Fimbrial Typing of \textit{Bordetella pertussis} Isolates: Agglutination with Polyclonal and Monoclonal Antibodies

On the basis of serotyping 62 strains of \textit{Bordetella pertussis}, Guiso and colleagues (N. Guiso, C. H. Wirsing von König, C. Becker, and H. Hallander, Letter, J. Clin. Microbiol. 39:1684–1685, 2001) concluded that monoclonal and polyclonal reagents showed good concordance but that the source of the monoclonal antibodies needed to be specified. We have conducted a similar study on 209 strains of \textit{B. pertussis} with the objective of identifying a standard set of serotyping reagents.

The polyclonal antisera to agglutinogens 1, 2, and 3 and monoclonal antibodies anti-Fim 2 from hybridoma F2B2G8 (FDA/F2) and anti-Fim 3 from hybridoma C10C2D5 (FDA/F3) were as used in the previous study (Guiso et al., letter, 2001). The monoclonal antibodies to Agg 1 (CAMR anti-Agg 1), Agg 2 (CAMR anti-Agg 2), and Agg 3 (CAMR anti-Agg 3) were provided by A. Robinson, Centre for Applied Microbiology and Research, Porton Down, Salisbury, United Kingdom. The reagents were assessed for agglutinating activity towards \textit{B. pertussis} strains as described previously except that the reactions were performed on glass slides (Guiso et al., letter, 2001). The \textit{B. pertussis} strains were supplied by U. Heininger, Paediatric Diseases Department, University Children’s Hospital, Basel, Switzerland.

With the exception of 26 strains, there was close concordance between the results obtained with the monoclonal antibodies from both sources and with the polyclonal antisera. All 26 discordant strains were agglutinated by polyclonal anti-Agg 1, but only 9 were agglutinated by the monoclonal CAMR anti-Agg 1. One strain was agglutinated by both monoclonal antibodies to Fim 2 but not by the polyclonal anti-Agg 2 serum. Ten strains were agglutinated by the CAMR anti-Agg 3 antibody and the polyclonal anti-Agg 3 but not by FDA/F3 antibody. However, one strain was agglutinated by FDA/F3 and polyclonal anti-Agg 3 reagents but not by the CAMR anti-Agg 3.

The CAMR anti-Agg 2 and FDA/F2 antibodies showed similar sensitivities, whereas CAMR anti-Agg 3 was clearly superior to FDA/F3. CAMR anti-Agg 2 and anti-Agg 3 and FDA/F2 could therefore be adopted as standard serotyping reagents. The CAMR anti-Agg 1 was inferior to the polyclonal reagent in sensitivity but may not have been detecting the same antigens. The identity of Agg 1 detected by polyclonal reagents has not been conclusively resolved and its epidemiological relevance is unclear. The CAMR and FDA reagents detect fimbrial components (1, 2), although there may be incomplete overlap of specificity. This and differences in isotype and/or subclass could explain the discrepancies noted in this and in the previous study.

REFERENCES


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