



Genetic View of Virulence and Resistance in Community-Acquired MRSA

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Whole genome sequencing of MW2, a representative community-acquired MRSA (C-MRSA), provided us an opportunity to compare its genome with those of health-care associated MRSA (H-MRSA) strains represented by N315 and Mu50. By analyzing a total of seven genome sequences available in the database including those of four other *S. aureus* strains, we have confirmed that the chromosome of *S. aureus* is a circular DNA of 2.8 Mbases in size, which is studded at several fixed loci with Genomic Islands (Gilands) and Islets (Gilets: small Gilands). Giland/Gilets may be present at or absent from the loci, and are quite varied from strain to strain in the repertoire of genes which they carry; i.e, there are “allotypes” for each of the Giland/Gilets. They carry genes encoding antibiotic resistance and/or the proteins associated with pathogenicity such as toxins and exoenzymes. Some Gilands, however, do not possess any genes apparently involved in the pathogenic potential or drug resistance; thus making the generic term “genomic island” preferable, which is only discriminated from the surrounding chromosome region by the deviated codon usage and/or GC content of the genes that they carry, or with the presence of remnants of genes involved in their mobility. MW2 has a distinct set of superantigen gene cluster on the Giland $\nu Sa\alpha$ from those of H-MRSA strains, and has an additional Giland $\phi Sa2$ that carries genes encoding Panton-Valentine leucocidins, potent toxins lysing the human white blood cells. Presumed high virulence of MW2 does not seem to be determined by the contribution of any of the single genetic determinants but by an overall effect of the genes carried by the combination of allotypes of Giland/Gilets that are specific to MW2. MW2 possesses type-IV staphylococcal cassette chromosome *mec* (SCC*mec*) that, on the contrary to those of H-MRSA strains harboring multiple drug-resistance genes, carries only methicillin-resistance determinant. This explains the multiple drug susceptibility of C-MRSA strains in the world in which type-IV SCC*mec* is the most prevalent Giland. Presence of bacteriocin-producing operon on Giland $\nu Sa\beta$ and absence of any intact insertion sequence (IS) or transposon (Tn) on the chromosome (except for a copy of IS431 on the SCC*mec*), together with the rapid growth capability of MW2, seem to well reflect the biological feature of MRSA in the community.