Genetic determinants of antimicrobial resistance in Streptococcus canis in France
Marisa Haenni, Cécile Hourquet, Estelle Saras, Jean-Yves Madec

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Letter to the Editor

Genetic determinants of antimicrobial resistance in *Streptococcus canis* in France

Sir,

*Streptococcus canis* belongs to the group G β-haemolytic streptococci and is mainly found in pets. *S. canis* is a commensal organism colonising the skin and mucosal surfaces but is also an opportunistic pathogen causing serious invasive diseases such as streptococcal toxic shock syndrome, necrotising fasciitis and septicaemia. Although the zoonotic potential of *S. canis* has been recognised, and despite the existence of shared clones in humans and house pets [1], human infections remain rare. Despite the importance of this pathogen both in veterinary and human medicine, dedicated studies on *S. canis* population diversity and antimicrobial resistance are still scarce [1–4].

In France and worldwide, companion animals, especially dogs, were recurrently shown as carriers of multidrug-resistant bacteria such as extended-spectrum β-lactamase-producing Enterobacteriaceae and meticillin-resistant *Staphylococcus pseudintermedius* (http://www.resapath.anses.fr). These resistance phenotypes might be attributed either to contact with humans or to specific antibiotic treatments. In this context of global high resistance rates, our goal was to assess whether streptococci are also following this trend, by detecting and characterising antimicrobial resistance phenotypes of *S. canis* isolated from pets.

Between January and December 2010, all non-duplicate *S. canis* isolates were collected through the Resapath, the long-term surveillance network for antimicrobial resistance in pathogenic bacteria in France (http://www.resapath.anses.fr) [5]. A total of 112 isolates (104 from dogs and 8 from cats) were collected and their identification was confirmed by Rapid ID 32 Strept (bioMérieux, Marcy l’Etoile, France). These pets mainly suffered from skin infections (*n* = 43; 38.4%) and otitis (*n* = 32; 28.6%). They all originated from different geographical locations throughout France and had no epidemiological link (none came from the same shelter or breeding colony). Pulsed-field gel electrophoresis (PFGE) of all bacteria resistant to tetracyclines or erythromycin (data not shown) using the *Smal* restriction enzyme showed that none of these strains presented an identical pattern and proved an overall high genetic diversity.

Minimum inhibitory concentrations (MICs) to penicillin, erythromycin, clindamycin, tetracycline, enrofloxacin and gentamicin (Table 1) were determined by Etest (bioMérieux) and the results were interpreted according to the guidelines of the Antibiogram Committee of the French Society for Microbiology (http://www.sfm-microbiologie.org). All of the isolates were susceptible to penicillin and displayed only intrinsic low-level resistance to gentamicin. Two isolates were resistant to enrofloxacin and 34 presented an intermediate phenotype, probably reflecting the low activity of fluoroquinolones against streptococci. Thirty-six isolates (36/112; 32.1%) were resistant to tetracyclines owing to the presence of five different tet genes as shown by gene-specific PCR. The tet(M) gene was the most abundant (86%; *n* = 31), followed by tet(O) (44%; *n* = 16), whereas tet(S)/tet(L) (*n* = 1) and tet(K) (*n* = 1) were rare. tet(M), tet(O) and tet(S) were found alone in 15, 1 and 3 isolates, respectively. The remaining isolates presented the following associations: tet(M) + tet(O) in 13 isolates; tet(M) + tet(O) + tet(S) in 2 isolates; and tet(M) + tet(L) + tet(K) in 1 isolate. The 23 isolates classified as intermediate to tetracyclines did not harbour a tet gene. These results are consistent with those of individually owned animals and are comparable with a recent study in Portugal, although the resistance proportion and genetic diversity reported here were slightly higher [1,3].

Erythromycin resistance was detected in 15 isolates (15/112; 13.4%), which were all also tetracycline-resistant. Eleven isolates

### Table 1

Minimum inhibitory concentrations (MICs) of six antimicrobials for 112 *Streptococcus canis* isolates.

<table>
<thead>
<tr>
<th>Antimicrobials</th>
<th>≤0.06</th>
<th>0.12</th>
<th>0.25</th>
<th>0.5</th>
<th>1</th>
<th>2</th>
<th>4</th>
<th>8</th>
<th>16</th>
<th>32</th>
<th>64</th>
<th>128</th>
<th>&gt;128</th>
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</thead>
<tbody>
<tr>
<td>Tetracycline</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>49</td>
<td>21</td>
<td>2</td>
<td>2</td>
<td>6</td>
<td>10</td>
<td>8</td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Enrofloxacin</td>
<td>2</td>
<td>74</td>
<td>26</td>
<td>8</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Penicillin</td>
<td>111</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gentamicin</td>
<td>5</td>
<td>19</td>
<td>68</td>
<td>19</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Erythromycin</td>
<td>95</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>Clindamycin</td>
<td>97</td>
<td>1</td>
<td>3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>10</td>
<td></td>
</tr>
</tbody>
</table>

* Light grey shading indicates isolates displaying an intermediate phenotype based on the breakpoints defined by the Antibiogram Committee of the French Society for Microbiology (CA-SPM). Dark grey shading indicates isolates displaying a resistant phenotype based on the breakpoints defined by the CA-SPM.

* For gentamicin, an MIC ≤250 μg/ml is considered as an intrinsic low-level resistant phenotype, whereas an MIC >500 μg/ml indicates the presence of acquired resistance.

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presented a macrolide/lincosamide/streptogramin B (MLS\textsubscript{B}) constitutive phenotype with MICs to erythromycin and clindamycin of \( \geq 256 \, \mu\text{g/mL} \), whereas the remaining four isolates presented an inducible MLS\textsubscript{B} phenotype as shown by disc diffusion with MICs to erythromycin of \( \geq 256 \, \mu\text{g/mL} \) and to clindamycin of \( \leq 0.12 \, \mu\text{g/mL} \). All but one inducible isolate harboured the \textit{erm}(B) gene as shown by gene-specific PCR. The only isolate presenting intermediate resistance to erythromycin was susceptible to clindamycin and did not harbour \textit{erm}(B) or \textit{mef}(A). Altogether, the \textit{mef}(A) gene was not detected.

In France, streptococcal dermatitis in cats and dogs is mainly treated with \( \beta\)-lactams (amoxicillin/clavulanic acid or cephalaxin, which are still consistently active against streptococci) and with tetracyclines and clindamycin. This use may explain the resistance phenotypes observed here. In the case of otitis, topical treatments with gentamicin, neomycin or marbofloxacin are generally prescribed, and acquired resistance towards these classes of antibiotics was virtually absent in this collection. Interestingly, even though tetracycline and clindamycin are only used to treat dermatitis, resistant strains were found more often in isolates from otitis (13/32; 41\%) than from skin infections (14/43; 33\%). This difference might not be significant owing to the small size of the sampling. Nevertheless, this suggests either selection and persistence of resistant strains that further infect another body site or the transmission of resistant strains from one animal to another.

In conclusion, these data show that streptococci infecting pets are still mainly susceptible to antibiotic treatments commonly used in veterinary medicine. This is encouraging and contrasts with what is observed for infections due to staphylococci or Enterobacteriaceae. However, resistance phenotypes are indeed circulating owing to a variety of transmissible genes. Thus, surveillance of \textit{S. canis} is recommended to monitor the emergence and spread of other resistances that may impair antibiotic treatments in the future.

**Ethical approval**

Not required.

**Acknowledgments**

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**References**


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**Conflict of interest**

None declared.