



Rob Hilton
BVSc(Hons)
MACVSc(Canine
Medicine) CertVD
graduated with first
class honours from
the University of
Melbourne in 1975.
Between 1976 and
2000 he was in mixed
then small animal
practice, gaining a
MACVSc in canine
medicine along the
way. Since 2001 he
has been focusing is
professional activities
on Veterinary
Dermatology, his
long time professional
interest. In 2006 he
successfully completed
all the requirements
and examinations of
the Royal College of
Veterinary Surgeons
Certificate in
Veterinary
Dermatology.
Rob is a member of
the Dermatology
Chapter of the
Australasian College
of Veterinary
Scientists, an associ-
ate member of the
European College
of Veterinary
Dermatology and
an active member
of Vetdermist.
Rob's particular
interests include
chronic pruritus and
immune mediated
skin disease and he
hopes to get his
translation and
update of Noli and
Scarampella's
"Canine and Feline
Dermatology" into
press early next year.
His practice is
restricted to referrals
and consultations
in Veterinary
Dermatology in a
number of locations
around Melbourne.
Rob can be contacted
on (03) 9436-2289,
rob_hilton@yahoo.com
or at the Lort Smith
Animal Hospital on
(03) 9328 3021

Bacterial infections of the skin

"It is wise to assume every case has some form of infection until proven otherwise. Skin scrape and tape every case. Do cytology to find bacteria, yeast, and possible acantholytic cells" (Hlinca, 2004)

How does the skin defend itself?

The skin has a number of defence mechanisms. These include:

- The normal turnover of epidermal cells by the shedding of corneocytes.
- The barrier function of the keratinised stratum corneum.
- The normal hydrolipid layer on surface of the epidermis (produced by glandular secretions and keratinocyte lamellar bodies). It contains antibodies, antibiotic peptides (defensins) and antiseptic fatty acid derivatives.
- The skin immune system actively recognises antigens and mounts a humoral and cell mediated immune response to pathogens.
- The production of antibiotics and inhibitory substances by the normal resident flora of the skin (*Micrococcus* spp, nonpathogenic staphylococci, _haemolytic streptococci, *Propionibacterium*, and *Clostridium* spp.). Note that pathogenic staphylococci (*S. intermedius*, *S. aureus* and *S. schleiferi*) are not members of the normal skin flora, they are simply passengers from mucocutaneous carriage sites and become opportunist pathogens if the normal skin defences are disrupted.

When one or more of these protective mechanisms is disrupted, the balance is tipped in favour of the pathogens.

The processes that predispose to bacterial infection of the skin include:

1. Immunosuppression

- Corticosteroids (iatrogenic/spontaneous Cushings syndrome)
- FIV
- Congenital immune defects (for example, german shepherd pyoderma)
- Systemic disease (diabetes, hypothyroidism)
- Allergic skin disease (A reduced cell mediated immune capacity is recognised in atopic dermatitis)

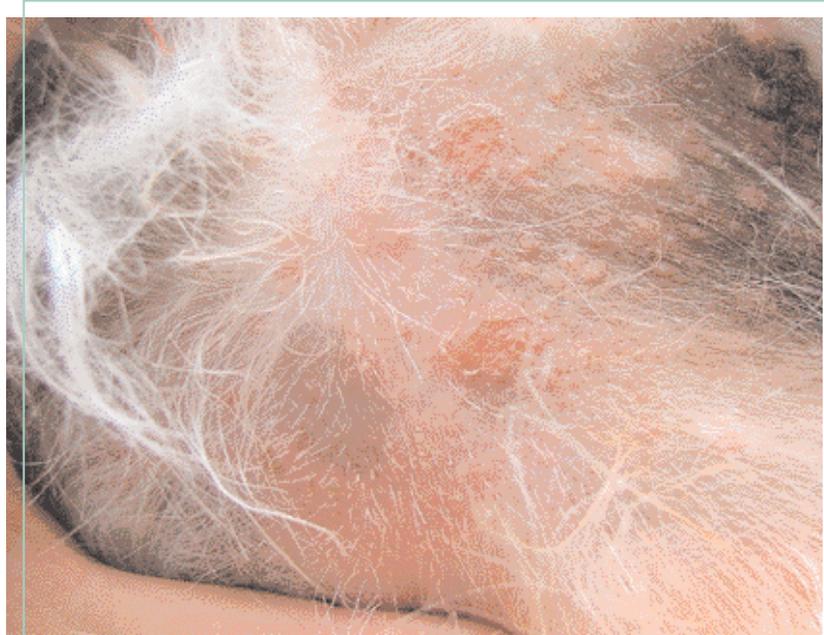


Figure 1 - Superficial pyoderma. Pruritic impetigo showing pustules, papules and epidermal collarettes. The lesions resolved with three weeks of antibiotic therapy

2. Defects in surface barrier

- Allergic reactions (atopic dermatitis, food, flea saliva)
- Self trauma
- Seborrhoea and keritinsation defects
- Loss of surface lipid layer and indigenous microflora by harsh shampoos
- Increased temperature and humidity (folds, axilla, groin)

3. Solar radiation damages keratinocytes, glandular structures and immune cells (especially antigen presenting Langerhans cells) and leads to actinic pyoderma.

4. Follicular diseases and plugging

- Demodicosis
- Dermatophytosis
- Follicular dysplasias (for example, colour dilution alopecia)
- Sebaceous adenitis
- Feline acne

Important skin pathogens

Staphylococci are the most common cutaneous pathogens of our domestic carnivores. Oral bacteria (streptococci, *Pasteurella* spp and oral anaerobes) are important in cats and those infections initiated by licking. Gram negative bacteria (*Pseudomonas*, *Proteus* or *E. coli*) are sometimes isolated, particularly in the feet and ears. Atypical infections such as Mycobacteria, *Nocardia* or *Actinomyces* are sometimes acquired (often by immunosuppressed individuals) from the soil or the bites of rodents (*M. lepraemurium* and others).

In the dog, *Staphylococcus intermedius* is by far the most common pathogen. Increasingly infections with *S. aureus* and *S. schleiferi* are being recognized. Pathogenic strains of *S. intermedius* are carried by dogs in the nasal cavity and other mucocutaneous junctions. It is alarming that human hospital strains of *S. aureus* can be isolated from dogs both from clinical infections and carriers. Transmission from hospitals to human nasal carriers and then to dogs has been shown. The reverse is more than likely also.

Attachment to corneocytes is a vital pathogenic mechanism for staphylococci to initiate an infection. Increased staphylococcal attachment is indeed one of the major reasons for secondary infections in allergic skin disease. *S. intermedius* has a much lower capacity to attach to human skin. The reverse is true for human stains of *S. aureus*. This may explain the relatively low rates of canine-human cross infection.

Staphylococcal resistance to _lactamase stable antibiotics (cephalosporins, methicillin and amoxicillin/clavulanate) is mediated by a gene called Mec-A which codes for penicillin binding protein 2a. Methicillin resistance is often associated with MULTIPLE drug resistance though concurrent horizontal trans-



Figure 2 - Superficial pyoderma in a long haired dog. Large spreading epidermal collarettes obscured by coat and scale

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Figure 3 - Superficial pyoderma in a Dalmatian manifesting as "bronzing syndrome"



Figure 4 - Folliculitis in a short haired dog. Note "moth eaten" appearance of coat

mission of resistance genes. There is still a high level of sensitivity to cephalexin and other beta-lactamase stable antibiotics in *S. intermedius* BUT a high proportion of *S. intermedius* strains have Mec-A like gene and express some PBP-2a. In the U.S., a recent paper detected rates of methicillin resistance in dog and cat staphylococci of *S. aureus* 35%, *S. intermedius* 17%, and *S. schleiferi* 40%. In Australia, we do not appear to have reached this level of resistance with respect to *S. intermedius* but we can expect an increasing rate of resistance in an organism we often assume to be sensitive to cephalexin.

Morris et al, 2006 state "*Methicillin resistant staphylococci may pose a significant risk to animal and public health. Therefore, to avoid selecting for resistant strains in cases of suspected staphylococcal infection, clinicians should consider culture and susceptibility testing early in the course of treatment*".

How do we class bacterial skin infections?

Cutaneous bacterial infections are classified on the basis of depth of infection. The depth of the infection has implications with respect to cause and therapy.

1. Surface Bacterial Infections: Surface layers of epidermis only

- Pyotraumatic dermatitis (hot spots)
- Intertrigo (fold dermatitis)

2. Superficial Pyoderma: Deeper layers of the epidermis and hair follicles

- Impetigo
- Folliculitis
- Mucocutaneous pyoderma
- Bacterial overgrowth syndrome

3. Deep Pyoderma: Dermis +/- subcutis

SURFACE BACTERIAL INFECTIONS

Pyotraumatic dermatitis "Hot Spots" are often secondary to allergy and result from the colonization and multiplication of bacteria on skin surface. They are intensely pruritic and may develop into a deeper pyoderma if left untreated.

Signs:

- Exudative surface infection +/- erosions
- Lack of papules, pustules and epidermal collarettes
- Lack of ulceration and furunculosis
- Cytology: Numerous neutrophils and extracellular bacteria

Treatment:

- Identify and treat the primary cause
- Topical treatment may be sufficient in some cases
- Corticosteroids may be used and may shorten course
- 14 days of systemic antibiotics may be sufficient
- Rapid resolution expected if diagnosis correct

It is important to differentiate pyotraumatic dermatitis (hot spots) from pyotraumatic furunculosis (a form of deep pyoderma). In the latter condition, as in all cases of superficial and deep pyoderma, corticosteroids are absolutely contraindicated.

Intertrigo (fold dermatitis) is surface bacterial colonization occurring in moist, warm and humid areas of the body. Friction additionally predisposes infection. The condition is breed or obesity related. Examples include lip fold, vulval fold, face fold etc. Signs include erythema, erosions and a foul smelling exudate. Cleaning and antibacterial therapy provide temporary relief. Resolution is mostly dependent on correction of the anatomical defect.

SUPERFICIAL PYODERMA

Superficial pyoderma is infection of the deeper layers of the epidermis and/or the hair follicle structures. The infection does not extend beyond the epidermal basement membrane. It is important to recognise because corticosteroids are contraindicated in its treatment despite the presence of pruritus. Think of corticosteroids as an immunosuppressant with an antipruritic side effect.

Superficial pyoderma – impetigo

Impetigo is infection of epidermis, with papules and pustules which may rupture to form epidermal collarettes. The pustules are not related to hair follicles. Juvenile impetigo is commonly found on the ventral abdomen of young dogs. A severe form, impetigo bullosa, is recognized in immunocompromised individuals. Pruritus is VARIABLE (intense to low).

Superficial pyoderma – folliculitis

Folliculitis is defined as infection of hair follicle. The primary lesions are follicle-based papules or pustules. These may rupture and expand as epidermal collarettes. Folliculitis is usually pruritic. If the infection breaks out of the hair follicle, it is called a furuncle and is then classed as deep pyoderma. In the cat, the appearance of pustules is a rare event.

Folliculitis has variable presentations in different breeds.

■ **Long haired breeds.** Lesions may be hidden by the coat and require clipping to demonstrate. The dominant lesions are epidermal collarettes, alopecia and scale.

■ **Short haired breeds.** Moth eaten appearance to coat. Small follicular papules appearing as "stinging nettle pricks"

■ **Dalmatians.** Bronzing discolouration of the coat. This disease was formerly call "Dalmatian bronzing syndrome" until its true nature was discovered.

■ **Bull dogs** may present with areas of hyperkeratotic plaque.

Cytology of a pricked pustule or from the scale at the leading edge of a collarette reveals mainly neutrophils. The neutrophils typically appear degenerate (swollen nuclei, disrupted cells and nuclear streaking). Intracellular bacteria are typical but not always seen. The appearance of intact neutrophils and acantholytic keratinocytes (large round basophilic cells of the stratum spinosum cleaved apart by auto-antibodies) should alert the clinician to the possibility of pemphigus foliaceus.

The principal differential diagnosis of papules, pustules and collarettes:

- Immune mediated disease (esp. pemphigus foliaceus)
- Parasites (*Sarcoptes*, *Demodex*, fleas etc)
- Allergic disease (especially food)
- Dermatophytosis
- Drug eruptions
- Erythema multiforme

Superficial pyoderma – mucocutaneous pyoderma

Mucocutaneous pyoderma is characterised by erosions crusts and fissures involving the lips and other mucocutaneous junctions (nose, eyelids, vulva, prepuce and anus). There may be hyperpigmentation or loss of pigmentation in chronic cases. German shepherds are predisposed.

Mucocutaneous pyoderma of the nose may be clinically and histologically indistinguishable from cutaneous "discoid" lupus. Diagnosis depends on 3 week antibiotic response trial.

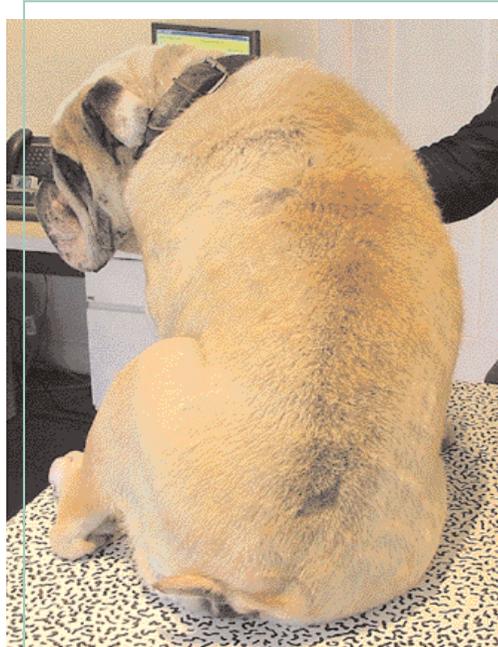


Figure 5 - Folliculitis in a Bulldog with hypothyroidism. Note focal areas of hyperkeratotic plaque



Figure 6 - Generalised epidermal collarettes with crusts and scattered pustules and papules. This presentation is consistent with either superficial pyoderma or pemphigus foliaceus. The diagnosis is based on cytology and histopathology

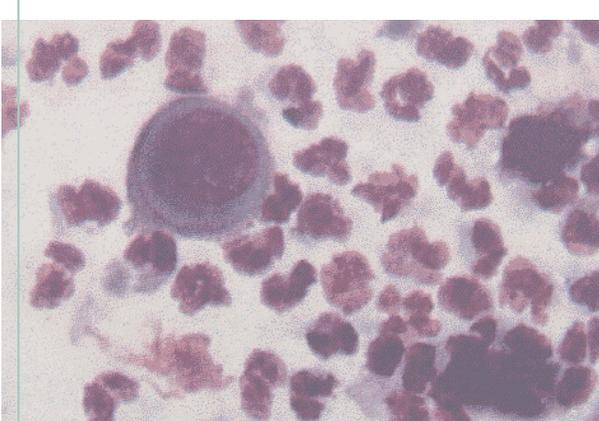


Figure 7 - Acantholytic keratinocytes (large round basophilic cells of the stratum spinosum) from a pustule in a case of pemphigus foliaceus

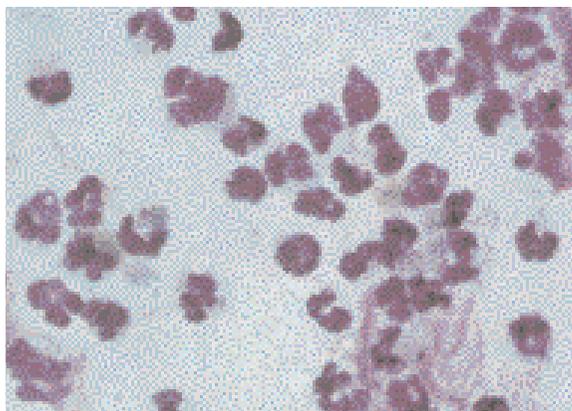


Figure 8 - Cytology of a pustule showing intracellular cocci

The principal differential diagnoses of mucocutaneous pyoderma are:

- Immune mediated disease (discoid lupus, pemphigus and bullous pemphigoid complex or drug reactions)
- Chemical burns
- Zinc responsive dermatosis (nordic breeds, bull terrier)
- Hepatocutaneous syndrome (Metabolic epidermal necrolysis)

Superficial pyoderma – bacterial overgrowth syndrome

Bacterial overgrowth syndrome is a recently characterized disease resulting from the overgrowth of *Staphylococcus intermedius* over the body surface. The syndrome may be secondary to allergy may be from other undetermined primary causes.

Clinical signs:

- Marked pruritus
- Greasy seborrhoea with offensive odour
- Erythema, lichenification, hyperpigmentation, excoriations and alopecia involving principally the axillae, inguinal and ventral aspect of the body
- NO papules, pustules, epidermal collarettes or crusts



Figure 9 - Deep and superficial pyoderma secondary to solar damage (actinic pyoderma)

Tape cytology reveals numerous cocci. The condition responds to systemic antibiotics. The differential diagnosis includes:

- Malassezia infection (may also be present in some cases)
- Chronic allergic dermatitis
- Keratinisation defects (for example, cocker spaniel or west highland whites)
- Sex hormone dermatosis

DEEP PYODERMA

Deep pyoderma may result from extension of superficial pyoderma into dermis and subcutis or direct inoculation into dermis.

Clinical signs:

- Fistulas
- Ulcers
- Furunculosis
- Haemorrhagic bullae
- Sometimes cellulitis and/or systemic signs

Deep pyoderma can be refractory to treatment because of sequestration of bacteria in denatured hair keratin, necrotic fat and within phagocytic cells. Several clinical syndromes are recognised as distinct entities under the subgroup of deep pyoderma.

German Shepherd pyoderma is a reoccurring deep pyoderma associated with a T-cell immunological defect.

Many cases of lick granuloma have an accompanying deep pyoderma. Corticosteroid therapy (symptomatic!) should be withheld until the clinician is certain that this has been resolved.

Pyotraumatic folliculitis and furunculosis can easily be confused with pyotraumatic dermatitis. This condition is a deep pyoderma and corticosteroids are absolutely contraindicated. The lesions most commonly occur on the cheek and neck. Breeds predisposed include the golden retriever, rottweiler and the St Bernard. The disease is very pruritic and present as an area of thick exuding plaque surrounded by satellite lesions of papules and pustules. The onset is acute and the papules and the peripheral papules and pustules will rupture and coalesce to extend the plaque.

Folliculitis and furunculosis of the chin (canine acne) is a chronic disease most frequently seen in younger short-coated dogs (for example, great dane, boxer). The initial lesions are pustules rupture to form ulcers with a haemo-purulent exudate. Feline acne, is a keratinization defect. It is often secondarily infected by oral bacteria (*P. multocida* and α -hemolytic streptococci), resulting in furunculosis.

Interdigital furunculosis is a chronic reoccurring disease that can be very frustrating to treat. The initiating cause is often not clear but can include:

- Conformation and anatomical defects predisposing the paws to continuous trauma.
- Immune mediated disease, for example, the newly characterised syndrome of canine lymphocytic-plasmacytic pododermatitis.
- Allergy.
- Demodicosis.
- Hookworm or Pelodera dermatitis.
- Fungal infections (dermatophytosis, *Malassezia*).
- Infection of sterile pyogranulomas. Short-haired breeds are predisposed (staffies, bulldog, boxer and great dane).
- Hormonal disease (for example, hypothyroidism).

Actinic pyoderma is a deep pyoderma resulting from secondary infection of sun damaged skin. It is seen on the lateral poorly pigmented areas and on the abdomen in those breeds, such as bull terriers, which rejoice in sunbathing.

The differential diagnosis of deep pyoderma includes those disease manifesting as ulceration of the skin and/or draining tracts.

- Deep mycosis /mycobacteria /atypical bacteria
- Immune mediated disease
- Vasculitis
- Sterile panniculitis
- Autoimmune disease
- Drug eruptions
- Severe erythema multiforme
- Toxic epidermal necrolysis

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- Neoplasia
- Chemical, physical or solar burns
- Juvenile cellulitis

TREATMENT OF SUPERFICIAL AND DEEP PYODERMA- CLINICAL ASPECTS

1. Culture and sensitivity testing should be undertaken in all cases of deep pyoderma as the course of treatment will be long and the probability of finding drug resistant organisms, fungi or atypical bacteria is higher. The samples should be collected deep within the tissue or a tissue core submitted for culture.

2. Culture and sensitivity is indicated in cases of superficial pyoderma where cytology detects rod forms. At present in Australia it is still reasonable to treat superficial pyoderma in which cytology reveals cocci with cephalixin. However, clinicians need to be aware of the trend to increasing drug resistance in animal staphylococci. If a case of superficial pyoderma in which cocci are seen fails to respond to antibiotics, consider either resistant bacteria or the possibility of another disease process (for example, pemphigus foliaceus) as described earlier. With increasing

effect and are indicated in cases of superficial pyoderma with extensive scale.

4. Antibiotic therapy must be carried out for long enough to achieve clinical cure. Superficial pyoderma requires a minimum of three weeks of therapy or 10 days after visible cure (whichever is longer). Deep pyoderma may require months of therapy and needs to be continued until one month after visible cure as bacteria may be sequestered in the deeper layers of the skin. If an infection reoccurs within a few weeks of ceasing infection, this is evidence that the course of treatment was not long enough.

5. Corticosteroids are absolutely contraindicated in all forms of deep and superficial pyoderma, no matter how pruritic the patient is. Kill the bugs = kill the pruritus. Try to kill the itch with corticosteroids = kill the immune system.

6. Always look for the underlying cause. Idiopathic cases of superficial and deep pyoderma occur but they are a minority. Always check for demodicosis.

Systemic antibiotics for the treatment of pyoderma

skin. There is moderate resistance amongst staphylococci and tetracyclines are mildly immunosuppressant.

Amoxicillin clavulanate 13-20mg/kg BID is generally as effective as cephalixin against staphylococci but is more costly. It is useful if oral bacteria (e.g. *Pasteurella* spp) involved, in cats or when indicated by culture and sensitivity.

Fluoroquinolones (enrofloxacin, marbofloxacin, difloxacin, orbifloxacin) display low resistance amongst staphylococci (presently) but rising. They have good activity against gram negative bacteria but poor activity against anaerobes and streptococci. Fluoroquinolones have good penetration into skin and intracellularly. Their activity related to peak blood levels, so don't divide the dose. Associated with retinal degeneration in cats and joint damage in growing dogs. They should be reserved for resistant cases and where indicated by culture and sensitivity. Not all fluoroquinolones are alike!! Kinetics and resistance vary. A ciprofloxacin extrapolation may not be valid to determine enrofloxacin sensitivity. They have a mild anti-inflammatory action so don't stop too soon.

Re-occurring cases

- Has the underlying cause been addressed?



Figure 10 - Pyotraumatic furunculosis in a Golden Retriever. This is deep pyoderma and corticosteroids are contraindicated despite the intense pruritus. Photo courtesy of Dr. Guillermina Manigot



Figure 11 - Progression from folliculitis (left) to deep pyoderma with furunculosis and ulceration with crusts (right). *Demodex canis* was isolated from scrapings. Note the comedones in the bottom of the picture that suggest demodicosis

resistance it may become standard practice to culture all cases of superficial pyoderma.

3. Topical therapy aids cure.

- Mupirocin 2% cream (Bactroban) has excellent action against gram +ve bacteria and penetrates well into granulomatous tissue. It is not effective against *Pseudomonas* spp as mupirocin is an antibiotic produced by this species. Silver sulphadiazine cream (Silvazine) is a good broad spectrum and cost effective antibacterial cream. - Shampoos can be used 2x week. Benzyl peroxide is an excellent antiseptic and is reputed to have follicle flushing effects. It is useful for folliculitis and furunculosis (in dogs only!). It has a significant drying effect on the skin and should always be accompanied with an emollient conditioner/bath oil after rinsing off. The drying effect often limits its use to short term only. Chlorhexidine when used at 2-4 % has excellent action against bacteria and *Malassezia*. It does not have the follicular flushing properties of benzoyl peroxide but is less irritating and better tolerated for long term use. Chlorhexidine binds to keratin for 3-5 days. Chlorhexidine can be used in cats. Shampoos containing 2% sulfur and 2% salicylic acid have a keratolytic, antibacterial and anti-pruritic

First generation cephalosporins (for example, cephalixin). There is low resistance amongst *S. intermedius* in Australia BUT high resistance amongst *S. aureus* or *schleiferi*. Expect resistance to rise! The dose rate of cephalixin for pyoderma is 25mg/kg+ BID. Cephalixin only has moderately good intracellular penetration. About 5% of dogs will vomit on cephalixin. There is currently no evidence, in canine superficial pyoderma caused by cocci, that an initial empirical course (initiated without culture and sensitivity testing) of enrofloxacin or amoxicillin clavulanate has a higher initial success rate than less expensive cephalixin.

Potentiated Sulphonamides have higher rates of resistance amongst staphylococci (30% + from some reports). There is a risk of drug induced keratoconjunctivitis sicca and immune-mediated drug reactions (esp. Doberman).

Lincosamides include lincomycin (20mg/kg BID) and clindamycin (10mg/kg). They are effective against about 75% of strains of staphylococci but resistance can develop quickly so they are suitable for short-term use only. Clindamycin has good activity against anaerobes. They penetrate skin and deep tissue well and reach good intracellular levels. Tetracyclines are bacteriostatic and show poor penetration into

- Was treatment long enough and at the correct dose?
- Are there resistance issues?
- Is there a primary or secondary immune deficit?

Reoccurrence within a few weeks indicates the course of antibiotics was not long enough. Reoccurrence 2-4 times a year best managed by repeated full course of antibiotics and shampoo therapy. More frequently re-occurring cases can be managed by weekend therapy = full doses for 2/7 days using a drug to which resistance develops slowly (cephalexin, enrofloxacin, amoxicillin/clavulanate).

There is evidence that autogenous bacterins are useful in controlling reoccurring pyoderma in cases where an underlying immune deficit is suspected. The bacterin is given 1-2x weekly until effective (10-12 weeks) then monthly for life. Many laboratories won't make autogenous bacterins.

There is anecdotal evidence of the use of type 1 interferons for reoccurring pyoderma, administered by injection or buccal spray. The doses used are lower than the antiviral dose (e.g. human alpha interferon 500-2000 IU/day (dog), 60-120 IU/day in the cat). Interferon can be diluted in saline and frozen.

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Figure 12 - German Shepherd deep pyoderma. T cell immune deficit. Photo courtesy of Dr. Candace Sousa

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Figure 13 - Bacterial overgrowth syndrome in a dog. Lesions show greasy seborrhea and hyperpigmentation involving the ventrum. Photo D. Pin and D.N. Carlotti.

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