The presence of the tetracycline resistance determinant tetM has been reported for Mycoplasma hominis (1, 2) and association with Tn916 genes established (3). To identify the mobile genetic element harboring this gene, genome analysis of M. hominis Sprott (isolated from a nongonococcal urethritis patient before 1978 [4]) and targeted PCR/amplicon sequencing of M. hominis 1630 (1984, arthritis isolate [5]) were undertaken. These analyses revealed that in both isolates, truncated Tn916 resides within an uncharacterized transposon, closely related to several from streptococci that site-specifically integrate into rumA (6, 7). Although Tn916 truncation precludes mobility of that conjugative transposon (CTn), the larger mosaic element retains competency for excision and circularization in M. hominis. While this letter was in preparation, this element was also noted in the M. hominis placental strain PL5 (isolated between 1991 and 1996 [8]).

Comparison of the Sprott genome sequence (GenBank accession number CP011538 [9]) to those from M. hominis LBD-4 (10) and PG21 (11) disclosed a 25,257-nucleotide (nt) insertion, which included a truncated tetM-harboring Tn916 unit (13.3 kb). The boundaries of the mosaic transposon span from the C-terminal RumA-encoding region to the C-terminal region of a serine recombinase (SR) gene. Insertion of the element extends the RumA C terminus by 102 aa (to generate RumA'), and the SR gene product acquires a C-terminal 8-aa extension (*) that is derived from rumA. (B) A related transposon with identical Tn916 and deletion boundaries is present in M. hominis 1630. PCR products corresponding to the junctions of the element and deletions are shown. Lanes a to c correspond to the amplicon regions depicted by solid black bars. M is a 1-kb DNA ladder (Promega). (C) Detection of an empty site and extrachromosomal forms of the M. hominis Sprott element. An empty site was detected by PCR (lane 1) using the left primer for the PCR product (panel B, lane a) and the right primer for that of amplicon c. An extrachromosomal form is shown in lane 2 using outwards-facing oligonucleotides (black triangles in the M. hominis Sprott diagram). In this structure, the 3' region of SR fuses in frame with the 102-aa-C-terminus-encoding region of rumA (red triangles). All PCR products were sequenced by Sanger sequencing.
insertion was 36.2% G+C, in contrast to the core \textit{M. hominis} genome (27.1%). The 25.3-kb insertion appears to derive from a larger, uncharacterized CTn based on 98% identity to a 75-kb \textit{Streptococcus agalactiae} GB0555 CTn that contains a complete 18-kb Tn916 copy. The \textit{M. hominis} derivative evidently underwent deletion of \textasciitilde 50 kb that eliminated 5 kb from Tn916 and 45 kb (encompassing conjugation genes) from the GB0555-related transposon (Fig. 1). In \textit{M. hominis}, the transposon contains an 8.8-kb region with 100% nucleotide identity to a gene cluster encoding putative efflux proteins from GB0555 and that is also present, at 97% nucleotide sequence identity, in ICESp2905 (12).

An increasing number of mobile genetic elements (MGE) that target the \textit{rumA} 3’ region have been reported (6, 7). The GB0555 and Sprott elements insert at the same nucleotide, resulting in a translational fusion where the RumA C terminus acquires a 102-residue extension that derives from the transposon. Thus, a burgeoning number of RumA proteins in GenBank have almost identical C termini (derived from related transposons) despite having quite divergent sequences otherwise. For example, those from \textit{M. hominis} and GB0555 are 29% identical (amino acids [aa] 1 to 440) but 100% identical in their 102-residue C termini. Furthermore, following integration, the serine recombinase (SR) open reading frame (ORF) product that is predicted to catalyze integration acquires a C-terminal tag (<20 aa) derived from the host RumA ORF.

An extrachromosomal form of the \textit{M. hominis} Sprott element could be detected by inverse PCR, sequence analysis of which verified the \textit{rumA} excision site. Furthermore, the 102-residue RumA C-terminal extension is fused in frame to the SR C terminus, suggesting that the variant that mediates integration may require this domain for activity.

Although \textit{tetM} and Tn916 sequences have been reported in \textit{M. hominis}, the data from three \textit{M. hominis} lineages (different patients, collected in different decades) suggest that the MGE responsible for tetracycline resistance is actually the composite unit. This is in contrast to the situation in \textit{Ureaplasma urealyticum} serovar 9 (strain ATCC 33175), in which a full-length Tn916 in which a full-length \textit{tetM} responsible for tetracycline resistance is actually the composite unit.

\textit{Patients}, collected in different decades) suggest that the MGE re-...