to recognise some of the common ingredients and their properties to understand what may have been used at home and to choose the best treatments in the practice.

**Ceruminolytics** soften and dissolve cerumen and dried debris in the ear canal. They are of little benefit in ears with a purulent discharge. Some ceruminolytic agents may be ototoxic, but usefully squalene is considered safe in the middle ear. Propylene glycol, lanolin, glycerine, squalene and mineral oils are all used as ceruminolytics.

Table 6. Common cleaners and their uses

Otitis	Type	Comments
<b>POE and inflamed ECOE</b>	<b>Aqueous flush</b> with disinfectant properties. e.g. Otodine (Nextmune), TRIZChlor (Dechra) and Hexaural (Mi-Vet)	Do not sting and have poor ceruminolytic properties. Considered to be Otosafe.
<b>ECOE</b>	<b>Maintenance cleaner</b> e.g. Cleanaural (Dechra), Clorexyderm (Nextmune), Epi-otic (Virbac), Douxo S3 Calm (CEVA)	Combine ceruminolytic and varying levels of antiseptic properties. Some of these agents sting and are not suitable for a very inflamed ear
<b>Severe Wax</b>	<b>Ceruminolytic cleaner</b> with no anti-septic properties containing squalene e.g. Cerumaural (Dechra) and Otoact (Nextmune)	Considered safe in the middle ear. As no antiseptic properties sometimes used with aqueous cleaners (8-12 hours after the cleaner)

**Surfactants** such as sodium ducosate emulsify debris. Such products can be irritating, particularly to the middle ear mucosa.

**Astringents** help prevent maceration by drying the surface of the ear canal. Alcohols, boric and acetic acid are commonly used astringents.

**Antimicrobial agents** are commonly employed in veterinary cleaners. Chloroxylenol (PCMX), TRIZ EDTA with chlorhexidine, boric and acetic acids, alcohol and monosaccharides all have actions to reduce bacteria and yeasts by direct killing or by reduced adherence. Note that TRIZ EDTA potentiates the action of many antimicrobials by changing the permeability of bacterial cell walls. This has a weak direct effect on bacteria but is very helpful when combined in a multimodal treatment plan.

**Biofilm dispersal.** TRIZEDTA and N-acetyl cysteine (NAC) help disperse biofilm. NAC also has direct antibacterial properties (May, Conklin et al. 2016; Walter, Verspohl et al. 2023), but some care is needed as the combination of NAC and some antibiotics has been shown to reduce bacterial killing (Landini, Di Maggio et al. 2016).

Thus cleaners, as well as removing excessive discharge, will physically remove bacteria and yeasts as well as increasing exposure of those organisms to further treatments, but beyond this often have antiseptic and biofilm busting effects. This is an **essential part of preventing antibiotic resistance as having multiple mechanisms of antimicrobial action prevents mutant escape in both static and induced resistant clones**. A summary of cleaner use is shown in Table 6.

**Picking the right cleaner.** It is common to see aqueous cleaners with poor ceruminolytic properties used when a maintenance cleaner would be more appropriate. For maintenance and proactive treatment of ear inflammation, the evidence for cleaners is relatively poor compared to the use of topical steroids and controlling generalised skin disease. A study showing the variation in antimicrobial properties of some cleaners is shown in Table 7 (Swinney, Fazakerley et al. 2008). Additionally, a commonly used commercial cleaner (Otodine®, Nextmune) was shown to have good antimicrobial

activity *in vitro* against a wide number of ear organisms supporting its use in purulent otitis (Guardabassi, Ghibauda et al. 2010).

**Table 7. Antimicrobial properties of selected cleaners**

Product	Active ingredients	Staph*	Pseud*	Malass*
Cleanaural <sup>(Dechra)</sup> pH 6.5	Boric acid, citric acid, isopropanol and propylene glycol	1/32	1/8	1/32
Epiotic <sup>(Virbac)</sup> pH 7.0	Salicylic acid 0.1% PCMX 0.1%, disodium EDTA Docusate sodium & propylene glycol base, Monosaccharides (l-rhamnose, d-galactose, d-mannose)	1/2	1/8	1/8
Malacetic Aural <sup>(Dechra)</sup> pH 4.0	2% acetic acid, 2% boric acid, glycerine, polysorbate triethanolamine	1/2	1/2	1/4
Sancerum <sup>(ScheringPlough)</sup> pH 3.0	Lactic acid 2.5%, Salicylic acid 0.1%, PCMX 0.1%, docusate sodium and propylene glycol base	1/16	1/16	1/8
TRIZ EDTA <sup>(Dechra)</sup> pH 7.9	Tromethamine (TRIZ), edetate disodium dihydrate (EDTA)	No antimicrobial activity - potentiates other agents (Buckley, McEwan et al., 2013)		
Otoclean <sup>(Elanco)</sup> pH 7.6	Salicylic acid 0.22%, Lactic acid 2.6%, Oleic acid 0.264%, Glycerine, Propylene glycol and Polyethylene glycol	1/4	1/4	1/8
TRIZChlor <sup>(Dechra)</sup> pH 8.0	Tromethamine (TRIZ), edetate disodium dihydrate (EDTA) and chlorhexidine 0.15% w/w	1/2	1/16	1/8

\* Minimal strength needed for 100% reduction in *Staphylococcus pseudintermedius* (Staph) *Pseudomonas aeruginosa* (Pseud) and *Malassezia pachydermatis* (Malass). Note: Other manufacturers produce similar products and ingredients may have changed over time since this study.

## Summary

We are fortunate to have large ranges of possible therapeutic and ancillary agents to treat ears. Using clinical and cytological examination supported by culture where necessary, we can avoid the use of antibiotics in many cases and still ensure the maximum chance of success of therapy. An example protocol for *Pseudomonas* otitis is shown in Table 8.

**Table 8. Example Pseudomonas treatment protocol**

Initial Steps	Comment
<p>Mild to moderate cases Clean the ear in the consulting room using saline and an aqueous cleaner:</p> <p>Moderate to severe cases Admit for flush under general anaesthetic Consider pre-treatment with glucocorticoids and analgesia for a few days before this procedure (if appropriate can get culture results in this period)</p>	<p>Clean the ear carefully to avoid canal trauma Inspect for foreign bodies and the integrity of the drum (visual and bubble test) Perform deep cytology sometimes this is different from superficial sampling Flush with warmed saline until clean Provide first treatments in the surgery Ensure good analgesia</p>
Home on	
<p>TRIZ-EDTA with added N-acetyl cysteine once a day TRIZ-EDTA with added 0.15 w/w chlorhexidine once to twice daily (commercial products such as Otodine should be used) Gentamicin* containing ear cream once to twice daily Prednisolone 0.4-0.7 mg /kg SID PO for 5 days and then every other day Paracetamol/Codeine 15mg/kg TID PO for 10 days in the first instance</p> <p>* Category C</p>	<p>Use first, allow to soak for 20-30 minutes before next treatment Use to clean the ear and then allow to soak for 20-30 minutes before next treatment Apply recommended amount and massage to cover the ear In the mildly affected ear, withholding oral steroids may be acceptable, but generally prednisolone is needed Ensure adequate pain relief. Care: owners may underestimate ear pain</p>
Re-examination	
<p>Check and cytology after 7-14 days Maintain therapy until cytological cure</p>	<p>Interval dependent on case Beware: very wet ears often show mild neutrophilic inflammation, but these will be less degenerate than normally seen in the ear (Malassezia may be seen to overgrow in resolving bacterial purulent otitis)</p>

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## WARNING

ALTHOUGH EVERY CARE HAS BEEN TAKEN TO ENSURE THAT THE INFORMATION GIVEN IN THESE NOTES IS ACCURATE, IT IS VITAL THAT THE CLINICIAN CHECKS THE CURRENT DATA SHEET AND OTHER SOURCES OF INFORMATION BEFORE USING ANY MEDICATION

SOME MEDICATIONS DESCRIBED DO NOT HAVE MARKETING AUTHORISATION IN THE UNITED KINGDOM AND SHOULD ONLY BE USED, WHEN APPROPRIATE, AFTER CONSIDERING THE CURRENT VETERINARY MEDICINE REGULATIONS INCLUDING CASCADE.