because in the time-kill kinetics assays, the subculture rifampin concentration was 4 mg/L.

We performed no classical fluctuation assays. We compared the Beijing genotype with the East African/Indian genotype to learn how M. tuberculosis strains differed in their capacity to withstand antituberculosis drug treatment. For reference strain H37Rv, mutation frequency was  $1.5 \times 10$ -6, higher than that found with higher subculture concentrations.

With regard to the 3 other issues, our drug-susceptibility testing of mutants showed a stable rifampin-resistant phenotype. We agree that these bacteria might represent preexisting mutants selected during drug exposure in a certain drug concentration window. By using different concentrations in subculture plates in our mutation frequency assay, we detected such preexisting mutants. Heteroresistance probably does not explain our observations because in our time-kill kinetics experiments, the whole mycobacterial population decreased over time in a drug concentration-dependent way, and regrowth of a drug-resistant subpopulation was not observed.

By not sticking to the fixed test conditions as used in the classical drug-susceptibility assays, research leads to highly interesting findings. One can conclude that serendipity flourishes with variation.

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## Correction: Vol. 18, No. 8

The name of author Arina Zanuzdana was misspelled in the article Vaccination of Health Care Workers to Protect Patients at Increased Risk for Acute Respiratory Disease. The article has been corrected online (http://wwwnc.cdc.gov/eid/article/18/8/11-1355\_intro.htm).

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