DIFFERENTIAL EXPRESSION OF UNIVERSAL STRESS PROTEINS IN 
BACILLUS SPECIES

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Abstract: The Bacillus species are composed of a diverse group of Gram-positive bacteria found throughout the world. Species include the insect pathogens Bacillus thuringiensis and Bacillus larvae and the human pathogens Bacillus cereus and Bacillus anthracis, the etiological agent of the zoonotic disease anthrax. These organisms are genetically related, with major differences associated with large virulence plasmids. Gene expression in all of the species is tightly regulated by environmental conditions that can induce sporogenesis, the production of highly resistant, dormant endospores. In many organisms, universal stress proteins are essential for survival in these conditions. Using clustering analysis of the Integrated Microbial Genome (IMG) database, we identified usp genes within the Bacillus genomes. Within the Bacillus cereus group, 2 conserved genes were identified within each strain. Expression of both usp genes were identified in Bacillus cereus 10876 during the latter stages of exponential growth. The hypothesis of this study is that usp are differentially regulated in response to growth conditions which might be an important factor in its ability to survive adverse conditions. Bacillus species vegetative cells were cultured in complex medium supplemented with hydrochloric acid (100µL), sodium hydroxide (100µL) or sodium chloride (2.5%) for 6 hours. Aliquots were removed and the amount of growth measured spectrophotometrically (OD₆₀₀). Total RNA was isolated and end point and limited cycle reverse transcription PCR were used to monitor usp expression. While there was no significant difference in growth detected, usp ZP_04315896 was differentially regulated in response to the various growth conditions. This gene was also expressed in B. cereus 14579, Bacillus thuringiensis, and Bacillus mycoides. The identification of universal stress proteins in Bacillus is of significant importance as it may play a critical role in elucidating the mechanisms that allow these organisms to survive adverse conditions and increase pathogenicity.

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