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Reply to Cao et al.

To the Editor—We thank Cao et al. for their response [1] to our recent article [2] on the alternative treatment of secondary bacterial pneumonia after influenza. We agree that interleukin (IL)–10 is an important cytokine in the response to many infections. One poorly understood aspect of severe lung infections is the contribution made by the host inflammatory response to disease and death. In preclinical models, the inflammatory response is needed to control bacterial infections [3], but too much inflammation leads to lung damage and increased mortality [4]. An emerging concept in the study of severe infections is that a balance between anti- and proinflammatory activity is necessary for the resolution of infection and survival [5]. In our preclinical model of secondary pneumococcal pneumonia after influenza, an exaggerated and dysfunctional cytokine response occurs and contributes to mortality [6, 7]. Treatment of these infections with cell wall–active antibiotics eliminates the infecting organisms, but the inflammatory burst that occurs after lysis of the bacteria can be fatal to the host [2, 8]. An ideal treatment regimen would eliminate bacterial pathogens while limiting inflammatory damage to the host, such that both morbidity and mortality would be reduced [9].

Indeed, in earlier studies using our mouse model, IL-10 levels were found to be strikingly elevated in mice with severe pneumonia [6]. In our recent study [2], IL-10 levels were similarly elevated (mean ± SD, 11,304 ± 2037 pg/mL; n = 5 mice) in the lungs of control mice infected with influenza virus followed 7 days later by Streptococcus pneumoniae. However, IL-10 levels did not change after treatment with either ampicillin (mean ± SD, 11,200 ± 1417 pg/mL; n = 8 mice) or clindamycin (mean ± SD, 11,643 ± 1473 pg/mL; n = 8 mice), as has been demonstrated for other proinflammatory cytokines and chemokines [2]. Therefore, although IL-10 may be important in the pathogenesis of severe lung infections, including those caused by bacterial superinfections after influenza, it is unlikely to be responsible for the difficulties inherent in the effective treatment of this disease.

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References

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Spontaneous Viral Clearance, Viral Load, and Genotype Distribution of Hepatitis C Virus (HCV) in European HIV-Infected Patients with Anti-HCV Antibodies

To the Editor—Soriano et al. [1] reported that hepatitis C virus (HCV) viremia was less common in HIV–infected patients with anti-HCV antibodies who were also positive for serum hepatitis B surface antigen. The increased probability of HCV clearance in patients with HBV-HCV coinfection was attributed by the authors to a viral interference phenomenon. We find it quite surprising that hepatitis delta virus (HDV) markers have not been reported in this study. In a nationwide survey performed in Spain, triple coinfection with hepatitis B virus (HBV), HCV, and HDV was demonstrated in ~70% of HIV-infected patients with coinfection due to multiple hepatitis viruses [2]. In addition, a number of studies from elsewhere in Europe have reported that, in HIV-infected patients with HBV, HCV, and HDV coinfection, HDV is the dominant virus that suppresses both HBV and HCV replication [3–5]. The authors should discuss whether the inhibition of HCV replication seen in their study was caused by HBV itself and/or by a possible undetected chronic infection with HDV. The current standard of care is to evaluate