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*).

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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
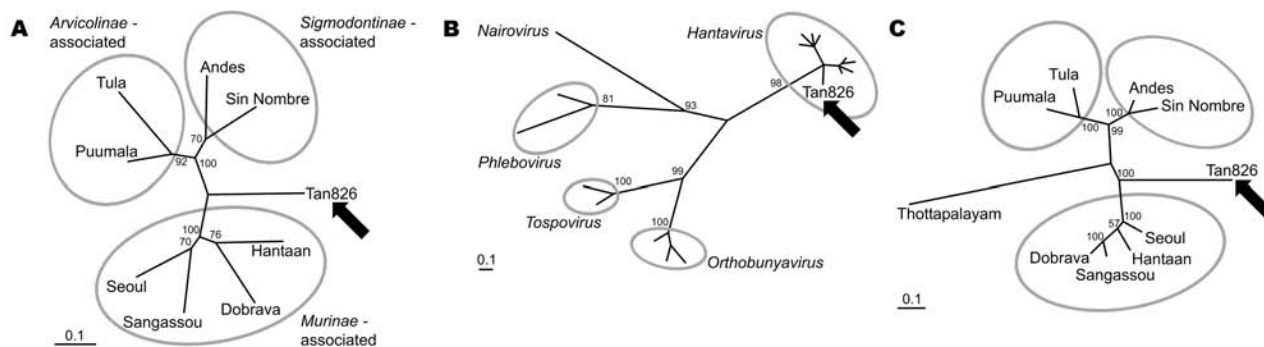
Figure. Maximum likelihood phylogenetic analysis of hantaviruses showing the phylogenetic placement of Tan826 (Tanganya virus, indicated by arrow) based on partial L segment nucleotide (A) and amino acid (B) sequences and partial S segment amino acid sequences (C); GenBank accession nos. EF050454 and EF050455, respectively. The values near the branches represent PUZZLE support values (*4*) calculated from 10,000 puzzling steps; only values ≥70% are shown. The scale bar indicates an evolutionary distance of 0.1 substitutions per position in the sequence. Gray ellipsoids indicate the 3 major hantavirus groups (panels A and C) or different genera of the *Bunyaviridae* family (panel B). A longer version of this caption providing a complete explanation of the analysis used is available online at www.cdc.gov/EID/content/13/3/520-G.htm

of 69.3% (Tula virus) to 76.6% (SANGV). In a maximum likelihood phylogenetic tree (Figure, panel A), Tan826 did not unambiguously cluster with any of the major groups (i.e., *Murinae*-, *Arvicolinae*-, *Sigmodontinae*-associated viruses) and showed equal relatedness to all 3 groups. This exceptional position of the Tan826 sequence within the tree is consistent with its detection in a shrew instead of a rodent host. Because the sequence is only distantly related to other hantaviruses, sequences from additional members of the *Bunyaviridae* family were analyzed. Despite use of a suboptimal dataset of very divergent and short sequences, the phylogenetic placement of Tan862 within the genus *Hantavirus* could be clearly demonstrated (Figure, panel B).

Furthermore, a partial S segment sequence (442 nt, 147 aa of the putative nucleoprotein) was determined to compare Tan826 directly with the shrew-associated TPMV (for which only an S segment sequence was available in GenBank). Rather unexpectedly, the Tan826 sequence showed the lowest similarity to TPMV: 47.5% on nt level and 39.4% on aa level. The identity values to other *Hantavirus* members were also extremely low, 52.2% (Sin Nombre virus) to 62.1% (SANGV) on nt level

and 50.6% (Andes virus) to 56.7% (Hantaan, Dobrava virus) on aa level. Corresponding aa sequences were then used for phylogenetic analysis to reduce problems derived from higher sequence diversities. In the resulting evolutionary tree, Tan826 and TPMV did not join any of the 3 major groups but also did not cluster together (Figure, panel C).

Our attempts to obtain more sequence data were hampered by the unique nature of the Tan826 virus sequence, which makes it difficult to design additional effective PCR primers, as well as by the limited amount of available biological material from the shrew. Nevertheless, the sequence and phylogenetic analyses of the 2 partial sequences strongly indicate that they represent a novel hantavirus. The amino acid sequences are highly divergent (≈25%–50%) from those of other hantaviruses and in phylogenetic trees; the Tan826 virus sequence appeared approximately equally related to those of all other hantaviruses. We propose to name the putative new species Tanganya virus (TGNV), after the locality where it was detected.

Detecting the virus in 1 of 32 *Crocidura* shrews, 15 of them *C. theresae*, is not sufficient to define *C. theresae* as a reservoir animal of this

novel virus. However, the unique position of TGNV in evolutionary trees supports the idea that a shrew instead of a rodent is the natural host of TGNV. Therefore, it is rather surprising that TGNV did not form a monophyletic group with TPMV. Before this observation becomes either a challenge or support for the hantavirus–host coevolution concept, more extensive sequence data (for comprehensive phylogenetic analysis) and epizootiologic studies (to confirm the natural hosts of both viruses) are necessary.

TGNV represents, after the recently described SANGV (*5*), a second hantavirus from Africa. Its low sequence similarity to other hantaviruses should make this virus serologically distinct from other hantaviruses, as shown for TPMV (*10*). Therefore, human infections by TGNV might be missed when using antibody detection assays based on antigens from conventional hantaviruses.

LETTERS

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Correction, Vol. 13, No. 1

In "Parvoviruses PARV4/5 in Hepatitis C Virus-infected Persons," by J. F. Fryer et al., an error occurred. The originally published title incorrectly included the word "Patient." The correct title is "Parvoviruses PARV4/5 in Hepatitis C Virus-infected Persons."
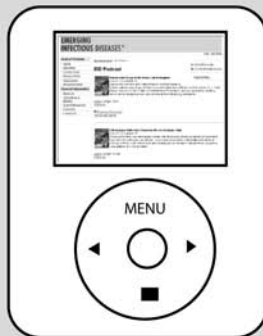
The updated article is available at http://www.cdc.gov/ncidod/EID/13/1/175.htm

We regret any confusion this error may have caused.

Correction, Vol. 13, No. 2

In "Avian Influenza Risk Perception, Europe and Asia," by Onno de Zwart et al., an error occurred. In the abstract, the last sentence should state, "Risk perceptions were higher in Europe than in Asia; efficacy beliefs were lower in Europe than in Asia."

The corrected abstract appears in the updated article, available at http://www.cdc.gov/EID/content/13/2/290.htm

We regret any confusion this error may have caused.