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Abstract

Prophage BPs Alters Mycobacterial Gene Expression and Antibiotic Resistance †

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Abstract: Diseases caused by mycobacteria such as *Mycobacterium tuberculosis* are the leading cause of death worldwide. With the emergence of strains that are resistant to first-line anti-tuberculosis drugs and naturally drug-resistant pathogens such as *M. abscessus*, there is a need to increase our understanding of mycobacterial fitness and virulence and identify new targets for drugs. The majority of the pathogenic species of the bacterial genus *Mycobacterium*, including *M. tuberculosis*, carry integrated viral genomes (prophages) that are hypothesized to contribute to virulence. Though we know many of the ways in which phage genes directly contribute to pathogenesis, e.g., the CTX prophage encodes the toxin in *Vibrio cholera*, we know little about the impact of phages that encode no obvious toxin or virulence gene. Using an RNAseq approach, our lab recently showed for the first time that the presence of a prophage alters the expression of 7.4% of genes in the pathogenic mycobacterial species, *M. chelonae*. The presence of prophage BPs increased the expression of genes in the *whi*B7 regulon, including *whi*B7, *eis*2, and *tap*, and decreased the expression of a *pad*R-family transcription factor. BP lysogens were more resistant to aminoglycosides (kanamycin and amikacin) and tetracycline than wild-type strains of *M. chelonae*. In order to determine how the BP prophage drives changes in bacterial gene expression and phenotype, we will test the effects of individual BP genes expressed during lysogeny, such as the immunity repressor, on bacterial gene expression and antibiotic resistance phenotypes.

Keywords: prophage; Mycobacterium; antibiotic resistance; whiB7