

Microbes paralyze immune system

About every fourth child is suffering from atopic dermatitis (AD) (often called atopic eczema) and adults are also often affected by this disease. The skin of AD patients is strongly damaged (Fig. 1). It is dry, itchy, red, swollen and cracked. Many patients suffer from infections in dry and open skin lesions, for example with pathogenic *Staphylococci*.



Fig. 1. The skin of AD patients is strongly damaged. It is dry, itchy, red, swollen and cracked.

There are about 200-fold more of these bacteria on AD skin in comparison to healthy skin resulting in infections and worsening of the disease. Normally immune system is responsible for combating pathogens. In AD this process is impaired. We found, how these bacteria avoid to be destroyed by immune cells. The microbes evolved a mechanism to paralyze immune cells (T cells). The substance mediating T-cell paralysis is part of the bacterial wall and is called lipoteichoic acid. It prevents immune cells to divide (Fig. 2). As lipoteichoic acid is set free during bacterial turnover and death, a large amount of this substance has access to immune cells some immune cells recognize this substance as a danger signal and alert the immune system. However, counterbalancing the immune response T cells are paralyzed and not able to exert their function.

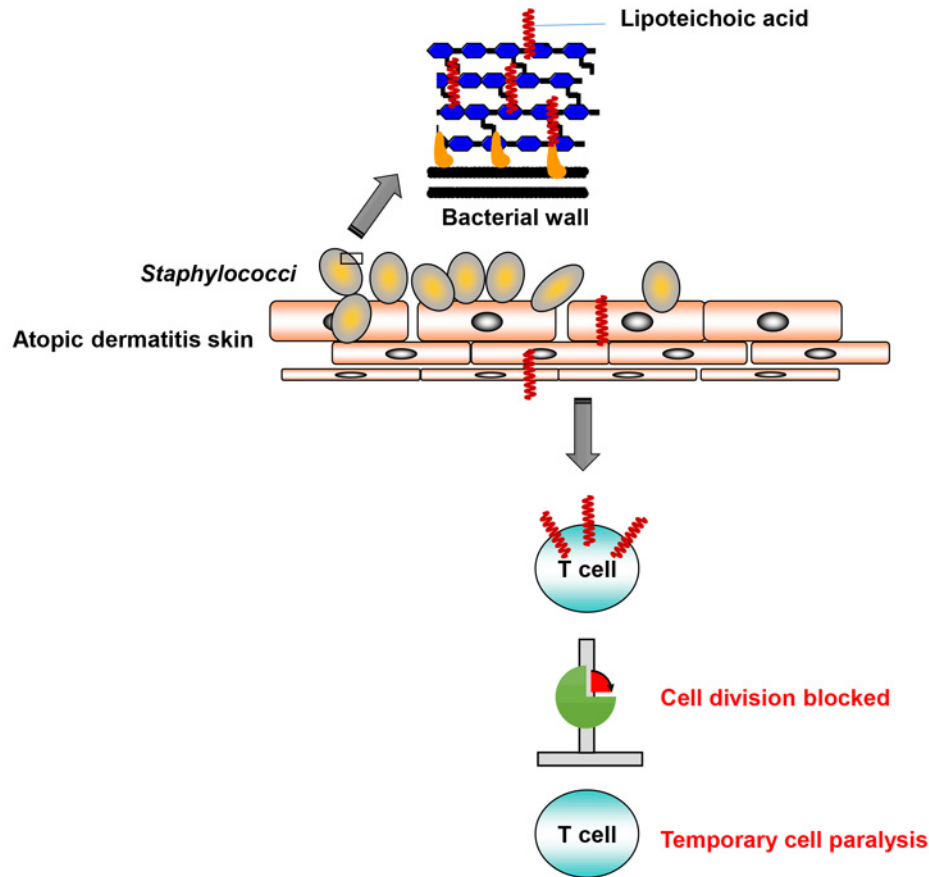


Fig. 2. In AD skin, lipoteichoic acid from Staphylococci interferes with immune cells thereby preventing activation and proliferation. As a result, the immune response is impaired, bacteria cause infection and worsen the disease.

As a result, bacteria can divide and cause more damage on the skin. Reducing bacteria thus also helps the immune system to function properly. Interestingly, the T cells are not dying and after removal of lipoteichoic acid immune cells are completely avoiding long-term damage to the immune systems. With this discovery we identified a new strategy how bacteria evade host immune responses.

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Publication

[Staphylococcus aureus-derived lipoteichoic acid induces temporary T-cell paralysis independent of Toll-like receptor 2.](#)

Kaesler S, Skabytska Y, Chen KM, Kempf WE, Volz T, Köberle M, Wölbing F, Hein U, Hartung T, Kirschning C, Röcken M, Biedermann T
J Allergy Clin Immunol. 2016 Mar 3