tered. Moreover the appearance of these resistant organisms has been reflected in an increased failure of the antibiotics in question to treat infections caused by these bacteria. A comparison of the tetracycline resistance plasmid from *H. influenzae* with ampicillin resistance plasmids from the same strain has shown that the 2 types of resistance plasmids are very similar, save that one carries ampicillin transposon (Tn3-like) and the other has one that specifies resistance to tetracycline (Tn10-like)[2]. Thus it seems probable that the emergence of these resistant strains of *H. influenzae* occurred in 2 stages: first the organism acquired a plasmid which specified no resistance traits but which was able to replicate stably in *H. influenzae*. Then this plasmid acquired either an ampicillin-resistance transposon, or a tetracycline resistance transposon as a separate event. Presumably the source of these transposons were members of the Enterobacteriaceae - since the 2 types of transposon involved are very prevalent on plasmids in these species. But exactly when and where the events occurred is quite unclear.

In summary: One can see that bacterial DNA - and this is particularly the case with respect to DNA which specifies antibiotic resistance - is organized at 3 hierarchical levels - that of the whole bacterial chromosome, that of the bacterial plasmid and that of the transposon. In practical terms these hierarchies of DNA are continually interacting. Bacteria infect man: plasmids infect bacteria: transposons infect plasmids. Nor is this a sequence that relates only to antibiotic resistance; but our studies on antibiotic resistance in bacteria do allow us to study evolution in action, and this is becoming increasingly possible at the molecular level.

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DNA rearrangements and their importance in the evolution of gene systems

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1 Transposable Genetic elements as agents of gene instability and chromosomal rearrangements.
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ABSTRACTS

Structure at the ends of bacteriophage Mu DNA

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Bacteriophage Mu exhibits genetic properties that are analogous in many details to those of established insertion mutants in procaryotic and eucaryotic chromosomes (for a review, see DNA insertion Elements, Plasmids and Episomes [1977], Cold Spring Harbor Monograph Series, edited by A.I. Bukhari, J.A. Shapiro and S.L. Adya). These properties, which include the ability to be transposed, and to delete DNA adjacent to the insertion site, seem to involve invariant DNA sequences in the insertion elements. In Mu these particular sequences must lie at the ends of the viral genome, and these ends are covalently bound to heterogenous bacterial DNA in vegetative, but presumably not in lysogenic Mu DNA.

We have analyzed the nucleotide sequences at the 2 ends of the Mu DNA using the Maxam and Gilbert method. In some cases, these ends were present in a genetic material derived from lysogenic Mu. They were included in plaque-forming *λ-lac*-Mu hybrid particles constructed by recombination between Mu lysogen and *lac λplac*5. In one case the Mu SE (or variable) end was present in a plasmid constructed with a vegetative Mu DNA fragment and pMB 9. Our analyses lead to 3 main conclusions. 1. They show that a short stretch of 5 identical bases is located at each Mu end, oriented as an inverted repeat. 2. They strongly suggest that identical Mu end sequences are present in lysogenic and vegetative DNA. 3. They demonstrate that the heterogenous bacterial DNA bound to vegetative Mu DNA is completely removed during lysogenization, thus implying that the transposition and deletion events are, at least in part, site specific.