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### Interactions of *Staphylococcus aureus* with Osteoclasts and Osteoblasts

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# INTERACTIONS OF *STAPHYLOCOCCUS AUREUS* WITH OSTEOCLASTS AND OSTEOBLASTS

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Osteomyelitis is infection-driven inflammatory disease of the bone primarily caused by *Staphylococcus aureus* (*S. aureus*), which results in pathological bone loss. Historically, *S. aureus* was thought to be an extracellular pathogen, yet new research has shown that *S. aureus* is internalized into many cells. To investigate the behavior of intracellular *S. aureus* in bone cells, we used an *in vitro* gentamicin protection assay to examine intracellular bacterial survival in osteoblasts, the cells that build bone, and osteoclasts, the cells that destroy bone, over several time points. We found that *S. aureus* persists in differentiated osteoblasts but is unable to replicate over the course of infection. However, in differentiated osteoclasts intracellular *S. aureus* is able to proliferate over time, whereas it is eliminated in osteoclast precursors. We next examined the intracellular location of *S. aureus* in osteoclasts to determine how *S. aureus* avoids elimination and replicates in these cells. We used fluorescence-based confocal microscopic imaging of the fluorescent dye LysoTracker, which marks acidified intracellular vesicles, with GFP-labelled *S. aureus* during *in vitro* infection of osteoclasts. We found that intracellular *S. aureus* is localized to lysosomes early in infection but not late in infection in osteoclasts, indicating a role for lysosomes in mediating clearance of intracellular *S. aureus*. Finally, to determine if the NLRP3 inflammasome affects intracellular *S. aureus* pathogenesis in osteoclasts, we utilized NLRP3 knockout osteoclasts in the *in vitro* gentamicin protection assay. Initial results suggest that loss of NLRP3 results in increased levels of intracellular bacteria over time, suggesting a role for the NLRP3 inflammasome in limiting bacterial growth in osteoclasts. Overall, the ability of *S. aureus* to persist within osteoblasts and osteoclasts and avoid progression of endocytic vesicles to lysosomes may provide a niche in which *S. aureus* can escape professional phagocytes and extracellular antibiotics, mediating the pathogenesis of osteomyelitis.