Since late 1970, nine isolates expressing dual capsules (W and Y) have been described in North America and Europe (1–4). Although the origin of such isolates and the precise composition of the capsular polysaccharide expressed by them are open issues, meningococcal vaccines that include serogroup W and Y polysaccharides likely induce protective immunity against these mutants (3, 4). Critical points for laboratory-based surveillance are the PCR methods used to diagnose meningococcal disease, because they are not designed to recognize the unique capsular composition of these isolates (2–4). Accordingly, a nucleic acid amplification assay to discriminate among serogroups W, Y, and mixed W/Y has been proposed (4).

We present a description of a Neisseria meningitidis isolate (RJ122/08) expressing mixed W/Y polysaccharides isolated in September of 2008 from a patient in Rio de Janeiro State, Brazil. The serogroup was determined by slide agglutination with specific rabbit antisera (BD Difco) and serogroup-specific PCR (5). Serotype and serosubtype were determined by immunoblot analysis at the National Meningitis Reference Centre (Instituto Adolfo Lutz, São Paulo, Brazil). Multilocus sequence type (MLST) analysis and sequencing of outer membrane protein genes porB, porA, fetA, and fHbp and DNA sequencing of the csd (formerly siaDw) gene were performed as previously described (3, 6).

The organism was isolated from the cerebrospinal fluid (CSF) of a 3-year-old girl with suppurative meningitis and a nonblanching rash. Although the isolate was identified as serogroup Y by PCR, it agglutinated with both anti-W and anti-Y antisera. Subcapsular antigens were characterized as 17,7:P1.5. The genotype was defined as 3-100:PL1.5-1,10-80:F1-7:ST-7694 (clonal complex 175 [cc175]), with fHbp in variant 3/subfamily A (peptide identification no. [ID] 162). Sequencing of the csd gene revealed a point mutation resulting in replacement of glycine (cysG) or proline (cwsG) with serine at amino acid position 310, which leads to this dual antigenic specificity (7). There were two other mutations at amino acid position 157 and 935, which result in the same amino acid sequence as that encountered in the serogroup W csd gene (3).

Since 2000, five additional serogroup Y meningococcal isolates have been recovered from patients in Rio de Janeiro State: three Y:17,10:PL1.5, one Y:19,14:PL1.5,2, and one Y:19,1:PL1.5. The isolates were genotypically characterized as 3-48:PL1.5-1,10-4:F4-12:ST-6526 (cc167), 3-48:PL1.5-1,10-4:F4-12:ST-7711 (cc167), 3-298:PL1.5-1,2:2F5-8:ST-23 (cc23), and 3-36:PL1.5-1,10-1:F4-1:ST-1655 (cc23); they all had fHbp variant 2/subfamily A (peptide ID 23 or 25). By MLST analysis, 98% of 57 W isolates belonged to cc11, many times characterized as serotype 2a (95%); a single W:19,21 isolate belonged to cc174.

Previous reports of isolates expressing dual capsules noted that those isolates had all the antigenic and genetic features characteristic of serogroup Y N. meningitidis, i.e., cc23 and cc167 (2, 3). The mixed-serogroup W/Y N. meningitidis isolate described here has a genetic background that is distinct from those of other serogroup Y or W isolates from Rio de Janeiro State but belongs to a clonal complex that is typically associated with serogroup Y in other Brazilian states, Chile, and Argentina (8). ST-175 complex meningococcal isolates from neighboring countries frequently possess porB, porA, and fetA alleles identical to those present in our isolate (8–11), suggesting that a common strain circulating in South America underwent a mutational event that led to the expression of the mixed polysaccharides described here. The monitoring of such isolates has implications for routine meningococcal disease surveillance.

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