Insertion with Long Target Duplication: A Novel Mechanism for Bacterial Gene Mobility Suggested from Genome Comparison

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1 Introduction

One of the surprising conclusions that may be drawn from the complete sequences of bacterial genomes is that genes for restriction modification enzymes — everyday tools of molecular biologists — may represent a form of life, just as transposons and viruses [2]. Some of the restriction modification (RM) gene complexes appear to play an important role in shaping the bacterial genome.

2 Mechanisms for gene mobility suggested from genome comparison

Complete genome sequences of two closely related cellular organisms became available for the first time for two *Helicobacter pylori* strains [1,4]. Their comparison at single base pair level suggested presence of a novel mechanism for gene mobility — insertion with long target duplication [3]. It is formally similar to transposon insertion, but the duplication is much longer (often in the range of 100 bp), and the insert size and ends do not appear defined (Fig. 1). Similar structure was identified in comparison between *Neisseria meningitidis* and *Neisseria gonorhoeae* genomes. Restriction modification enzyme genes are often within or adjacent to the insertion. Horizontal transfer of restriction modification genes is suggested from their codon bias analysis and phylogenetic analysis in two *H. pylori* genomes. This as well as two more types of rearrangements linked with restriction modification genes — simple substitution structure (Fig. 2A) and tripartite structure composed of substitution/ inversion/ deletion — are hypothesized to result from attack of restriction enzyme on the chromosome [3]. Threat to a restriction modification gene complex would result in failure of methylation of chromosomal recognition sites and their attack by the restriction enzyme (Fig. 2B). Only if the broken ends are joined together with a DNA segment carrying the restriction modification gene complex, the methylation would resume and the restriction attack would cease. In brief, the parasite-host type interaction between restriction modification gene complex and the genome may have resulted in transposition of restriction modification gene complexes and genome rearrangements. This mechanism may have mediated insertion of pathogenicity island in *H. pylori*.

Acknowledgments

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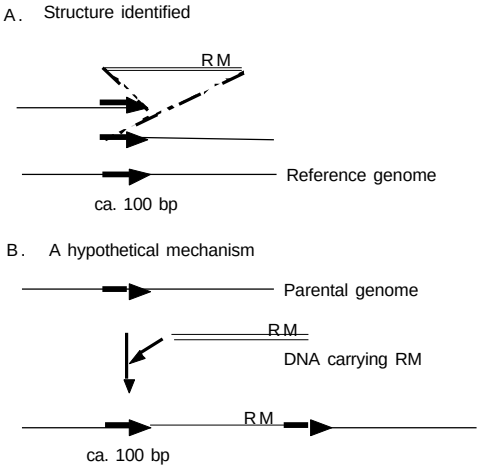

Figure 1: Insertion with long target duplication.

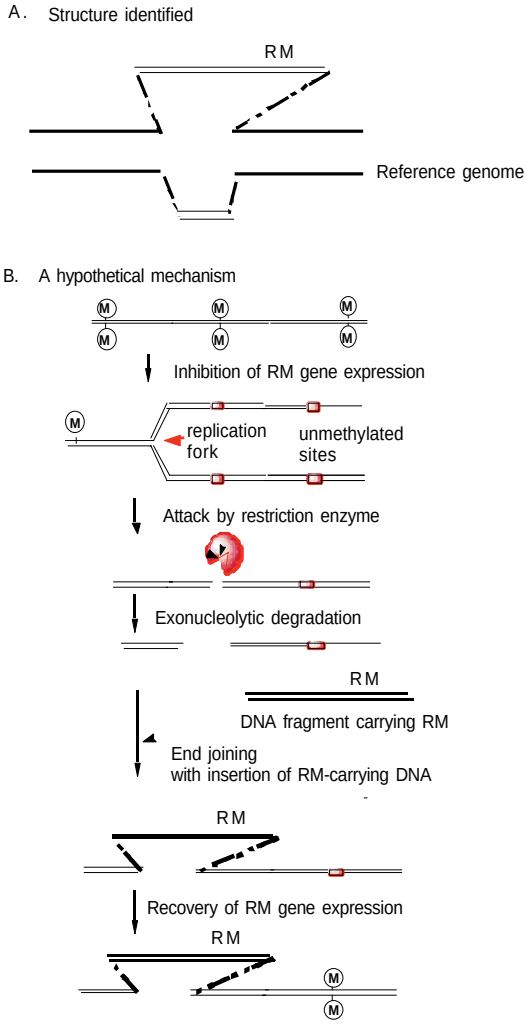

Figure 2: Insertion/ deletion (substitution)

References

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