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Dynamics of acquisition and loss of carriage of *Staphylococcus aureus* strains in the community: The effect of clonal complex☆,☆☆

Ruth R. Miller [a,b,*,h], A. Sarah Walker [a,c,h], Heather Godwin [c,d], Rowena Fung [a,c], Antonina Votintseva [a], Rory Bowden [c,e,f], David Mant [g], Timothy E.A. Peto [a,c], Derrick W. Crook [a,c,i], Kyle Knox [c,g,i]

[a] *Nuffield Department of Clinical Medicine, University of Oxford, John Radcliffe Hospital, Oxford OX3 9DU, United Kingdom*
[b] *UBC School of Population and Public Health, British Columbia Centre for Disease Control, 655 West 12th Avenue, Vancouver, BC V5Z 4R4, Canada*
[c] *National Institute for Health Research Oxford Biomedical Research Centre, John Radcliffe Hospital, Oxford OX3 9DU, United Kingdom*
[d] *Oxford University Hospitals National Health Service Trust, Oxford OX3 9DU, United Kingdom*
[e] *Department of Statistics, University of Oxford, Oxford OX1 3TG, United Kingdom*
[f] *Wellcome Trust Centre for Human Genetics, Oxford OX3 7BN, United Kingdom*
[g] *Department of Primary Care Health Sciences, University of Oxford, Oxford OX1 2ET, United Kingdom*

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KEYWORDS
Staphylococcus aureus;
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Summary *Background: Staphylococcus aureus* nasal carriage increases infection risk. However, few studies have investigated *S. aureus* acquisition/loss over >1 year, and fewer still used molecular typing.

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* Corresponding author. British Columbia Centre for Disease Control, 655 West 12th Avenue, Vancouver, BC V5Z 4R4, Canada. Tel.: +1 604 707 2491; fax: +1 604 707 2516.
E-mail address: Ruth.Miller@bccdc.ca (R.R. Miller).
[h] Ruth Miller and A. Sarah Walker are considered to be equal contributors.
[i] Derrick W. Crook and Kyle Knox are considered to be equal contributors.

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Colonisation;
spa-typing;
Carriage duration

Methods: 1123 adults attending five Oxfordshire general practices had nasal swabs taken. 571 were re-swabbed after one month then every two months for median two years. All *S. aureus* isolates were *spa*-typed. Risk factors were collected from interviews and medical records.
Results: 32% carried *S. aureus* at recruitment (<1% MRSA). Rates of *spa*-type acquisition were similar in participants *S. aureus* positive (1.4%/month) and negative (1.8%/month, $P = 0.13$) at recruitment. Rates were faster in those carrying clonal complex (CC)15 (adjusted (*a*)$P = 0.03$) or CC8 (including USA300) ($aP = 0.001$) at recruitment versus other CCs. 157/274 (57%) participants *S. aureus* positive at recruitment returning ≥12 swabs carried *S. aureus* consistently, of whom 135 carried the same *spa*-type. CC22 (including EMRSA-15) was more prevalent in long-term than intermittent *spa*-type carriers ($aP = 0.03$). Antibiotics transiently reduced carriage, but no other modifiable risk factors were found.
Conclusions: Both transient and longer-term carriage exist; however, the approximately constant rates of *S. aureus* gain and loss suggest that 'never' or truly 'persistent' carriage are rare. Long-term carriage varies by strain, offering new explanations for the success of certain *S. aureus* clones.
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Introduction

Staphylococcus aureus is an important cause of infections in both primary and secondary care. Carriage prevalences of ~30% have been found consistently in studies performed over six decades,[1] with the anterior nares the primary site of colonisation.[1–3] Nasal carriers are at greater risk of infection than non-carriers[4–7] and the carried and invasive strains are indistinguishable in ~80% of cases.[5,8] Non-carriers of *S. aureus* have a higher mortality following *S. aureus* bacteraemia suggesting recent *S. aureus* acquisition around the time of infection is associated with poorer subsequent outcome.[5]

The dynamic nature of *S. aureus* carriage creates complexity for cross-sectional and longitudinal studies, with people acquiring and losing all genotypes of *S. aureus* (the species level) and also acquiring and losing different genotypes within *S. aureus*.[9] For example, one study found multiple genotypes were present in 7% of carriage samples.[10]

Rather than considering *S. aureus* loss and acquisition as separate events, studies have almost universally combined both these aspects and classified individuals as "persistent", "intermittent" or "non" carriers. "Persistence" has most commonly been defined on the basis of (i) >80% positivity of 10–12 swabs taken weekly over ~3 months (not considering strain-type)[11] or (ii) two positive cultures one week apart, since this had good performance for predicting persistent carriage defined by definition (i) in one study.[12] Human polymorphisms associated with "persistent" carriage using definition (ii) have been identified,[13] but bacterial factors have not, to date, been associated with different carriage types. Very long-term carriage and strain switching undoubtedly occur; for example 12/17 "persistent" *S. aureus* carriers according to definition (i) carried *S. aureus* on a single swab taken eight years later, but only three carried highly similar *S. aureus* strains.[11] However, few studies appear to have repeatedly sampled individuals over intermediate periods of >1 years,[14,15] or systematically investigated carried genotypes over these timescales. The rates of acquisition and median carriage duration of newly acquired strains, and the rates of loss of individual strains present in an initial sample with unknown acquisition date, have also rarely been described outside the specific setting of methicillin-resistant strains in hospitalised patients.[16–18] Longer-term follow-up might further support experimental studies which found no distinction between non- and intermittent carriers defined following definition (i) in terms of rates of loss of carriage of a nasal inoculum.[19]

Here we investigate *S. aureus* nasal carriage in individuals from primary care, swabbed bi-monthly for up to 36 months. We *spa*-typed all *S. aureus* isolates to identify acquisition and loss that would be unrecognised at the species level. Our primary objective was to describe the dynamics of *S. aureus* carriage (loss, gain) in the general population, and to investigate potential risk factors, in particular the contribution from particular *spa*-types.

Methods

Study population

Eligible participants were consecutive adults aged ≥16 years attending one of five Oxfordshire general practices (each a group of family doctors) in the Thames Valley Primary Care Research Partnership (all in the catchment area for the Oxford University Hospitals (OUH) NHS Trust). All participants provided written informed consent. 200 participants were recruited from each general practice sequentially over December 2008–December 2009, in age/sex strata approximately representing the UK population. Recruitment was completed in each practice before starting in the next. To increase numbers of younger participants, students registering at one practice were recruited during the University Freshers' week. For the first four general practices, we invited only those participants whose recruitment swab grew *S. aureus* to continue longitudinal follow-up. All participants from the last practice and all students were invited to continue longitudinal follow-up. Assuming 35% participants were *S.*

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