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Reply to Smithson et al.

**To the Editor—** Mannose-binding lectin (MBL) deficiency has been associated with a predisposition to numerous infectious diseases, and our recent data indicate that patients with pneumococcal infection who have MBL levels <0.5 µg/mL are more likely to die [1]. The data included in our recent meta-analysis excluded the initial study that showed a strong association between *MBL2* variant allele homozygosity and invasive pneumococcal infection [2], because these patients’ MBL blood levels were not measured. If these patients could have been included, the association we ascertained between MBL deficiency and death in pneumococcal infection would very likely have been even stronger than our finding of a OR of 5.62 (95% CI, 1.27–24.92). In their letter, Smithson et al. [3] indicate that conclusions regarding the significance of MBL deficiency need to be viewed in the context of in vitro data that describe the binding of this protein to bacterial cells and the consequent deposition of complement. The earliest data relating to *Streptococcus pneumoniae* demonstrated binding of the bacteria to MBL [4], but more-recent data indicate that encapsulation of pneumococci abrogates binding [5]. One crucial aspect of the contribution of MBL to the killing of pneumococci that has not been studied to date is the contribution of neutrophils. Until these data are available, it is premature to ignore the strong association between MBL deficiency and poor outcomes of pneumococcal infection.

It is important to examine the possible association between *Staphylococcus aureus* sepsis and MBL deficiency, because of the in vitro observations of MBL binding to *S. aureus* and the resulting increased phagocytosis [6]. The only published data on *S. aureus* sepsis come from our 2 studies [1, 7] and are summarized in the recent meta-analysis. On the basis of 49 patients with staphylococcal sepsis, no clear association with MBL deficiency was demonstrated. If we assume that the magnitude of effect of MBL deficiency is the same in staphylococcal and pneumococcal sepsis, a minimum sample size of 60 would be required for an appropriately powered study. The study of the relationship between MBL deficiency and staphylococcal sepsis is certainly a priority in this field of research.

The central theme of the biology of MBL is its pleuripotency. This pattern-recognition molecule binds to pathogen-associated molecular patterns from an extraordinarily broad range of organisms. The numerous documented associations of MBL with diverse infections [8] support this broad range of action, but proof of a critical role in the prevention or amelioration of infectious diseases morbidity relies on human clinical trials.

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**Laboratory-Acquired Clostridium difficile Polymerase Chain Reaction Ribotype 027: A New Risk for Laboratory Workers?**

**To the Editor—** *Clostridium difficile* is not recognized as a pathogen that presents a risk of acquisition in the laboratory, and no particular safety precautions are recommended for working with this microorganism [1]. We report 2 cases of laboratory acquisition of *C. difficile* infection