protracted oromucositis in BMT recipients [7, 9] that is of quite a different character than the milder form induced by cytarabine. In contrast, high doses of cytarabine can be profoundly toxic to the gut and lungs [10]. The colonization of the stomach or digestive tract might therefore provide the alternative portal of entry that Bochud and colleagues suggested. Extensive colonization of the stomach and the small intestine would also be facilitated by any H2 antagonists used to manage the dyspepsia that frequently occurs following cytostatic chemotherapy.

The use of these agents was also implicated by Elting and associates [5] as a significant risk factor for the development of the so-called alpha strep shock syndrome. Moreover, patients with oromucositis tend to swallow large volumes of slimy mucus, which may assist in protecting the oral streptococci. Therefore, the presence of gastrointestinal colonization in patients with bacteremia due to *S. mitis* might explain why only a minority of these patients go on to develop the alpha strep shock syndrome; the microbial load may well be sufficient to elicit the release of cytokines that are necessary to induce sepsis syndrome, adult respiratory distress syndrome, and, in some cases, fatal multiorgan failure.

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Reply

SIR—We agree with Donnelly et al. that bacteremia due to oral viridans streptococci (OVS) in neutropenic patients with cancer has occurred not only while the patients were receiving prophylaxis with fluoroquinolones but also while they were receiving other prophylactic antibiotic regimens. This finding was inadvertently omitted in our text but cited in the references. The interesting data of Donnelly et al. as well as data from other centers, including ours, clearly suggest that aggressive cytostatic chemotherapy is probably the key factor predisposing neutropenic patients to OVS bacteremia. However, it appears clear that several widely used prophylactic agents, including fluoroquinolones and co-trimoxazole, are not effective in preventing OVS bacteremia. Moreover, OVS bacteremia was practically unknown before the use of these prophylactic regimens, and two case-control studies have shown an association between the use of quinolones or co-trimoxazole and the occurrence of OVS bacteremia [1, 2]. Thus, certain prophylactic antibiotics may not only be ineffective in preventing OVS bacteremia, but they may also alter the endogenous bacterial flora in a way that predisposes susceptible patients to the infection.

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