INTRODUCTION

- *S. epidermidis* is a component of human skin flora but can cause nosocomial infection (Table 1).
- 3 multi-resistant global lineages of *S. epidermidis* have been identified, with both rifampicin resistance and raised teicoplanin MICs.²
- Long-term surveillance data for coagulase-negative staphylococci (CoNS) are scarce.
- We reviewed susceptibility data of *S. epidermidis* collected by the BSAC Bacteraemia Surveillance Programme.

<table>
<thead>
<tr>
<th>Association</th>
<th>Clinical Association</th>
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<tbody>
<tr>
<td>Medical devices</td>
<td>Foreign body-related bacteraemia</td>
</tr>
<tr>
<td>Other</td>
<td>Native valve endocarditis</td>
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<td>Infections in neonates</td>
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<td>Infections in neutropenic patients</td>
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TABLE 1. Infections caused by *S. epidermidis*.

METHODS

- The BSAC surveillance has collected CoNS (179-225 p.a.) causing clinically-significant bacteraemia from 22-36 hospitals throughout the UK and Ireland between 2001 and 2017 (Fig.1).³
- Identification was by PCR from 2001-2005 and by MALDI-ToF from 2013; CoNS were not identified to species level between 2006 and 2012.
- MICs were determined centrally by agar dilution; with *mecA* positive.
- MCIs were determined centrally by agar dilution with *mecA* resistance.
- Current EUCAST breakpoints were used.⁴
- *mecA* was sought by PCR.⁵

RESULTS

- Among 3533 CoNS tested, 1698 (48%) were identified to species level.
- 1082/1698, (64%) were *S. epidermidis* with rifampicin MICs >0.25mg/L and/or *mecA* positive.
- 3 multi-resistant global lineages of *S. epidermidis* have been identified, with both rifampicin resistance and raised teicoplanin MICs.²
- Geometric mean teicoplanin MICs were higher among oxacillin-R and rifampicin-R *S. epidermidis* than other groups.
- The rise in teicoplanin MIC is likely to be due to a change in media given the lack of change to teicoplanin MICs for contemporaneous *S. aureus* or control strains.
- Genotypic analysis is required to investigate this phenomenon further.
- Long-term surveillance is crucial to our understanding when unexpected resistance linkages are recognised.

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<thead>
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<tbody>
<tr>
<td>Oxacillin</td>
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<td></td>
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<tr>
<td>S</td>
<td>&lt;0.25mg/L</td>
<td>65</td>
<td>3 (5%)</td>
</tr>
<tr>
<td>S</td>
<td>≥16mg/L</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>R</td>
<td>&lt;0.25mg/L</td>
<td>257</td>
<td>18 (7%)</td>
</tr>
<tr>
<td>R</td>
<td>≥16mg/L</td>
<td>46</td>
<td>12 (26%)</td>
</tr>
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</table>

TABLE 2. Teicoplanin MICs and geometric mean values of *S. epidermidis* categorised by oxacillin and rifampicin susceptibility. *The Table omits 8 isolates with rifampicin MICS between 0.5-8mg/L.

CONCLUSIONS

- Rifampicin resistance (MIC >4mg/L) has increased in all groups of *S. epidermidis* regardless of oxacillin and rifampicin resistance status.
- Nevertheless, teicoplanin MICs were higher among oxacillin-R and rifampicin-R *S. epidermidis* than other groups.
- This may reflect the spread of one or more of the epidemic lineages with this phenotype.
- The rise in teicoplanin MIC is unlikely to be due to a change in media given the lack of change to teicoplanin MICs for contemporaneous *S. aureus* or control strains.
- Genotypic analysis is required to investigate this phenomenon further.
- Long-term surveillance is crucial to our understanding when unexpected resistance linkages are recognised.

ACKNOWLEDGEMENTS

- BSAC is grateful to all of the companies that have sponsored the Programme (current sponsors: MSD and Pfizer);² sentinel laboratories submitting isolates, and staff at the Central Testing Laboratory, PHE, London.
- The BSAC Standing Committee on Resistance Surveillance: Dr M. Allen, Dr D.F.J. Brown, Prof. D.M. Livermore, Dr C. Longshaw, Prof. A. Johnson, Prof. A.P MacGowan and Prof. N. Woodford.

REFERENCES

3) www.bsacsurv.org.uk, incl. sponsor list.
5) http://www.eucast.org/clinical_breakpoints.

TO REQUEST ISOLATES

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