

Bloom Syndrome Protein: A Study of Protein Partnerships

K.L. Bergeron, Y.I. Chekaluk, P.B. Falcao, E.L. Murphy and K.H. Almeida. Rhode Island College, Providence, RI

Background: Bloom syndrome is a rare recessive disorder characterized by premature death due to a predisposition to a wide array of cancerous states. An increase in genomic instability is used to diagnose Bloom Syndrome however; there is no known cure. Bloom Syndrome is caused by mutations of the BLM gene. The Almeida lab focuses on the partnerships of the BLM protein known to influence genomic stability.

Method: The N- and C-termini of BLM may affect stability by partnering with the homologous recombination repair protein Rad51. Therefore, a systematic set of deletion mutants has been generated for each terminus. DNA corresponding to each polypeptide fragment was cloned into Gateway entry vectors, sequenced and recombined into destination vectors for expression in *E. coli*. The oligopeptide fragments contain both a 6x His epitope tag on the N-terminus and a Flag epitope tag on the C-terminus.

Results: Each fragment was enriched to near homogeneity on Nickel affinity columns using the BioLogic LP system. The remaining impurity should not affect interaction studies, as the impurity is endogenous to the *E. coli* background.

Conclusions: Co-Immunoprecipitation analysis will determine the strength of each fragment's partnership with Rad51. Further experiments will investigate complex formation in the presence of DNA. Together these studies will clarify the role of BLM in maintaining genomic stability.

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