

528e Early Versus Late Growth *S. Aureus* Adhesion to Immobilized Platelets under Physiological Shear Regimes

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Staphylococcus aureus causes a wide range of infections in humans. Infection is initiated by the adhesion of surface proteins on *S. aureus* to host extracellular matrix proteins including fibronectin (Fn) and fibrinogen (Fg). Since platelets can bind Fn and Fg via the $\alpha_{\text{IIb}}\beta_3$ receptor, the molecular mechanisms of infection and thrombosis are likely to be intertwined. *S. aureus* is known to bind Fn through the adhesins FnbpA and FnbpB. FnbpA has also been shown to interact with Fg. In addition, our previous studies have shown that Fg can mediate *S. aureus* – immobilized platelet interactions via the adhesins ClfA and SdrCDE. While *S. aureus* adhesins ClfA and SdrCDE are expressed in the late exponential growth phase, fibronectin binding proteins (Fnbps) are expressed in early exponential growth. Hence staphylococci could use alternate mechanisms to adhere to immobilized platelets depending on their growth phase. In this study, we demonstrate shear dependant adhesion of *S. aureus* to platelets via Fnbps both in the presence and absence of exogenously added fibrinogen and fibronectin. At all shear rates, a significant decrease in adhesion is reported with Fnbp negative mutant in the absence of exogenously added fibronectin. The above findings may lead to the development of novel therapeutics and preventive approaches against staphylococcal infections.