Vβ Expression in Healthy Subjects and during Streptococcal Infection

To the Editor—Using a polymerase chain reaction (PCR) method, Watanabe-Ohnishi et al. [1] found a decrease in the proportions of T cells expressing Vβ1, Vβ5.1, and Vβ12, with no increase in any other Vβ subsets, after severe group B streptococcal infections. In contrast, an expansion of the Vβ2-expressing subset of T lymphocytes has been documented by flow cytometry (FACS) in 2 cases of streptococcal toxic shock syndrome [2]. Further, the control data in these two studies differed significantly as have reports of the proportions of Vβ2 and Vβ8 cells in normal subjects [1–8]. Watanabe-Ohnishi et al. reported the normal percentage of Vβ2 cells as 15% (all other estimates have been 6.5%–9%), and their normal percentage of Vβ8 was 5% (other estimates have been 2.5%–9%). Three variables might account for these findings. First, the techniques used (PCR vs. FACS) may be responsible. Second, control populations may vary widely in their expression of these chains—a bimodal distribution of Vβ2 expression has been described [6] and expression may be HLA linked [9]. Third, it is possible that the proportions of CD4 or CD8 cells differ in control populations; expression of Vβ chains differ in these subsets [8].

A practical method to enable the comparison of different data sets would be to document characteristics of T cell populations in addition to their Vβ chain expression. Such features should include the Vβ distribution on CD4 and CD8 cells and measures of T cell activation, such as the expression of surface interleukin-2 receptor, CD45 isofrom, or expression of a marker cytokine gene. With dual or triple labeling, one may start collecting a larger body of control data, leading to more accurate descriptions of the in vivo effects of bacterial infection and superantigen activity.

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References

My colleagues and I analyzed the Vβ repertoire of 11 patients who had STSS or were severely infected with group A streptococci and observed that some of the Vβ families were expanded, but there was no consistent pattern [2]. For example, the STSS patient depicted in figure 2 (of [2]) showed expansion of Vβ2 and Vβ3, but other patients showed expansion of different Vβs. In contrast, the pattern of Vβ depletion was more consistent among the cases we studied. We concluded that the expansion of certain Vβs is a compensation for the depletion in Vβ1, Vβ5.1, and Vβ12, because the total percent expression should be 100. If we had analyzed only 2 cases, we may have, by coincidence, picked 2 persons with an apparent Vβ2 expansion. However, we cannot rule out the possibility that different outbreaks of severe streptococcal infections at different times or in different geographic areas may be associated with different strains of Streptococcus pyogenes that produce different superantigens and thus may have different effects on a patient’s T cell Vβ repertoire. Thus, the patient’s Vβ repertoire may be a helpful marker in the identification of putative superantigens involved in disease in a particular patient or group of patients.