JACOBS UNIVERSITY RESEARCHERS EXPLAIN HOW HERPESVIRUSES ESCAPE FROM THE IMMUNE SYSTEM
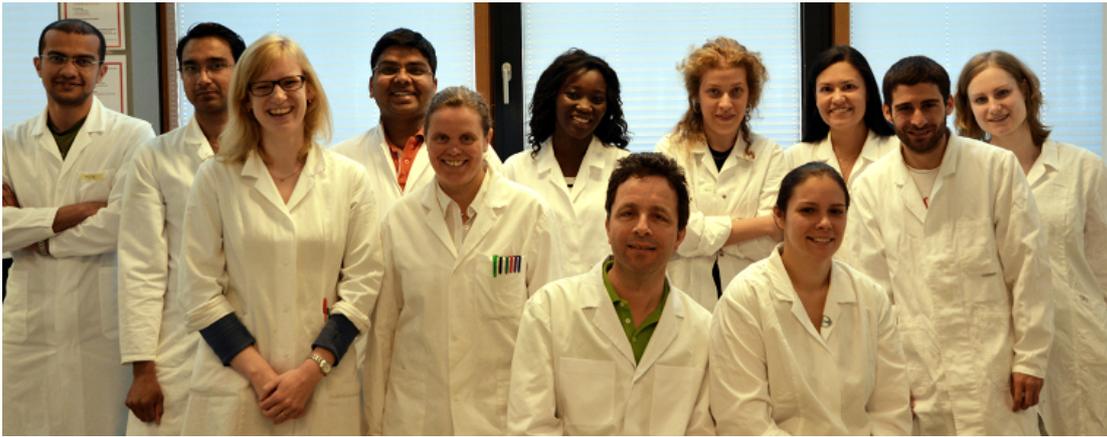
The research group of Professor Sebastian Springer (sitting in the first row) at Jacobs University Bremen. Photo: Jacobs University

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Herpesviruses are the cause of many diseases, such as shingles or mononucleosis ('kissing disease') that compromise our quality of life. While the human immune system can usually prevent the spread of herpesviruses, it cannot entirely eliminate them from our bodies. This is because these viruses contain a vast arsenal of factors that thereby conceal the virus from the immune system. These are the so-called immuno-evasins. The research group of Sebastian Springer, Professor of Biochemistry and Cell Biology at Jacobs University Bremen, have now explained the function of one such immuno-evasin.

When herpesviruses infect a cell and multiply, they can be detected by the T cells of the immune system because the infected cell will show certain proteins on its surface, the so-called MHC-I proteins. These proteins act as a warning signal: they bind other proteins on the surface of the T cell and activate it to destroy the virus-infected cell. The herpesvirus MCMV blocks this warning signal: it contains a factor, known as gp40, that prevents the transport of the MHC-I proteins to the cell surface. The virus evades the immune response, and so gp40 belongs to the class of the immuno-evasins.

The Springer group, supported by DFG and the Tönjes Vagt Foundation of Bremen, have now gathered decisive insights into how the gp40 immunoevasin achieves its function. Springer describes the processes with a simple analogy: "Imagine that gp40 has two

PhD student Venkat Raman Ramnarayan.
Photo: Jacobs University

hands. With one hand, It holds on to a fixed point within the cell, and with the other hand, it grabs the MHC-I protein. Of course now, the MHC-I protein can no longer make its way to the cell surface." Springer's PhD student, Venkat Raman Ramnarayan, has managed to identify this fixed point in the cell interior, and it turned out to be a cellular protein called TMED10, with which the Springer Lab was already familiar. "We were very well equipped to investigate the interaction between gp40 and TMED10", says Springer. "And now, we also know how the MCMV virus can escape from the immune response."

The results, which have already been published in the prestigious journal "Cell Reports", are not directly useful for patients, yet they do lay the foundation for future therapies which can strengthen and support the immune response against viral infections.

Source:

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https://www.cell.com/cell-reports/pdf/S2211-1247(18)30750-2.pdf

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