

Escherichia coli Cluster Evaluation

To the Editor: Gupta et al. raise important issues regarding molecular profiling as an epidemiologic tool (1). First, since all living organisms are related, the goal of genomic profiling in public health epidemiology is not really to determine “whether such isolates are truly related” (1) (they are), but to define the degree of similarity—or, more specifically, to determine whether isolates are sufficiently closely related that the probability of their deriving immediately from the same point source is high enough to warrant epidemiologic investigation. Second, definitive assessment of genetic similarity relationships is challenging because of the limited accuracy and resolving power of conventional methods such as pulsed-field gel electrophoresis (PFGE) analysis (2) and the impracticality and expense of better performing technologies. Sequential use of multiple methods (such as PFGE with additional restriction enzymes) will predictably detect additional differences, thereby improving resolving power (2). Third, even if genetic similarity could be precisely defined, the relationship between the degree of genetic similarity and the probability of point-source spread is unknown and doubtless varies in relation to pretest probability, depending on the epidemiologic context (e.g., localized vs. multistate clusters). Even <100% similarity may be compatible with point-source spread when genetic drift exists within the reservoir, leading to dissemination of highly similar but nonidentical clones.

Gupta et al. interpret their experience as indicating that, with geographically dispersed isolates, a higher degree of genomic similarity than is reliably provided by single-enzyme PFGE is necessary to improve specificity, thereby avoiding fruitless investigative efforts (1). However,

whether the subclusters shown by their second-round PFGE were more epidemiologically meaningful than the original cluster remains unclear, nor do we know how representative this experience is. Determination of optimal genetic similarity parameters for geographically distributed epidemiologic surveillance (e.g., through PulseNet) would seem to require more in-depth empirical assessment, possibly incorporating Bayesian likelihood (3).

James R. Johnson*

*University of Minnesota, Minneapolis, Minnesota, USA

References

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Address for correspondence: James R. Johnson, VA Medical Center, Infectious Diseases (111F), Rm 3B-101 1 Veterans Dr, Minneapolis, MN 55417, USA; email: johns007@umn.edu

Novel Hantavirus Sequences in Shrew, Guinea

To the Editor: Hantaviruses, family *Bunyaviridae*, have been known as causative agents of hemorrhagic fever with renal syndrome in Asia and Europe (1,2) and hantavirus cardiopulmonary syndrome in the

Americas (3). Hantaviruses are spread by aerosolized rodent excreta and are strongly associated with their natural hosts, rodents of the family *Muridae*. Based on phylogenetic analyses, hantaviruses have been divided into 3 major groups that resemble 3 subfamilies of their natural hosts (Figure, panel A).

Recently, we found the first indigenous African hantavirus, Sangassou virus (SANGV), in an African wood mouse (*Hylomyscus simus*) collected in Guinea (5). Thottapalayam virus (TPMV), isolated from an Asian house shrew (*Suncus murinus*) in India (6), is the only known hantavirus to be hosted by a shrew instead of a rodent (7,8). We report the recovery of hantavirus RNA of a novel sequence from a shrew, collected in Guinea, West Africa.

During a study of rodentborne hemorrhagic fever viruses performed in Guinea in 2002–2004, 32 shrews of the genus *Crocidura* were collected and screened for hantavirus RNA by reverse transcription–PCR (5). An RNA sample designated Tan826 produced a PCR product of the expected size. The animal host was a male *Crocidura theresae* collected in the grassland savannah around the village Tanganya (10°00′02″N, 10°58′22″W) in January 2004. Species identification, following the taxonomic nomenclature (9), was performed on the basis of morpho-anatomical characteristics and was supported by molecular analyses.

Partial L segment sequence of 412 nt was determined by cloning and sequencing of the obtained PCR product. Nucleotide sequence comparisons between Tan826 and other representatives of the genus *Hantavirus* showed very low sequence identity values, ranging from 67.7% (Andes virus) to 72.3% (Puumala virus). Corresponding sequences of deduced viral RNA polymerase (137 aa) showed only slightly higher similarity values