

GUEST COMMENTARY

Evolving Insights: Symbiosis Islands and Horizontal Gene Transfer

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A common theme emerging from microbial genome studies is that the genes required for pathogenic or symbiotic interactions with eukaryotic hosts are often part of the accessory gene pool of the microbe, acquired by horizontal transfer (5). They may be clustered on plasmids or on the chromosome as genomic islands. Within a particular environment, such islands provide a selective advantage to organisms that carry them, leading to the term “fitness islands” (7), with the pathogenicity islands (PAIs) that endow many bacterial pathogens with their virulence constituting a particular subset (1). In many cases it appears that the transfer machinery of genomic islands has been lost through reductive evolution following their acquisition, anchoring the islands in the host chromosome (2). However, a particularly striking example of a genomic island that has retained its transmissibility is the *Mesorhizobium loti* strain R7A symbiosis island. The research that led to the characterization of this island represents an engaging and attractive “case history,” in which initial findings from a field experiment have logically progressed to the report by Sullivan et al. (10) in this issue of the sequence and annotation of the entire 502-kb island. This work impacts on diverse areas of microbiology, including the rhizobium-legume symbiosis, microbial ecology, and microbial evolution.

The studies had their origin in 1986, when *Lotus corniculatus* seeds, coated with a single *M. loti* inoculant strain, were planted in a remote field site in New Zealand. No indigenous rhizobia capable of forming root nodules on *Lotus* were present in the soil, and uninoculated plants died of nitrogen starvation. Seven years later, strains isolated from nodulated *Lotus* plants at the site were found to be genetically diverse but share the same chromosomally located symbiotic DNA (8). It was subsequently shown that these “new” *M. loti* strains arose by transfer of a 500-kb “symbiosis island” from the original inoculant strain to nonsymbiotic mesorhizobia present in the soil. The symbiosis island was found to integrate into the phenylalanine-tRNA gene of the recipients (9), in a process mediated by a P4-type integrase encoded at the left end of the island (cited in reference 10). This mode of integration has been shown, or is implied, for many other elements including several PAIs (2).

The similarities in structure and implied mechanism of transfer of the symbiosis island and other islands including PAIs are fascinating in view of the very different effects the

presence of these genes has on their respective eukaryotic hosts. Thus, future research on the mechanism and factors influencing transfer of this symbiotic island may reveal information directly relevant to unraveling the transfer mechanisms of other genomic islands. The island encodes a mating pore system similar in many respects to those of plasmids found in members of the family *Rhizobiaceae*, indicating it is likely to transfer by conjugation; however, the genes involved in the DNA processing reactions which precede transfer are not readily apparent (10).

The appearance of the symbiotic island in diverse nonsymbiotic mesorhizobia in a 7-year period indicates that transfer of this island is rapid in the soil/rhizosphere environment. Details regarding the actual kinetics of transfer, and the host range of transfer of the island remain to be determined. The latter is particularly interesting as there are genes within the symbiotic island which may well enhance the fitness of bacteria other than mesorhizobia.

Sullivan et al. (10) were able to extract information on the more recent evolutionary history of the R7A symbiosis island by comparing it to the sequence of the 611-kb putative symbiotic island from the recently sequenced genome of *M. loti* strain MAFF303099 (4). The two islands share a highly conserved ancestral backbone of only 248 kb. In both islands, the backbone has been disrupted by multiple insertions and deletions in a pattern reminiscent of the differences observed between the genomes of the related enteric bacteria *Escherichia coli* K-12 and *E. coli* O157:H7 (3, 6). A particularly intriguing finding is the likelihood that both islands encode the capacity to transfer molecules to plant cells, each employing a different mechanism. The R7A island encodes a type IV secretion system with strong similarity to the *vir* pilus from *Agrobacterium tumefaciens*. Only vestiges of this system remain on the MAFF303099 island; however, the MAFF303099 island in turn encodes a type III secretion system not found on the R7A island. Many of the hypothetical genes in the R7A island are clustered and entirely absent from the MAFF303099 genome, implying that they represent other genetic elements independently acquired by the R7A island. In addition, each island has a different complement of insertion sequences, suggesting that invasion by insertion sequences contributed substantially to the diversification of the two islands (10). One is left with a picture of the symbiosis islands being in a dynamic state, continually subjected to invasions by horizontally transferred DNA that are countered by gene loss through deletion events. Overall, the structures of the islands emphasize the remarkable similarities in the evolutionary strategies adopted by symbionts and pathogens in their quest to interact with eukaryotic hosts (5).

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REFERENCES

1. **Hacker, J., and E. Carniel.** 2001. Ecological fitness, genomic islands and bacterial pathogenicity—a Darwinian view of the evolution of microbes. *EMBO Rep.* **2**:376–381.
2. **Hacker, J., and J. B. Kaper.** 2000. Pathogenicity islands and the evolution of microbes. *Annu. Rev. Microbiol.* **54**:641–679.
3. **Hayashi, T., K. Makino, M. Ohnishi, K. Kurokawa, K. Ishii, K. Yokoyama, C. G. Han, E. Ohtsubo, K. Nakayama, T. Murata, M. Tanaka, T. Tobe, T. Iida, H. Takami, T. Honda, C. Sasakawa, N. Ogasawara, T. Yasunaga, S. Kuhara, T. Shiba, M. Hattori, and H. Shinagawa.** 2001. Complete genome sequence of enterohemorrhagic *Escherichia coli* O157:H7 and genomic comparison with a laboratory strain K-12. *DNA Res.* **8**:11–22.
4. **Kaneko, T., Y. Nakamura, S. Sato, E. Asamizu, T. Kato, S. Sasamoto, A. Watanabe, K. Idesawa, A. Ishikawa, K. Kawashima, T. Kimura, Y. Kishida, C. Kiyokawa, M. Kohara, M. Matsumoto, A. Matsuno, Y. Mochizuki, S. Nakayama, N. Nakazaki, S. Shimpo, M. Sugimoto, C. Takeuchi, M. Yamada, and S. Tabata.** 2000. Complete genome structure of the nitrogen-fixing symbiotic bacterium *Mesorhizobium loti*. *DNA Res.* **7**:331–338.
5. **Ochman, H., and N. A. Moran.** 2001. Genes lost and genes found: evolution of bacterial pathogenesis and symbiosis. *Science* **292**:1096–1098.
6. **Perna, N. T., G. Plunkett, V. Burland, B. Mau, J. D. Glasner, D. J. Rose, G. F. Mayhew, P. S. Evans, J. Gregor, H. A. Kirkpatrick, G. Posfai, J. Hackett, S. Klink, A. Boutin, Y. Shao, L. Miller, E. J. Grotbeck, N. W. Davis, A. Limk, E. T. Dimalanta, K. D. Potamouisis, J. Apodaca, T. S. Anantharaman, J. Y. Lin, G. Yen, D. C. Schwartz, R. A. Welch, and F. R. Blattner.** 2001. Genome sequence of enterohaemorrhagic *Escherichia coli* O157:H7. *Nature* **409**:529–533.
7. **Preston, G. M., B. Haubold, and P. B. Rainey.** 1998. Bacterial genomics and adaptation to life on plants: implications for the evolution of pathogenicity and symbiosis. *Curr. Opin. Microbiol.* **1**:589–597.
8. **Sullivan, J. T., H. N. Patrick, W. L. Lowther, D. B. Scott, and C. W. Ronson.** 1995. Nodulating strains of *Rhizobium loti* arise through chromosomal symbiotic gene transfer in the environment. *Proc. Natl. Acad. Sci. USA* **92**:8985–8989.
9. **Sullivan, J. T., and C. W. Ronson.** 1998. Evolution of rhizobia by acquisition of a 500-kb symbiosis island that integrates into a phe-tRNA gene. *Proc. Natl. Acad. Sci. USA* **95**:5145–5149.
10. **Sullivan, J. T., J. R. Trzebiatowski, R. W. Cruickshank, J. Gouzy, S. D. Brown, R. M. Elliot, D. J. Fleetwood, N. G. McCallum, U. Rossbach, G. S. Stuart, J. E. Weaver, R. J. Webby, F. J. de Bruijn, and C. W. Ronson.** 2002. Comparative sequence analysis of the symbiosis island of *Mesorhizobium loti* strain R7A. *J. Bacteriol.* **184**:3086–3095.

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