During previous studies, we identified the fatty acid kinase FakA (also called VfrB) as a potent regulator of α-hemolysin and other virulence factors in *Staphylococcus aureus*. More recently, we have demonstrated that FakA is a positive activator of the SaeRS two-component regulatory system. This was revealed through mutational and activation studies. Specifically, generating a constitutively-active SaeS or by increasing that activation state of SaeS, we were able to bypass the FakA necessity for SaeRS-dependent promoter activation or repression. The result of FakA activation of SaeRS is changes in a multitude of key virulence factors. *In vivo*, the *fakA* mutant causes enhanced virulence in a murine model of skin infection. Collectively, our studies demonstrate multiple roles for FakA in *S. aureus* physiology and links virulence factor production to exogenous fatty acid metabolism.