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New *emm* (M Protein Gene) Sequences of Group A Streptococci Isolated from Malaysian Patients

To the Editor: We analyzed the M-type–specific *emm* gene sequences of 24 random *Streptococcus pyogenes* isolates from sterile- and nonsterile-site clinical specimens of Malaysian patients. In contrast to isolates in the United States, which rarely have new *emm* sequences, 6 of these 24 Malaysian isolates had new *emm* gene sequences, which suggests a large reservoir of group A streptococci expressing new M-type specificities in Malaysia.

The M protein is a surface-exposed principal virulence factor of group A streptococci (GAS) and a potential vaccine candidate. The hypervariable M-type–specific N-terminal portion of the M molecule extends from the cell wall and evokes protective antibodies. Approximately 75 M antigenic types of GAS are recognized, and several provisional types have been proposed (1). Formulation of a universally effective vaccine is complicated by the M-type–specific nature of protective anti-GAS antibodies, temporal and geographic variations in GAS M-type prevalence (2), and lack of information on GAS M types from areas where rheumatic fever and rheumatic heart disease, sequelae of GAS pharyngitis, are endemic (3). The lack of specific M-typing antisera is also a limiting factor in determining type distribution. Recently, Beall and colleagues (4,5) demonstrated that sequence analysis of the hypervariable portion of the *emm* gene encoding M-type specificity (*emm* typing) was an alternative when M-typing antisera were not available.

Attempts to type selected Malaysian strains of GAS by M protein status have yielded poor results. Fewer than 16% of strains were typable with standard M-typing antisera (6). The existence of new M types in Southeast Asia was suggested as an explanation. To investigate this possibility, we subjected 27 selected strains (6 from blood, 15 from pharyngitis, 3 from pus, and 3 pharyngeal carrier cultures) collected between January 1994 and December 1996 to *emm* typing. Initial isolation, serogrouping, T typing, and determination of opacity factor production were performed in Kuala Lumpur, by standard techniques, commercial media, reagents, and antisera (7). Strains were transported to the Centers for Disease Control and Prevention in Atlanta, Georgia, USA, for *emm* typing, where serogrouping, T typing, and opacity factor determinations were repeated, and *emm* typing was performed (4,5). DNA sequences were subjected to homology searches against all known *emm* sequences by Genetics Computer Group Software, Version 9. (Most sequences in this database were found in strains isolated from patients living in Europe and North America.)

Of the 27 cultures analyzed, 24 were GAS, 2 were group G streptococci, and 1 was nongroupable Streptococcus. Ten of the 24 GAS strains were standard *emm* types *emm*3, *emm*12, *emm*22, *emm*60, and *emm*76 (encoding the classic M types M3, M12, M22, M60, and M76, respectively); 4 were the provisional *emm* types *pt180*, *pt2841*, and *pt5757*; and 3 were previously identified *emm* sequence types *st64/14* and *st2034* (GenBank accession numbers X72932 and U74320, respectively). The *st2034* sequence, originally identified in children from Papua New Guinea, has also been found in Brazil, California, and Hawaii (B. Beall, R. Facklam, unpub. data). One GAS had a sequence previously found in group G streptococci (*emmLG6*, accession number U25741). Finally, 6 were of five new *emm* sequence types discovered in this study

(*st4529*, *st4547*, *st4532*, *st4545*, and *st3018,* with accession numbers AF060368, AF052426, AF077666, AF077668, and AF077669, respectively). The newly found group A *st4545* sequence was more similar to various group G streptococcal *emm* sequences than to known group A *emm* sequences. One group G isolate had a previously found group G 5' *emm* sequence (*stLG6*, accession number U25741). The nongroupable Streptococcus had an *emm* sequence previously associated with group L Streptococcus (Beall and Facklam, unpub. data). These results demonstrate the usefulness of *emm* typing in areas where specific M-typing antisera are not available.

Identifying 6 (25%) of 24 GAS with new *emm* types provides further evidence of new M serotypes of GAS in Malaysia. The deduced amino acid sequences of the mature hypervariable N termini of ST4529, ST4532, ST4547, and ST3018 ranged from 43% to 82% identity to M proteins of known sequence (data not shown). The M nontypability of these isolates suggests unique serologic specificity. ST4547, ST4532, and ST3018 had 70% to 82% identity over the first 55-variable-region amino acids, with their closest matching known M proteins (M70, M27, and M22, respectively), but whether antibodies against any of these proteins would cross-protect against strains expressing these M proteins is unknown. Even though the M70 protein is 70% identical over its first 50 variable N terminal amino acids to the M33 protein, antibodies against the M70 and M33 proteins do not cross-protect, which suggests that no cross-protection would occur. The new deduced M protein with the lowest similarity to any known M protein was ST4529, whose closest match had a 43% identity over the N-terminal 55 residues of the M5 protein. *st4529* likely encodes a new serospecifically unique M protein.

These findings potentially affect vaccine development. Although new *emm* sequences were identified in a survey in the United States (5), the percentage of new strains with new *emm* sequences was much lower (6%) than was found with these Malaysian isolates. *emm* typing of a larger number of strains from rheumatic fever–and rheumatic heart disease–endemic areas is required to deduce amino acid sequences for the development of a suitable M protein–based vaccine.

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Mutant Chemokine Receptor (CCR-5) and Its Relevance to HIV Infection in Arabs

To the Editor: Approximately 10% of HIV-infected patients may remain AIDS-free for a long time; moreover, some persons do not become infected with HIV despite multiple high-risk sexual exposures (1,2). Factors responsible for this relative resistance to infection and disease include cytotoxic T cells, neutralizing antibodies, high concentrations of certain chemokines (e.g.,