Genetic Control of Antibody Responses to Pneumococcal Capsular Polysaccharides

To the Editor—Brousseau et al [1] have nicely shown that about 50% of vaccine-naive children who develop invasive pneumococcal disease (IPD) fail to develop antibody >0.35 µg/mL to their infecting serotype after infection. Further, they report that some children infected with a specific serotype of *Streptococcus pneumoniae* have deficient capacity to make antibody to that serotype after vaccination with protein conjugate pneumococcal vaccine (PCV), whether the vaccine is given prior to or following the IPD. They state that this result is unexpected because “PCV is highly immunogenic for all vaccine serotypes.” Although that statement is true, in general, it does not apply to all individuals.

In careful studies of healthy adults and family clusters, we previously reported that some persons fail to respond to pneumococcal polysaccharide vaccine (PPSV23) and that the capacity to respond is genetically controlled [2]. This finding was not surprising, although there has been remarkably little attention to the genetics of responses to polysaccharides compared to responses to protein antigens. We subsequently reported [3] that this genetic lack of responsiveness was overcome in some, but not all, instances by vaccinating with protein-conjugate polysaccharides. This work predated the availability of PCV7, so we were dependent upon a variety of protein-conjugate preparations. Nonetheless, the findings suggested that, although protein conjugation enables some individuals to overcome the genetic inability to generate antibody to pneumococcal capsular polysaccharides, there are others who fail to respond even after vaccination with polysaccharide conjugates. In other words, responses to vaccination are governed by as-yet unrecognized genetic factors, and it is not really surprising that some individuals fail to respond to the battery of capsular polysaccharides contained in PCV7 or PCV13. It is, of course, also true, as suggested by Brousseau et al [1], that hyporesponsiveness to the vaccine after the antigenic stimulus of infection may be responsible, perhaps related to the generation of suppressor T cells [4].

Note

Potential conflict of interest. Author certifies no potential conflicts of interest. The author has submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

Daniel M. Musher^1,2

1Baylor College of Medicine, and 2Medical Care Line, Infectious Diseases Section, Michael E. DeBakey Veterans Affairs Medical Center, Houston, Texas

References


Correspondence: D. M. Musher, One Baylor Plaza, Houston, TX 77030 (daniel.musher@va.gov).

Clinical Infectious Diseases® 2016;62(1):132–3
© The Author 2015. Published by Oxford University Press for the Infectious Diseases Society of America. All rights reserved. For permissions, e-mail journals.permissions@oup.com. DOI: 10.1093/cid/civ810