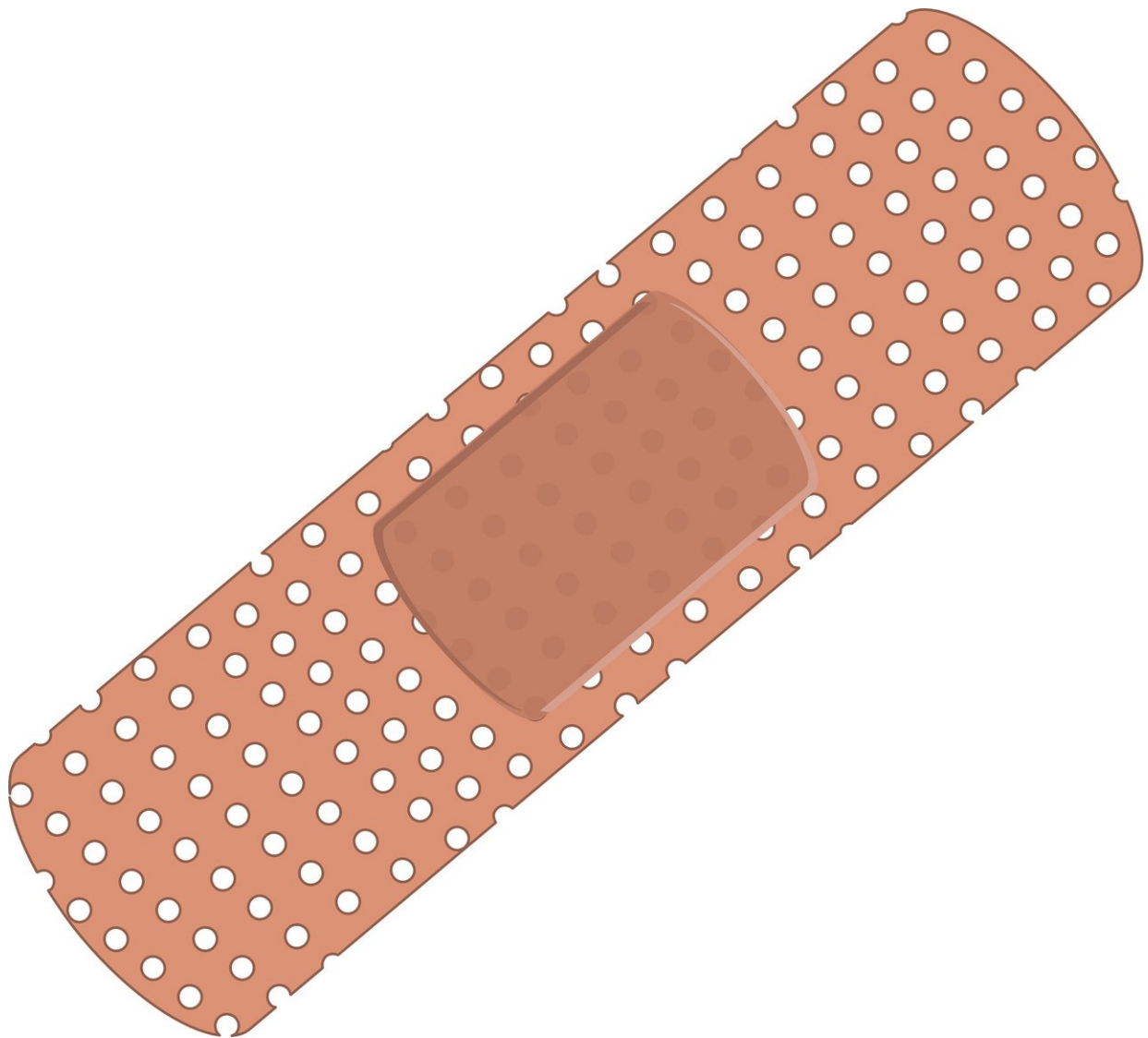


Researchers discover a new defensive mechanism against bacterial wound infections

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Wound inflammation that results in impaired wound healing can have serious consequences for patients. Researchers from Charité - Universitätsmedizin Berlin have discovered a new defensive mechanism that enables skin to actively kill bacteria. Central to this mechanism is a cellular messenger molecule known as interleukin 6, whose mode of action may be used in the future to prevent wound infections. Results from this research have been published in *PNAS*.

Skin wound colonization by bacteria or other pathogens can lead to severe inflammation. In the worst cases, this can result in septicemia or amputation. Prompt treatment is therefore essential. However, growing numbers of bacteria developing [antibiotic resistance](#) have resulted in limiting treatment options. A team of researchers, led by PD Dr. Frank Siebenhaar of the Department of Dermatology, Venereology and Allergology on Campus Charité Mitte, has now identified a new endogenous mechanism that may prevent wound infection without the need for antibiotics.

The researchers investigated the hypothesis that the skin's host defense against pathogens might include [mast cells](#), a type of defensive immune system cell which is known to play a major role in allergies. Mast [cells](#) are responsible for the body's response to otherwise harmless substances, producing symptoms such as runny nose or itching. However, researchers suspect that their role goes beyond this mediation of abnormal immune responses, with some results suggesting that they play a role in the body's defense against pathogens. This led PD Dr. Siebenhaar's research team to study whether and to what extent mast cells might be involved in the skin's host response to bacterial wound infection and in [wound healing](#).

Using an [animal model](#), the researchers studied the effects of an absence of mast cells on wound healing after infection. The researchers observed that on day five after infection, the total number of bacteria present in the wound was 20 times higher if mast cells were absent. This resulted in the infected wound taking several days longer to close. According to the researchers' findings, the mast cells' bacteria-killing effect is a product of the release of the messenger molecule interleukin 6. This molecule stimulates cells within the superficial layer of the skin, prompting them to release antimicrobial peptides, short protein chains that kill bacteria, viruses and fungi.

"Our study has demonstrated the nature and extent of mast cell involvement in the skin's host defense mechanism against bacteria," says PD Dr. Siebenhaar. He adds, "This helps us to better understand the significance of mast cells in the human body and how their role goes beyond that of a mere mediator of allergic reactions."

Exploiting their knowledge of interleukin 6 and its key function, the researchers found that the application of interleukin 6 to the wound prior to [infection](#) resulted in an improved defense against [bacteria](#), even in animals with intact immune systems. The researchers were also able to replicate this effect in human tissue. "It is in theory feasible that the application of interleukin 6, or of substances with a similar mode of action, could be used to prevent [wound infections](#)," explains the dermatologist. "As a next step, we will explore the functions of both mast cells and interleukin 6 in patients with chronic wound-healing problems. We will be going into greater detail in the hope of working towards a new and preferably antibiotics-free wound treatment method."

More information: C. Zimmermann et al, Mast cells are critical for controlling the bacterial burden and the healing of infected wounds, *Proceedings of the National Academy of Sciences* (2019). [DOI: 10.1073/pnas.1908816116](https://doi.org/10.1073/pnas.1908816116)

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