## Two AraC/XylS-family transcription factors contribute to virulence and oxidative stress tolerance in *Acinetobacter baumannii* AB5075

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*Acinetobacter baumannii* is a major cause of hospital-acquired infection and a critical antimicrobial resistance threat. As treatment options narrow, targeting virulence regulators may provide an alternative therapeutic strategy. We focused on on AraC/xylS-like transcriptional regulators (AFTR) in *A. baumannii* 5075 (AB5075), *virF* and *alkR.* VirF from AB5075 is conserved across diverse recent clinical isolates and shares predicted structural similarity with VirF of *Shigella flexneri,* while previous work has implicated *alkR* in *Galleria mellonella* infection. To test their contribution to virulence, we performed *G. mellonella* killing assay and found that disruption of *virF* or *alkR* significantly attenuated virulence in vivo. We then assessed antibiotic susceptibility, biofilm formation and architecture, motility and growth under multiple stress conditions. Although the mutants did not differ from the wild type in antibiotic susceptibility, biofilm, or motility, both showed impaired growth under paraquat stress, but not hydrogen peroxide stress. These findings suggest that virFA and alkR contribute to virulence through adaptation to oxidative stress, potentially in response to superoxide-generating conditions. One next step will be to test whether these regulators control oxidative-stress genes and intracellular survival in THP-1 cells. Together, these findings identify VirFA and AlkR as candidate virulence regulators in *A. baumannii* AB5075, and, given the reported inhibition of AraC/XylS-family regulators by fatty acids, suggest a potential route for anti-virulence intervention.