Response to Giufre et al

To the Editor—We thank Giufre et al for sharing their interesting data regarding the clonal distribution of *Escherichia coli* clinical isolates from their locale in relation to resistance phenotype, and we support their call for more such studies [1]. Their findings agree closely with ours regarding the predominance, since at least 2006, of sequence type (ST) 131 and, specifically, its H30 subclone, among
fluoroquinolone-resistant (FQ-R) E. coli isolates, as compared to ST131’s quite low prevalence among fluoroquinolone-susceptible (FQ-S) E. coli isolates [2].

A novel observation by Giufre et al pertains to the relative prevalence of the H30 ST131 subclone, compared with that of other ST131 subclones (mainly, H22), within the minor ST131 subset among FQ-S isolates. Specifically, Giufre et al found that the H30 subclone accounted for 13 (72%) of their 18 total FQ-S ST131 isolates from 2006, 2009, and 2012 combined [1]. In contrast, we have consistently found that the H30 subclone’s contribution to the FQ-S subset within ST131 is relatively small (<25%), including in our initial convenience sample study [2], a subsequent nationwide systematic survey of E. coli isolates from US veterans in 2011 [3], and a recent population-based survey from Olmsted County, Minnesota [4].

Although these differences could simply reflect study design differences, they also may indicate true geographical differences in the prevalence of FQ-S H30 subclone isolates. The relative prominence of FQ-S H31 isolates observed by Giufre et al suggests that factors other than just the FQ-R phenotype, such as high metabolic potential [5] or enhanced virulence, may partially explain, at least in some locales, the H30 subclone’s dramatic success over the past decade.

We agree strongly with Giufre et al that FQ resistance likely emerged within the H30 ST131 subclone on a single occasion, in a FQ-S H30 progenitor, via a specific combination of mutations in gyrA and parC, followed by the explosive expansion of the new FQ-R H30 subset [1]. Elucidation of the basis for the H30 ST131 subclone’s dramatic expansion after it acquired FQ resistance should provide critical insights into what allows an E. coli clone to “go viral,” which could lead to the development of sorely needed preventive interventions. Giufre et al have contributed additional evidence implicating the ST131 H30 subclone as the currently most successful strain of extra-intestinal pathogenic E. coli. Because of its clinical predominance and multidrug resistance, the H30 lineage conceivably has as great a social and economic impact as that of any lineage of methicillin-resistant Staphylococcus aureus.

Notes

Financial support. This work was supported by the Office of Research and Development, Medical Research Service, Department of Veterans Affairs (to J. R. J.), and by the National Institutes of Health (ARRA award 1RC4AI092828 to E. V. S.).

Potential conflicts of interest. J. R. J. has received grants and/or contracts from ICET, Merck, Rochester Medical, and Syntiron. E. V. S. has a research grant from Group Health. J. R. J., L. B. P., and E. V. S. have patent applications pertaining to diagnostic assays for E. coli lineages, including H30.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

James R. Johnson,1 Lance B. Price,2,3 and Evgeni V. Sokurenko4

1Veterans Affairs Medical Center and University of Minnesota, Minneapolis; 2Translational Genomics Research Institute, Flagstaff, Arizona; 3George Washington University, Washington, D. C.; and 4Department of Microbiology, University of Washington School of Medicine, Seattle

References


Received 31 July 2013; accepted 17 September 2013.
Correspondence: James R. Johnson, MD, Infectious Diseases (111F), 1 Veterans Dr, Minneapolis, MN 55417 (johns007@umn.edu).

The Journal of Infectious Diseases 2014;209:630–1
Published by Oxford University Press on behalf of the Infectious Diseases Society of America 2013. This work is written by US Government employee(s) and is in the public domain in the US.
DOI: 10.1093/infdis/jit582