We thank Gelfand and Cleveland for their letter (1) regarding our meta-analysis on vancomycin-induced renal toxicity (2). They raise two additional important points. First, they suggest additional sources of bias with respect to interactions with pharmaceutical companies. The reason why we elected to perform this subgroup analysis was to prevent readers from suggesting that our findings are a direct result of vested interests by pharmaceutical companies to reduce the future role of vancomycin in therapy. Finding similar results in nonconflicted studies, we argue, strengthens the overall finding that vancomycin-induced nephrotoxicity is likely to occur more frequently with higher troughs. Nevertheless, we agree that all additional aspects listed by Gelfand et al. may be potential sources of bias which potentially favor opposing sides of the debate to various degrees, and thus, as pointed out by the authors, these would be impossible to ascertain and, consequently, correct for. A further limitation, not mentioned, which may have an even greater impact on our results is the reluctance of journals to publish negative studies, and thus, the precise estimate of vancomycin-induced nephrotoxicity is likely to remain uncertain.

Second, Gelfand et al., point out the long-term effects of acute renal injury, irrespective of cause, on an individual’s morbidity and mortality. Although this may be the case (supported by the cited evidence) we were unable to make this claim based on all the studies reviewed. To our knowledge, no study has followed patients with vancomycin-induced nephrotoxicity for an extended period, and thus long-term outcomes remain speculative. As mentioned in the discussion, however, it is likely that similar arguments about causality will arise, with more emphasis on host factors, rather than cause. Nevertheless, the long-term consequences of any adverse event should always form part of the decision process when choosing any antimicrobial agent for therapy.

REFERENCES

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