A SMALL BACTERIOPHAGE PROTEIN DETERMINES THE HIERARCHY OVER CO-RESIDENTIAL JUMBO PHAGE IN *BACILLUS THURINGIENSIS* SEROVAR *ISRAELENSIS*

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Bacillus thuringiensis serovar *israelensis* is the most widely used biopesticide against insects, including vectors of animal and human diseases. Among several extrachromosomal elements, this endospore-forming entomopathogen harbors two bacteriophages: a linear DNA replicon named GIL01 that does not integrate into the chromosome during lysogeny and a circular-jumbo prophage known as pBtic235. Here, we show that GIL01 hinders the induction of cohabiting prophage pBtic235. The GIL01-encoded small protein, gp7, which interacts with the host LexA repressor, is a global transcription regulator and delays the induction of pBtic235 after DNA damage to allow GIL01 to selectively produce its own progeny. In a complex with host LexA in stressed cells, gp7 down-regulates the expression of more than 250 host and pBtic235 genes, many of which are involved in the cellular functions of genome maintenance, cell-wall transport, and membrane and protein stability. We show that gp7 homologs that are found exclusively in bacteriophages act in a similar fashion to enhance LexA's binding to DNA, while likely also affecting host gene expression. Our results provide evidence that GIL01 influences both its host and its co-resident bacteriophages.