



Prevalence of methicillin-resistant staphylococcal pyoderma in dogs and persistence of colonization after clinical resolution of disease

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INTRODUCTION

Staphylococci, particularly *S. pseudintermedius*, are the main cause of canine pyoderma. The emergence and dissemination of methicillin-resistant *S. pseudintermedius* (MRSP), along with methicillin-resistant *S. aureus* (MRSA) and methicillin-resistant *S. schleiferi coagulans* (MRSScoag), has been rapid and dramatic in many regions.

Methicillin-resistant staphylococci (MRS) can pose clinical challenges because of limited antimicrobial options as well as zoonotic disease concerns that vary by staphylococcal species.

Longitudinal study of dogs with pyoderma is lacking, and is needed to determine the dynamics MRS infection and colonization, including whether resistant staphylococci remain after clinical resolution and whether treatment of methicillin-susceptible infections may select for resistant strains.

OBJECTIVES

- 1) To determine the prevalence MRS infection and colonization in dogs with pyoderma.
- 2) To determine the prevalence of MRS colonization in dogs after clinical resolution of pyoderma.

MATERIALS AND METHODS

173 dogs that were presented to a veterinary dermatology referral practice between Nov 2009 and Dec 2010 and diagnosed with pyoderma based on clinical and cytological findings were enrolled (Figure 1, Table 1).

Swabs were collected from the nares, rectum and skin lesions at presentation and after clinical resolution. Nasal and rectal swabs were also collected twice, 6 weeks apart, from a convenience sample of 41 healthy household pet dogs with no history of dermatological disease or recent antimicrobial exposure.

Swabs were inoculated in staphylococcal enrichment broth containing (per litre) 10 tryptone, 75 g NaCl, 10g mannitol and 2.5 g yeast extract, and incubated at 35C for 24h. Broth was then subcultured onto MRSA Chromogenic agar (BBL CHROMagar MRSA) and mannitol salt agar with 2 ug/ml oxacillin (MSA-OX).

Isolates were presumptively identified based on colony appearance, Gram stain, catalase test, coagulase test and *S. aureus* latex agglutination test (LAT), then confirmed by species-specific PCR (Sasaki et al 2010) and/or *sodA* sequence analysis. Methicillin-resistance was confirmed by PBP2a LAT.

Categorical comparisons were performed using Fisher's exact test or chi-squared test. A P value of ≤ 0.05 was considered significant.

Clinical	Cytological
Papules, pustules, collarettes, scale, with degenerate neutrophils or crust or lichenification	Presence of bacteria at $>5-10/$ oil immersion field associated intracellular bacteria

Table 1: Clinical and cytological inclusion criteria.

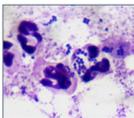


Figure 1: Pyoderma cytology

RESULTS

40% of dogs were diagnosed with MRSP pyoderma (Table 2). 51/70 (73%) dogs with MRSP pyoderma carried MRSP in their nose or rectum vs 8/103 (7.8%) others ($P < 0.0001$).

MRSA was detected in nasal or rectal swabs of 1/3 (33%) dogs with MRSA pyoderma versus 10/170 (5.9%) others ($P = 0.18$).

There was no association between prior antibiotic or corticosteroid use and MRSP, MRSA or MRSScoag infection or colonization (individually or combined), however there was a non-significant ($P = 0.087$) association between prior corticosteroid or cyclosporine use and decreased likelihood of MRSP infection.

There was no significant difference in nasal vs rectal carriage of MRSP, MRSA or MRSS (all $P > 0.14$).

Site	MRSP	MRSA	MRSScoag	P
Skin	70 (40%)	3 (1.7%)	5 (2.9%)	<0.0001
Nose	46 (26%)	9 (5.0%)	5 (2.8%)	<0.0001
Rectum	42 (23%)	3 (1.7%)	4 (2.2%)	<0.0001
Nose and/or rectum	59 (34%)	11 (6.4%)	7 (3.9%)	<0.0001

Table 2: Isolation of MRSP, MRSP and MRSScoag from 173 presented with pyoderma. P value indicates comparison of the prevalence of different organisms at each sample site.

102 dogs were tested 3-15 (mean 6.6) weeks after clinical resolution (Tables 3 and 4). MRSP carriage dramatically increased in dogs without MRSP pyoderma, most of which had received cephalosporin or another beta-lactam.

After successful treatment, there was no difference in prevalence of skin or nasal/rectal MRSP carriage in dogs that initially had MRSP versus non-MRSP pyoderma (all $P > 0.09$).

Site	MRSP	MRSA	MRSScoag	P
Skin	36 (35%)	1 (1%)	2 (2%)	<0.0001
Nose	23 (23%)	1 (1%)	2 (2%)	<0.0001
Rectum	24 (24%)	1 (1%)	3 (3%)	<0.0001
Nose and/or rectum	36 (35%)	1 (1%)	4 (4%)	<0.0001

Table 3: Isolation of MRSP, MRSP and MRSScoag from dogs after clinical resolution of pyoderma (n=102)

	Dogs with MRSP pyoderma initially
Skin	19/42 (45%)
Nose/rectum	20/42 (48%)
	Dogs with non-MRSP pyoderma initially
Skin	17/60 (28%)
Nose/rectum	16/60 (27%)

Table 4: Isolation of MRSP after clinical resolution of MRSP versus non-MRSP pyoderma

No coagulase positive MRS were isolated from control dogs at baseline or acquired during the study period. Control dogs were significantly less likely to carry MRSP than dogs successfully treated with pyoderma, regardless of the etiology (both $P < 0.0001$). They was no significant difference in MRSA or MRSScoag carriage.

RESULTS

Agreement between nasal and rectal swabs for MRSP was moderate ($\kappa = 0.54$). Only 28/59 (47%) colonized dogs were positive at both nasal and rectal sites.

There were significant differences in isolation rates with MRSA Chromogenic agar versus MSA-OX (Table 5).

Organism	MSA-OX	MRSA Chromogenic agar	P
MRSP	207/224 (92%)	67/224 (30%)	<0.0001
MRSA	9/17 (53%)	12/17 (71%)	0.029
MRSScoag	19/20 (95%)	2/20 (10%)	<0.0001

Table 5: Comparison of recovery of MRSP, MRSA and MRSScoag using different media.

Overall, 61 MR-tube coagulase negative staphylococci (MR-CoNS) were identified, including *S. epidermidis* (31%), *S. haemolyticus* (16%), *S. schleiferi schleiferi* (11%) and *S. lugdunensis* (11%). *S. epidermidis* was the most common MR-CoNS at all three body sites.

CONCLUSIONS

The high prevalence of pyoderma caused by MRS, particularly MRSP, was not too surprising in light of the dramatic increase in prevalence noted anecdotally. This highlights the commonness of these important pathogens, at least in referral dermatology in some regions.

The majority of dogs infected with MRSP also harboured the pathogen at other body sites. This may have implications for infection control measures aimed at reducing MRSP contamination or transmission.

The prevalence of MRSP after clinical resolution was striking. Dogs with MRSP pyoderma should be considered likely carriers of MRSP for at least a few weeks after clinical resolution, however the typical duration of carriage is unknown.

The high prevalence of MRSP carriage in dogs that did not initially have MRSP pyoderma was astounding and highly concerning. This might be because of exposure to antimicrobials, but the source of MRSP exposure is unknown. The potential impact of 'routine' antimicrobial use on MRSP carriage rates requires further study.

True colonization is difficult to determine with point prevalence of long timeframe longitudinal studies, so the possibility that some positive samples indicated transient contamination cannot be ruled out.

Multiple body sites need to be sampled for optimal detection of colonization. Additional sites such as the pharynx must also be considered.

The choice of culture media can play a significant role of MRS recovery. Combinations of solid media are required for studies evaluating both MRSP and MRSA.

ACKNOWLEDGEMENTS

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