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Staphylococcus aureus panton-valentine leukocidin induces cytotoxicity in human monocytes through enhancing ROS and pro-inflammatory cytokines production

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The *Staphylococcus aureus* pore-forming toxin Panton-Valentine Leukocidin (PVL) is most likely causative for life-threatening necrotizing infections characterized by massive tissue inflammation and necrosis. Whereas the cytotoxic action of PVL on human neutrophils is already established, the PVL effects on other sensitive cell types are less clear and the mechanism of action of this toxin in *Staphylococcus aureus* virulence is controversial. Here we used an endemic PVL-positive SSTI-causing CA-MRSA strain from Taiwan, together with an isogenic PVL-knockout mutant (Δpvl) and complemented PVL-positive derivative to evaluate the role of PVL in the pathogenesis of CA-MRSA in the THP-1 human monocyte cell. We showed that PVL-positive CA-MRSA infected monocytes and macrophages leading to higher level of Reactive Oxygen Species (ROS) production, pro-inflammatory cytokines IL-1β and IL-18 expression and cell death than the isogenic Δpvl mutant. Specific inhibition with ROS inhibitor, diphenyleneiodonium, or caspase-1 inhibitor, AC-YVAD-CMK significantly reduced ROS generation, cytokines expression and cell death. The results suggest that PVL is an important virulence factor that enables CA-MRSA to trigger ROS production, increase the expression of caspase-1-dependent pro-inflammatory mediators and lead to cell death in human monocytes. These findings provide insight into leukocidin function and staphylococcal virulence and offer an important therapeutic target to severe staphylococcal disease.

Biography

Yun-Ju Lee has graduated from National Chiayi University of Department of Microbiology, Immunology and Biopharmaceutical. She specializes in immune assessment and has many years of animal experimentation and surgery experience.

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