source of infection, a statement that we did not make. In the original article [4], we were asked to make the case for our hypothesis that hospital water was a potential source of *Aspergillus* conidia. We did so; however, we reiterated that this hypothesis needs to be considered in hospitals in which cases of nosocomial aspergillosis continue to occur despite the strict implementation of air-quality precautions. Gangneux and colleagues mention a study [5] that showed a correlation between airborne mold concentration and the incidence of invasive aspergillosis, which does not conflict with our hypothesis about waterborne mold. As discussed in our editorial, fungal conidia could have been “secondarily airborne” from a water source.

We thank Gangneux et al. [1] for raising these important questions, and we re-iterate the need for continued implementation of air-quality precautions in high-risk hospital wards and for additional studies to determine the exact contribution of alternative sources (i.e., water and other sources) to nosocomial and community-acquired aspergillosis.

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[1], which “originated in a retail source in which cooked meats were cross-contaminated” did not involve animal-derived VTEC with a number of such serotypes.

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Reply
Sr—Goldwater and Bettelheim [1] make the important and relevant point that non-O157:H7 and non–lactose-fermenting verocytotoxigenic Escherichia coli (VTEC) O157 strains are important contributors to the etiology of both VTEC disease and HUS. However, the authors appear to have overlooked the fact that the Central Scotland outbreak occurred in 1996; their useful and relevant work has all been published since 2000. In addition, the Central Scotland outbreak had the features of a protracted, single-source–type outbreak: it centered on a single butcher’s premises, and all cases of infection that occurred in patients who were included in the outbreak cohort fitted a clear case definition. In all cases in which a pathogen was identified (81% of the total), the infecting microorganism was confirmed to be the same strain (phage type 2; VT 2). Isolates from the butcher’s premises, from meat, and from infected individuals were confirmed to be an identical strain by pulsed-field gel electrophoresis. Although it is conceivable that other strains were involved, it is highly unlikely, under the circumstances.

Goldwater and Bettelheim’s letter [1] highlights an important difference in approach between investigation of a recognized outbreak of acute VTEC disease in which a predominant strain has been identified and retrospective investigation of cases of HUS. The role of non–lactose-fermenting and non-O157 VTEC cannot be underestimated, but such microorganisms were not thought to be relevant in the context of the Central Scotland outbreak.

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Linezolid-Induced Pancytopenia
Sr—I read with interest the recent article “Thrombocytopenia Associated with Linezolid Therapy,” by Attassi et al. [1]. I wish to report 2 cases of linezolid-induced pancytopenia.

Patient 1 was an 88-year-old woman with a vancomycin-resistant Enterococcus faecium infection associated with a prosthetic hip who refused prosthesis removal and was treated with suppressive linezolid therapy (600 mg twice daily). The patient had normal renal and hepatic function at baseline, was receiving no myelosuppressive medications, and was not receiving heparin. She developed mild nausea, vomiting, diarrhea, and progressive pancytopenia while receiving linezolid. On day 21 of linezolid therapy, the WBC count had decreased from 4.9 × 109 cells/μL at baseline to 2.5 × 109 cells/μL; the hemoglobin level had decreased from 11.5 to 7.9 g/dL; and the reticulocyte count was 0.55%. The platelet count had decreased from 298 × 109 to 160 × 109 platelets/μL by day 15 and continued to decrease progressively, to 99 × 109 platelets/μL on day 21. Linezolid therapy was stopped on day 21 after initiation of treatment. The patient’s WBC count and platelet count reached nadir 2 days after administration of linezolid ceased (2.3 × 109 cells/μL and 93 × 109 platelets/μL, respectively) and increased spontaneously 5 days after the drug was discontinued; the anemia was initially treated with erythropoietin alfa but was not corrected, and transfusions were required. There were no hemorrhagic or new infectious complications.

Patient 2 was an 84-year-old man with a group B streptococcus infection associated with a prosthetic knee who was treated with oral linezolid (600 mg twice daily) after removal of the prosthesis. Linezolid was used because the patient had documented allergic reactions to penicillin, cefazolin, sulfamethoxazole, vancomycin, and clindamycin. This patient was taking warfarin sodium for atrial fibrillation and was receiving no myelosuppressive drugs. On day 7 of linezolid therapy, the complete blood count was at baseline (WBC count, 5.7 × 109 cells/μL; hemoglobin level, 11.7 g/dL; platelet count, 170 × 109 platelets/μL); on day 20, however, the WBC count had decreased to 4.6 × 109 cells/μL, the hemoglobin level to 10.7 g/dL, and the platelet count to 82 × 109 platelets/μL. Linezolid adminis-