LINKED RIFAMPICIN AND TEICOPLANIN RESISTANCE IN *STAPHYLOCOCCUS EPIDERMIDIS* FROM BACTERAEMIA IN THE UK AND IRELAND

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INTRODUCTION

- *S. epidermidis* is a component of human skin flora but can cause nosocomial infection (Table 1).[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.

Association	Clinical Association
Medical devices	Foreign body-related bacteraemia
	Foreign body local infection
Other	Native valve endocarditis
	Infections in neonates
	Infections in neutropenic patients

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;[4] with rifampicin tested from 2003 and teicoplanin throughout.
- Current EUCAST breakpoints were used.[5]
- *mec*A was sought by PCR.[6]

FIGURE 1
Distribution of participating laboratories throughout the UK and Ireland.

RESULTS

- Among 3533 CoNS tested, 1698 (48%) were identified to species level.
- 1082/1698, (64%) were *S. epidermidis* with rifampicin data: 376 from 2003-2005; 706 from 2013-2017.
- 835/1082 (77%) were oxacillin-resistant (MIC >0.25mg/L) and/or *mec*A positive.
- 825/1082 (76%) were resistant to ≥3 antimicrobial classes.
- Isolates were categorised according to oxacillin and rifampicin status (Table 2).
- Rifampicin MICs were highly stratified; 88% of values ≤0.25 mg/L and 11% ≥16 mg/L (Fig. 2).
- Geometric mean teicoplanin MICs, and the prevalence of resistance (MIC >4 mg/L) rose for all groups (Table 2).
- The highest rate of teicoplanin resistance (57%) was for recent isolates (2013-2017) resistant to oxacillin and rifampicin (Table 2 and Figure 3).
- Geometric mean teicoplanin MICs did not change for contemporaneous *S. aureus*, and there was no evidence of drift in the MICs for control *S. aureus* (data not shown).

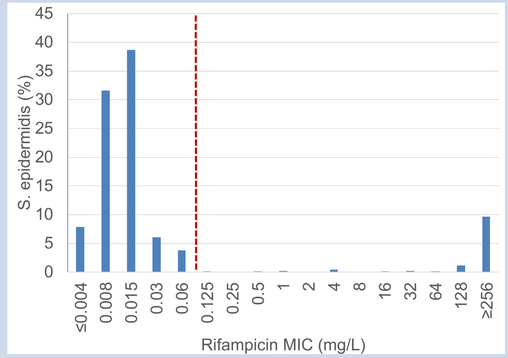

FIGURE 2. Rifampicin MIC distribution among *S. epidermidis* (n=1082). The dashed line indicates the clinical breakpoint.

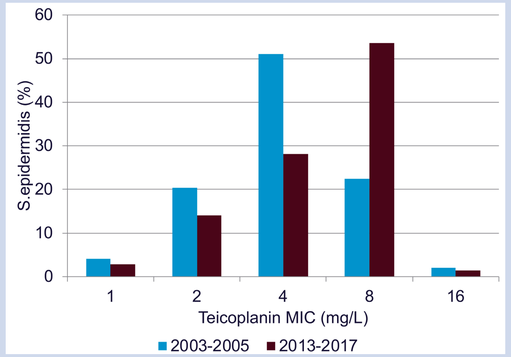

FIGURE 3. Teicoplanin MICs for *S. epidermidis* according to collection period.

Antimicrobial		2003 - 2005		2013 - 2017		Geometric mean teicoplanin MIC	
Oxacillin	Rifampicin	n*	Teicoplanin MIC >4 mg/L	n*	Teicoplanin MIC >4 mg/L	2003-2005	2013-2017
S	≤0.25mg/L	65	3 (5%)	174	31 (18%)	1.76	3.24
S	≥16mg/L	3	0	3	0	Too few	
R	≤0.25mg/L	257	18 (7%)	458	137 (30%)	2.67	4.24
R	≥16mg/L	46	12 (26%)	68	39 (57%)	3.94	5.32

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

- Teicoplanin 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);[3] 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.

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TO REQUEST ISOLATES

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