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## Nickel related *Staphylococcus aureus* infections in atopic dermatitis

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Pollution of the air, water and food by metals results in changes of microbiota because metals are common enzymatic cofactors of bacterial cells. Concentration of nickel in the air in Krakow is approximately 20 ng/m<sup>3</sup> in PM10. Nickel allergy is often found in patients with atopic dermatitis. Number of colonies of *Staphylococcus aureus* increases in acute phase and is reduced in remission of atopic dermatitis. Thus, it is believed that staphylococcal-derived infections exacerbate allergic inflammation. Nickel participates in virulence of *Staphylococcus aureus* and is required as a cofactor for bacterial enzymes including urease that can regulate pH. It is still unknown whether nickel-sensitive urease can regulate pH of the skin changed in patients with atopic dermatitis. In our current studies, 85% of patients with atopic dermatitis is infected by difficult to treat infections by methicillin-resistant *Staphylococcus aureus*. Bacterial infections are initiated by specific adhesion of a bacterium to the target environment. *Staphylococcus aureus* can attach to nickel nanostructures with dimensions comparable to the size of a single bacterium. Changes of cytokine milieu due to nickel action on T cells can increase number of immature Th0 and it can also promote staphylococcal infections because it reduces bacterial clearance. Thus, more immature T cells less cellular immune mechanisms protecting against staphylococcal infections. Therefore we believe that nickel allergy can promote *Staphylococcus aureus* infections in atopic dermatitis. All these studies are required to fully understand patho-mechanism of atopic dermatitis that is useful for more individual and consequently better treatment of patients.

### Biography

Anna Magdalena Bogdali graduated with her Master's degree at the Department of Biochemistry, Biophysics and Biotechnology of Jagiellonian University in Krakow. She completed her PhD on Atopic Dermatitis from Jagiellonian University Medical College in Medical Biology. She participated in the Socrates/Erasmus and 6<sup>th</sup> and 7<sup>th</sup> Framework European Programs. She stayed in the Angioedema Hungarian Center at the Semmelweis University in Budapest and she is involved in projects on hereditary angioedema concerning genetic background and immune mechanisms at the Jagiellonian University Medical College in Krakow. Her interests are immune and genetics mechanisms mostly related to T cells in the skin and circulatory system.

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