CORRESPONDENCE

No Evidence for Serosubtype-Restricted Protection among Teenagers Vaccinated with the Norwegian Group B Outer Membrane Vesicle Vaccine

To the Editor—We read with great interest the article by Jones et al. [1] describing the dynamics of carriage of Neisseria meningitidis among military recruits. Of particular interest was their finding of serosubtype-specific bactericidal responses to the acquired meningococcal strains, a finding that supported previous results from various laboratories on the role of PorA antigens in eliciting bactericidal antibodies. However, we disagree with their statement when discussing the protection of the Norwegian B:15:P1.7,16 outer membrane vesicle (OMV) vaccine. Referring to a study that involved only a small number of vaccinees who became ill with meningococcal disease [2], Jones et al. claimed that meningococcal infections in recipients of this vaccine were caused by heterologous strains only, indicating serosubtype-specific protection.

We have analyzed all strains from participants who contracted meningococcal disease during the protection trial done among teenagers in Norway [3]. In the blinded part of the trial, 6 of 12 vaccinees and 10 of 24 placebo controls became ill with strains expressing PorA with P1.7 or P1.16 (or both) subtypes [4]. When the results for the first year of the following open trial were included, 10 of 20 vaccinees and 16 of 35 controls were infected with corresponding strains [5]. These data show no significant differences in the distribution of strains with vaccine-like subtypes causing disease among vaccinees and controls (χ² test with Yates’s correction, P > .90).

Guttormsen et al. [2] reported that 4 of 7 vaccinees and 3 of 18 nonvaccinees were infected with B:15:P1.12 strains. Such strains were later found to be localized mainly to the western part of Norway [6]. This is the region where most of the patients studied by Guttormsen et al. [2] live, which might explain the authors’ assumption of some subtype-specific protection. However, from their data, we find no significant difference in the distribution of the P1.12 subtype between the 2 patient groups (Fisher’s exact test, P = .066). Three of the vaccinees infected with B:15:P1.12 strains had been immunized 3 years prior to disease [2], and the protection of the OMV vaccine has been shown to wane following a two-dose regimen [7]. The possibility that vaccination might select for infectious strains with mutations in the P1.7,16 PorA has also been analyzed previously [8], but no indication for the emergence of such strains was demonstrated. Thus, the data available give no evidence to conclude that infection of teenagers vaccinated with the Norwegian OMV vaccine was only caused by heterologous strains.

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The Journal of Infectious Diseases 1999;180:242
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Reply

To the Editor—In their paper, Guttormsen et al. [1] concluded that of 7 vaccinees who contracted meningococcal disease after being vaccinated with outer membrane vesicle (OMV) vaccine, “none . . . was infected with meningococcal strains containing class 1 protein homologous or partly homologous to that of the vaccine strain, indicating serosubtype-specific protection.” Wedege et al. now provide additional data to indicate that this conclusion represents a narrow geographic interpretation of the outcome of the Norwegian OMV vaccine trial. We fully accept their analysis.

That the OMV vaccine failed to protect some individuals from infection with homologous strains emphasizes the importance of improving our understanding of the relative contribution that individual antigens make toward immunity to meningococcal infection and the dynamics of that protective effect. We reiterate the findings of our study [2], which indicate