ABSTRACT

RESEARCH PAPER: Simvastatin reduces *S. aureus*-stimulated expression of C5aR on macrophages

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Inflammation is the hallmark response of the innate immune system in response to invading pathogen. It is orchestrated by macrophages and serves to contain and destroy pathogens. Not all pathogens can be immediately contained and eliminated. The spread of microorganisms can initiate sepsis, a life-threatening medical condition defined as a systemic inflammatory response resulting from infection. *Staphylococcus aureus* is a Gram-positive bacterium that is a prevalent cause of systemic infections resulting in sepsis. Statins, a class of lipid-lowering drugs, have been shown to be protective for sepsis due to their anti-inflammatory properties. While previous studies have examined the effects of statin treatments on macrophages and inflammation, the goal of these studies was to examine alterations in surface protein expression due to short-term, low dose simvastatin pretreatment of macrophages infected with *S. aureus* in vitro. Simvastatin treatment did not decrease expression of activation markers, MHC Class II, CD80, CD86, and CD40 on macrophages in response to *S. aureus*, but was sufficient to decrease expression of C5aR. These data demonstrate the potent and specific ability of simvastatin to influence gene expression and highlight the regulatory potential of simvastatin on complement activity.