

The skin is not only a physical barrier but also an active immune organ that protects us from infections. It acts as a frontline defence against environmental threats, including harmful microbes like *Staphylococcus aureus*, a bacterium commonly found on human skin that can cause infections and worsen chronic inflammatory conditions. Diseases such as atopic dermatitis (AD) are often accompanied by colonization or infection with *S. aureus*, which can exacerbate symptoms and complicate treatment.

This project explores the role of Regnase-1, a regulatory protein that modulates immune and inflammatory responses in skin cells. Regnase-1 is known to control the expression of key genes involved in inflammation, and its dysfunction may lead to an imbalanced immune environment that promotes susceptibility to infection. Importantly, studies have shown that Regnase-1 levels are reduced in skin lesions of patients with AD, which may be associated with increased bacterial colonization.

By investigating how Regnase-1 influences the skin's response to *S. aureus* infection, the project aims to uncover the molecular mechanisms that link inflammation and bacterial colonization. Using mouse models that closely mimic human skin infection and inflammation, we will analyse changes in gene expression and immune signalling pathways during the course of infection and inflammatory responses.

These insights could provide new targets for therapies to improve skin health, reduce infection risk, and alleviate chronic inflammation. Ultimately, this research will deepen our understanding of the complex interactions between the immune system, skin cells, and microbes, with potential implications for treating a range of inflammatory skin diseases complicated by bacterial infections.