

## SMALL NON-CODING RNAS REGULATE STRESS RESISTANCE PROPERTIES OF UROPATHOGENIC *ESCHERICHIA COLI*

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Urinary tract infections (UTIs) are among the most common bacterial infections seen in an outpatient setting. The vast majority of UTIs are caused by Uropathogenic *Escherichia coli* (UPEC). During infection, UPEC activates host inflammatory response pathways, including the generation of reactive oxygen species. Reactive oxygen species can damage bacterial nucleic acids, lipids, and proteins. Bacteria have evolved many mechanisms to counter the damaging effects of reactive oxygen species. Some of these mechanisms are regulated by small non-coding RNA (sRNA) molecules.

Bacterial cultures were grown in 1mM methyl viologen (MV) LB broth to produce an oxidative stressful environment. Mutant strains that lack the sRNA Spot 42 ( $\Delta spf$ ) displayed growth attenuation in the presence of MV compared to wild type. qRT-PCR analysis was performed to determine mRNA transcript levels. Cultures were grown to an OD<sub>600</sub> of 0.6 and RNA was isolated. Spot 42 has known regulatory association with a handful of metabolically relevant genes. Of the 16 known targets, we saw no significant mRNA transcript variation with the exception of *ytfJ*. *ytfJ* transcripts were significantly increased in  $\Delta spf$  during oxidative stress (Fig. 1). This suggests that misregulation of *ytfJ* by Spot 42 may be leading to the observed phenotype. Unfortunately, the protein YtfJ has no known function. We predicted the structure of YtfJ by using the Phyre2 algorithm (Fig. 2) and compared it to Phyre2 web portal database. The proposed structure of YtfJ displays similarities to the thiol-disulfide oxidoreductase Etrx2 of *Streptococcus pneumoniae*. Thiol-disulfide oxidoreductases function to balance the redox environment of the cell. Because of this finding, we are developing a knockout strain of *ytfJ* and an overexpression *ytfJ* vector strain to determine if this gene is contributing to the growth defect observed in  $\Delta spf$  under oxidative stress.

Fig. 1.

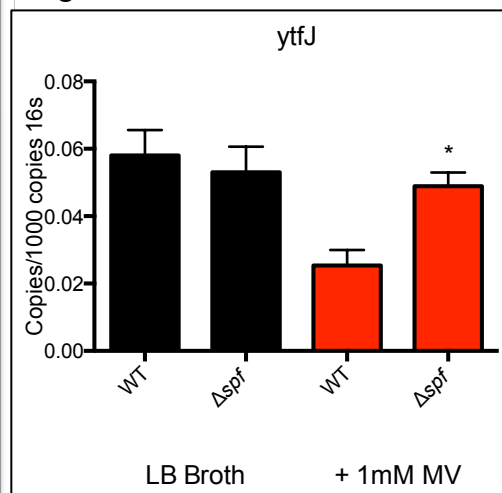


Fig. 2.

