Biofilms are structured communities of bacteria that adhere to surfaces and shield bacteria from hostile environments. They are a major problem in healthcare, accounting for an estimated 80% of human infections. Biofilms are inherently resistant to both antibiotics and host defense mechanisms. Thus, existing treatments often fail to eradicate biofilms resulting in chronic infections. We are working to develop enzyme-based therapeutics to prevent and/or disrupt biofilm formation. Our enzymes target a bacterial signalling system called quorum sensing. It is a precursor process to biofilm formation for many clinically relevant bacterial pathogens. The disruption of this signalling – “quorum quenching” – is a promising new approach for preventing biofilm formation. Many Gram-negative bacteria, including *Pseudomonas aeruginosa*, *Burkholderia* and *Acinetobacter* species, use acyl-homoserine lactones (AHLs) as signals for quorum sensing. We are engineering acylase enzymes to degrade these signalling molecules. Native acylases degrade AHLs by irreversibly cleaving them into their fatty acid and homoserine lactone products. However, the few AHL acylases characterised to date have poor heterologous expression, low yields and limited substrate specificity. To overcome this, I am using a highly expressed, well-characterised acylase to engineer quorum-quenching enzymes. While this acylase acts on different substrates to AHL acylases, the catalytic residues are conserved, and they share a similar structure. To date, I have made 630 variants using rational design and directed evolution, and have identified 2 mutants with activity towards AHLs using a high-throughput screen. Characterisation of the wild-type and variant enzymes will be presented.