Evaluating Splice Variant Expression of the DNA Polymerase Beta Gene in BE2C and HEK Cells Exposed to *Borrelia burgdorferi*

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Introduction

The DNA Polymerase Beta gene is a gene that plays important role in DNA repair [1]. While it is expected to find *B. burgdorferi*, the primary bacterial factor for Lyme Disease, causes significant DNA damage, it is not known how some repair pathways nor different splice variants react [2].

Materials and methods

Cell Culture consisted of growing BE2C and HEK-293 cells. The cells were then infected with *B. burgdorferi*. Of which, RNA was extracted and was used to synthesize cDNA. qPCR was then used to determine gene expression and confirm the

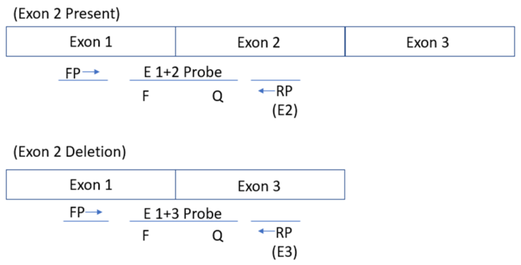

Figure 1. *Probing for exon deletions via overlapping an exon junction.*

Results

Our data indicates the expression of the key DNA repair Polymerase Beta gene (PolB) has a consistent upregulation when cells are exposed to *B. burgdorferi*. This is regardless of whether it is the complete or the splice variant missing exon 2 (ΔE2). However, it appears the bacteria affect BE2C cells as little as three-fold and as great as ten-fold higher than HEK, and the complete transcript tends to be more upregulated than ΔE2 isoform. It is novel that in every sample, there was a clear and meaningful upregulation.

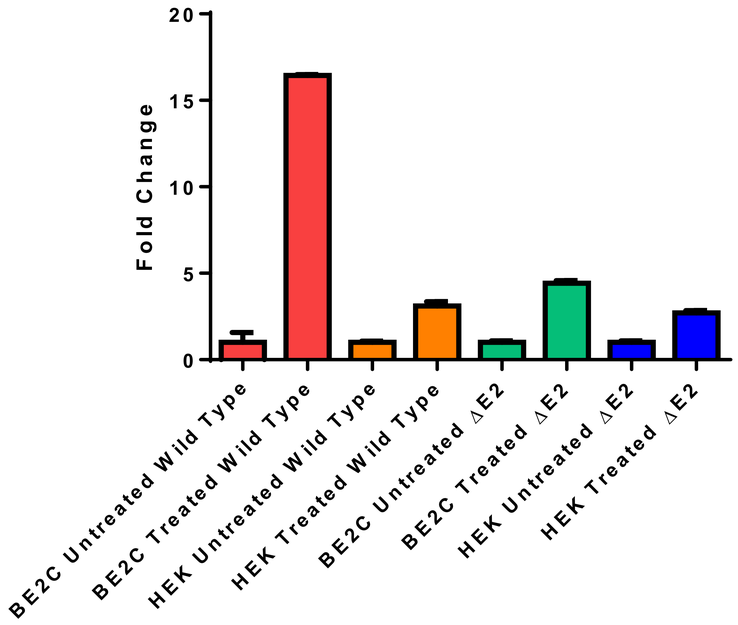

Figure 2. *Gene expression of the PolB gene in exposure to Borrelia burgdorferi. The expression of PolB shown by the graph above indicate a consistent upregulation of the gene in the treated cells.*

Conclusions

This study shows that infected cells have a significant upregulation of the Polymerase Beta gene indicating an increased use of DNA base-excision repair pathways. Previous studies have shown nucleotide-excision repair is run in tandem with Lyme disease, but base-excision repair seems much less investigated [4-6]. If the Polymerase Beta gene expression is impaired due to the *Borrelia burgdorferi* infection, then an in-depth analysis may provide some insight on the overall repair process and its contribution to pathogenesis of Lyme disease.

Literature cited

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