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Press Release

How staphylococci trigger blood poisoning

Tübingen researchers identify lipopeptides as the culprit

Tübingen, 4 August 2016

Septicemia or blood poisoning caused by *Staphylococcus aureus* leads to thousands of deaths each year in Germany alone. Just how the infection begins – and can lead to multiple organ failure – was little understood until now. There are few options for diagnosing and treating the disease. Researchers at the University of Tübingen's Interfaculty Institute for Microbiology and Infection Medicine (IMIT) and the German Center for Infection Medicine (DZIF) headed by Dr. Dorothee Kretschmer, Dennis Hanzelmann and Professor Andreas Peschel have now uncovered a major cause of this life-threatening condition. The results of their study have been published in the latest issue of *Nature Communications.*

Many other pathogens – Gram-negative bacteria – form endotoxin molecules, which can lead to septicemia. But because staphylococci do not contain endotoxins, it was unclear just how they caused septicemia. The researchers have now shown that lipopeptides play a key role in triggering the disease and that certain staphylococcus bacteria form additional molecules – known as PSM peptides – which release these lipopeptides. Infections caused by PSM-negative staphylococci are also common but these staphylococci do not release lipopeptides and hardly set off septicemia in experimental infections.

Knowing that only certain staphylococci release PSM peptides may help doctors to better assess how dangerous a patient's *Staphylococcus* strain may be. It will also enable them to develop new treatments to prevent the formation of PSM and lipopeptides, thereby avoiding severe cases of blood poisoning.

Publication:
Dennis Hanzelmann, Hwang-Soo Joo, Mirita Franz-Wachtel, Tobias Hertlein, Stefan Stevanovic, Boris Macek, Christiane Wolz, Friedrich Götz, Michael Otto, Dorothee Kretschmer & Andreas Peschel: Toll-like

receptor 2 activation depends on lipopeptide shedding by bacterial surfactants. *Nature Communications*, 29 July 2016, DOI: 10.1038/ncomms12304

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