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Acinetobacter baumannii ATCC 19606 Carries GI*sul2* in a Genomic Island Located in the Chromosome

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During the course of routine antibiotic resistance testing, we noticed that *Acinetobacter baumannii* ATCC 19606, which was isolated prior to 1948 (1) and is often used in genetic studies, is resistant to the sulfonamide compound sulfamethoxazole (MIC, >128 mg/liter). Three groups have sequenced the genome of ATCC 19606 (GenBank accession numbers JMRY01000000 [2], ACQB01000000, and APRG01000000). The draft genome in GenBank (accession number APRG01000000) was used to search for a gene (*sul1*, *sul2*, or *sul3*) accounting for the sulfonamide resistance phenotype as it includes longer contigs than the others. The *sul2* sulfonamide resistance gene was found in a 210,382-bp contig (supercont1.1.C1; GenBank accession number APRG01000001). The context of the *sul2* gene was analyzed, and it is located in GI*sul2* (genomic island *sul2*) (Fig. 1b), a 15.5-kbp integrative element (3).

GI*sul2* was reannotated and found to include several genes encoding putative arsenate and arsenite resistance proteins and a toxin-antitoxin, in addition to the resolvase and an integrase noted previously (3) (Fig. 1). However, ATCC 19606 was not significantly resistant to arsenite and arsenate ions. GI*sul2* in ATCC 19606 lacks ISAba1, which is found upstream of *sul2* in *A. baumannii* ATCC 17978 (3). In *A. baumannii* ATCC 17978, GI*sul2* is now known to be located in a large conjugative plasmid, pAB3 (GenBank accession number CP012005). As the MIC for ATCC 17978 is also >128 mg/liter of sulfamethoxazole, the promoter in ISAba1 is not essential for high-level sulfonamide resistance.

Inspection of the sequences on either side of GI*sul2* in ATCC 19606 revealed that it is located within a larger genomic island that has been inserted into the chromosome of ATCC 19606 (Fig. 1b). This genomic island, including GI*sul2*, is 36,157 bp long and is located between two open reading frames with locus identifiers F911_00136 and F911_00184 in the data located at GenBank accession number APRG01000001 (Fig. 1). These two reading frames are adjacent to one another in the genome of many other *A. baumannii* strains, e.g., ATCC 17978-mf and A1 (GenBank accession numbers CP012004 and CP010781, respectively). This genomic island is flanked by 5-bp target site duplication (Fig. 1b) indicative of transposition, and there is a copy of ISAba11 at one end (Fig. 1). Seven fragments (ranging in size from 155 to 3,467 bp) of the 13-kb and 6.5-kb regions flanking GI*sul2* match parts of pXBB1-9 (GenBank accession number CP010351), which is a very large (398,857-bp) plasmid in *Acinetobacter johnsonii* strain XBB1, with DNA identities ranging from 96.4% to 98.7%. Hence, it appears that ISAba11, a 1,101-bp insertion sequence belonging to the IS*701* family that creates 5-bp target site duplications (4), has mobilized a large DNA segment from a plasmid related to pXBB1-9 and inserted it into the chromosome of ATCC 19606. However, it is not possible to infer when GI*sul2* became part

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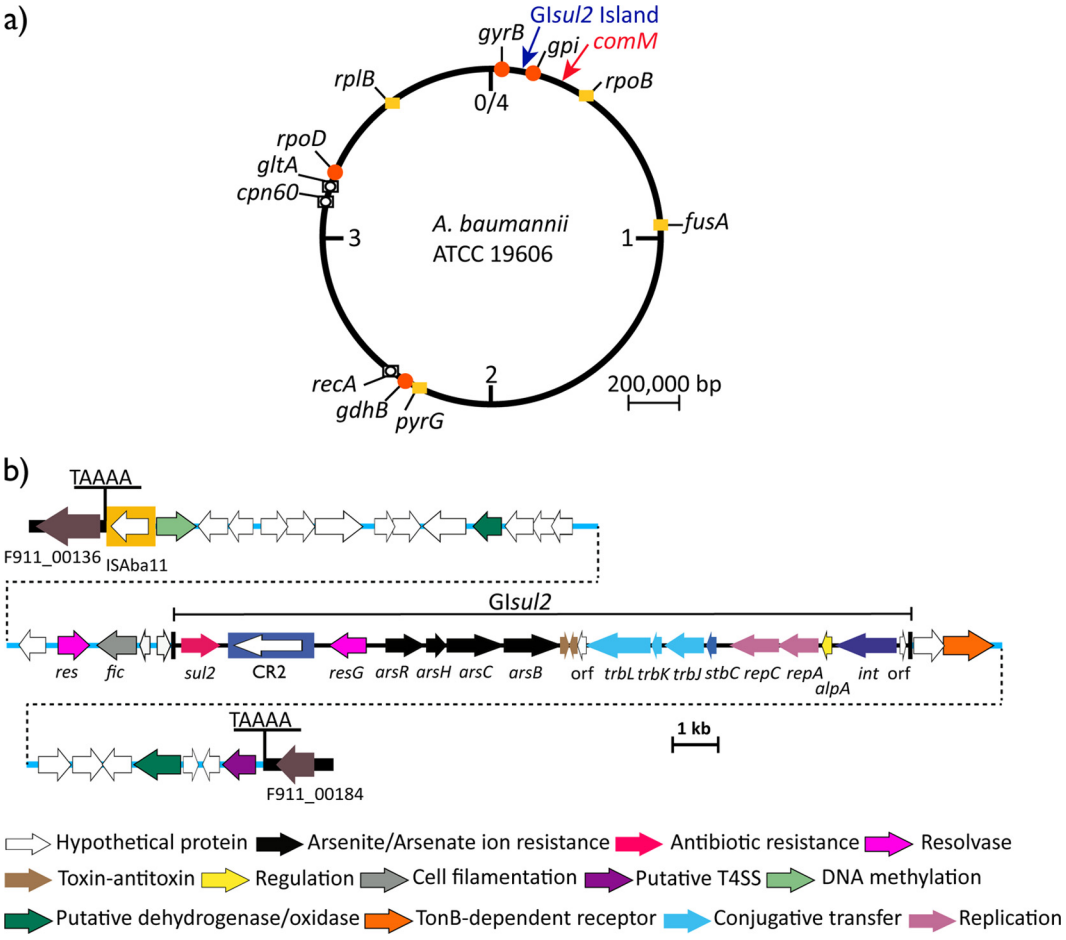

FIG 1 Schematic representation of the GI*sul2* island found in the genome of *A. baumannii* ATCC 19606. (a) Chromosomal location of the GI*sul2* island relative to the multilocus sequence type (MLST) markers used in Oxford schemes (red dots) or in Institut Pasteur schemes (yellow boxes) or both types of schemes (open circles on boxes). (b) Structure of the GI*sul2* island in the chromosome of *A. baumannii* ATCC 19606. Arrows indicate the extent and orientation of genes. The central blue line indicates the backbone of the genomic island, and the thick horizontal black line represents the chromosome, with locus identifiers indicated at the bottom of the panel. The extent of GI*sul2* is also shown. Functions of the genes/orfs are color coded and indicated at the bottom of the panel. Scale bars are also shown. T4SS, type IV secretion system.

of this segment. To date, several IS elements, e.g., ISEcp1, ISKpn23, and ISAba1, have also been shown to mobilize adjacent DNA segments (5–7).

GI*sul2* and segments derived from it have been found in various resistance regions, and it has been suggested that GI*sul2* is the main element responsible for the mobilization of the *sul2* gene (3, 8–10). Here, we described the occurrence of GI*sul2* in a new location leading to sulfonamide resistance in ATCC 19606.

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