

## Retraction

FOR OUR REPORT "A LINK BETWEEN VIRULENCE and ecological abundance in natural populations of *Staphylococcus aureus*" (1), we conducted a case-control study of severe *S. aureus* disease. Using multilocus sequence typing (MLST), we compared the genetic population structure of isolates recovered from patients with severe disease with that of isolates associated with asymptomatic nasal carriage. We found that (i) isolates fell into a number of clonal complexes of related genotypes, (ii) the most commonly carried clones were disproportionately more likely to cause disease, and (iii) each clonal complex diver-

sified through recombination from the putative ancestral genotype, a process associated with a reduction in the proportion of isolates derived from the patients with disease.

Since these results were published, we have extended our use of MLST to study the genetic population structure of other similar populations of carried and disease-causing *S. aureus* and have found a lower degree of diversity, particularly in the carried populations. Therefore, we revisited the results of the original case-control study, and after repeating much of the experimental work, we found that significant errors had occurred in the earlier MLST. These errors, involving ~0.1% of nucleotides and amplified by the

process of allele and sequence type assignment, occurred disproportionately in the carried organisms. Although in the revised population structure, isolates still segregate into clonal complexes (Fig. 1), and the most abundant carried clones remain the most common causes of disease, there are no significant differences in population structure between the carried and disease-

causing populations (Table 1). Furthermore, diversification of clonal complexes from the ancestral genotype is mainly through mutation, not recombination (2). Therefore, the revised results no longer support the contention that ecologically abundant clones are disproportionately likely to cause disease.

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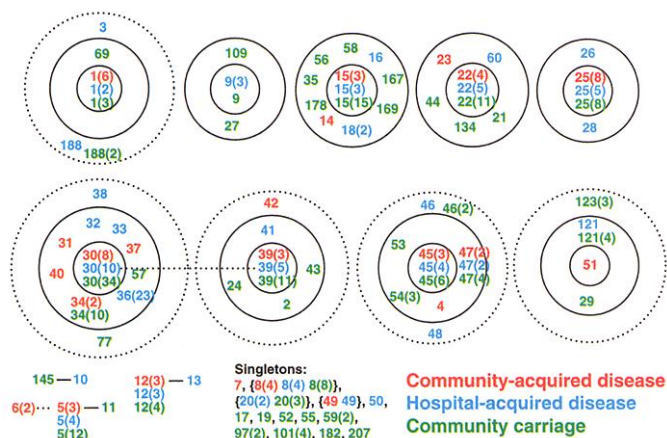
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### References and Notes

1. N. P. J. Day et al., *Science* 292, 114 (2001).
2. \_\_\_\_\_, data not shown.
3. For further details, see the BURST readme file at <http://burst.mlst.net>



**Fig. 1.** Diagrammatic representation of clonal complexes. Each number represents a MLST sequence type (ST). Where a ST is represented by multiple isolates, the number of isolates with that ST are shown in parentheses. Green numbers denote nasal carriage isolates, red numbers community-acquired invasive disease isolates, and blue numbers hospital-acquired disease isolates. No inferences are made concerning the relations between clonal complexes. The central circle of each clonal complex contains the ancestral clone of that clonal complex. Single-locus variants (SLVs) of an ancestral clone lie within the next (solid line) concentric circle, and double-locus variants lie within the outer (dotted line) circle. A solid straight line between two STs denotes a single-locus difference between them, a dotted straight line a double-locus difference. Three small groups of related STs where the ancestral genotype cannot be predicted are also shown. Singletons are isolates having STs that differ from those of all other genotypes at more than two loci. In one clonal complex, some SLVs were assigned as secondary ancestral clones on the basis that they differed by a single locus from at least two other genotypes that had not already been assigned as SLVs. The secondary ancestral clone and associated clonal variants were treated as primary ancestral clones in the analysis and were assigned in rank order according to the number of SLVs they define (3). The clonal complexes are named according to the ST of the primary ancestral genotype, but with the prefix "CC" (for clonal complex). The sequence types of the isolates being sequenced at the Sanger Centre [ST1 (MSSA) and ST36 (EMRSA-16)] remain unchanged.

process of allele and sequence type assignment, occurred disproportionately in the carried organisms.

Although in the revised population structure, isolates still segregate into clonal complexes (Fig. 1), and the most abundant carried clones remain the most common causes of disease, there are no significant differences in population structure between the carried and disease-

**Table 1.** Distribution of disease-causing and nasal carriage isolates within clonal complexes (see Fig. 1). A 3 × 2 Fisher's exact test comparing the distribution within the ancestral clones, single-locus variants, and double-locus variants of community-acquired disease and nasal carriage isolates is not significant ( $P = 0.35$ ).

Position within clonal complex	Nasal carriage isolates, n (%)	Community-acquired disease isolates, n (%)	Hospital-acquired disease isolates, n (%)
Ancestral clone	89 (50)	36 (59)	37 (39)
Single-locus variant	39 (22)	10 (16)	35 (37)
Double-locus variant	8 (4)	1 (2)	5 (5)
Singleton (not within a clonal complex)*	43 (24)	14 (23)	17 (18)

\*For the three minor clonal complexes shown in Fig. 1, it is not possible to assign an ancestral clone; these are included in "singletons."