Complete Genome Sequences of Two *Pseudomonas aeruginosa* Temperate Phages, MP29 and MP42, Which Lack the Phage-Host CRISPR Interaction

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We report the complete genome sequence of two *Pseudomonas aeruginosa* phages MP29 and MP42. Their genomes are similar to those of *P. aeruginosa* temperate phages DMS3 and MP22, whose lysogens are impaired in swarming motilities, involving the host CRISPR loci. Both MP29 and MP42 lysogens, however, were proficient in swarming, suggesting the absence of the phage-host CRISPR interaction.

*Pseudomonas aeruginosa* is one of the ESKAPE pathogens, which are the major cause of the nosocomial infections and, more importantly, can escape from the effects of currently available antibiotics (5). It can cause not only acute infections but also chronic infections within the matrix-encased microbial community called biofilm. Biofilm formation in *P. aeruginosa* requires two types of group motilities (4): one is twitching, requiring type IV pili (TFP), and the other is swarming, involving flagellar function. The bacterial group behaviors, including group motilities and cell-cell communication, are newly emerging targets for antibacterial therapy in the era of antibiotics resistance.

It was recently observed that the swarming motility of *P. aeruginosa* strain PA14 was inhibited by lysogenization of temperate phages DMS3 (GenBank accession number DQ631426) (6) and MP22 (GenBank accession number DQ873690) (data not shown), which involves the non-identity-mediated interaction between the phage ORF42 and the CRISPR2 spacer 1 (5 mismatches in the 32-nucleotide [nt] spacer) (1). The host CRISPR loci constitute a bacterial immune system to the invading nucleic acids and generate small RNAs that assemble with the Cas (CRISPR-associated) proteins to form a nucleic complex and base pair (i.e., matches) with the target sequences, resulting in their destruction, similarly as in the eukaryotic RNA interference (3).

To increase the phage repertoire in these regards, we isolated a total of 7 new phages from the culture supernatants of clinical *P. aeruginosa* isolates. Among them, two phages, MP29 and MP42, were plaque-purified using *P. aeruginosa* strain PA14 as a host. Both phages require TFP for infection and, based on their morphologies, belong to the Siphoviridae family, as do DMS3 and MP22 (2). It was remarkable that the swarming motility was not inhibited by lysogenization of either MP29 or MP42, unlike DMS3 and MP22 (data not shown). To elucidate the phage genetic elements in the phage-host CRISPR interaction, we here present the complete genome sequences of both MP29 and MP42, in comparison with the DMS3 and MP22 genomes.

We performed whole-genome shotgun sequencing to 7.42- and 8.51-fold coverage of the MP29 and MP42 genomes, which were 36,632 bp and 36,847 bp in length, with 5ʹ-3ʹ-G-T-G ʹ at both ends of their linear genomes and G + C contents of 64.3% and 64.2%, respectively. They displayed synteny more closely to each other than to MP22 and DMS3, with similar organization of the 51 (for MP29) and 53 (for MP42) putative coding regions, which include the DNA transposition proteins involved in lysogenization.

Both phages contain two 32-bp target sequences in the ORF42, the nucleotide sequences of which are identical to those of MP22 and DMS3 (data not shown). Therefore, the nonidentity in this region might be necessary but not sufficient in the phage-host CRISPR interaction leading to swarming inhibition by these temperate phages. Further investigation based on the genome comparison between those phages could help to elucidate the molecular basis which underlies the role of the CRISPR-Cas system in the phage-mediated modulation of bacterial physiology.

**Nucleotide sequence accession numbers.** The whole-genome sequences of phages MP29 and MP42 have been deposited in GenBank under accession no. EU272036 and JQ762257, respectively.

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**REFERENCES**


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