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Sugar governs how antibodies work in the immune system

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Antibodies protect the body against diseases – but can also harm their own organism if the reactions are misdirected. Researchers from the University of Zurich have now discovered that a particular sugar in the antibodies determines whether one of the body's own cells is destroyed or not. This result could lead to new treatment possibilities for patients with autoimmune diseases.

The immune system is our biological defense shield. Antibodies protect the organism against invading pathogens such as viruses or bacteria. In the case of certain autoimmune diseases, however, this defense behavior is misdirected: The antibodies don't just target foreign substances; they also attack the body's own cells. Once the antibody binds to the cell surface, they can activate specific proteins, so-called complement factors, which can damage the cell membrane and thus kill the cell.

Sialic acid protects from antibody-induced cell killing

A team of researchers headed by Professor Jan Lünemann from the Institute of Experimental Immunology at the University of Zurich has now discovered that a particular sugar structure in the antibody plays a key role in the complement-dependent destruction of the body's own tissue. Antibodies consist of protein and coupled sugar groups. Earlier studies revealed that antibodies with the sugar structure sialic acid are detectable more rarely in patients suffering from autoimmune diseases than in healthy people.

"We observed that patients suffering from an autoimmune disease felt better the more sialic-acid-carrying antibodies they had in their blood," reports Isaak Quast, a doctoral student in Lünemann's group and the study's first author. Different versions of antibody-coupled sugar structures were produced in the lab. "We managed to demonstrate that antibodies containing the sugar sialic acid only destroy the body's own cells to a very limited extent. Our data indicates that the coupling of sialic acid to antibodies might be a potential strategy in treating patients with autoimmune diseases," summarizes Lünemann.

More information: "Sialylation of lgG Fc domain impairs complement-dependent cytotoxicity." The *Journal of Clinical Investigation*, 2015, October 5. DOI: 10.1172/JCI82695

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