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The right messenger for a healthy immune response

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Researchers from the Molecular Immunology group at the Helmholtz Centre for Infection Research (HZI) in Braunschweig, Germany have now shown that Beta-Interferon also plays a crucial role during an immune response: without Beta-Interferon immune cells are unable to show "wanted posters" of pathogens to other cells. As a consequence, these cells will not recognize the pathogen and the immune response does not start properly. The group's results have now been published in the current issue of the scientific magazine *Journal of Immunology*.

During an infection, immune cells produce Beta-Inferferon. Interestingly, an immune response is even stronger when a low amount of Beta-Interferon has already been present before the infection occurs. Scientists call this behaviour "priming". A healthy basal level of Beta-Interferon facilitates a faster immune reaction against microbial and viral threads.

Researchers from the HZI have now managed to show why this is the case: Beta-Interferon is a key regulator and of vital importance in enabling the immune system to display fragments of pathogens, so-called antigens. Immune cells present these antigens on their surface and in this way communicate with one another: antigens are the "wanted posters" of the virus or the bacterium which has to be destroyed.

The researchers discovered the important role of Beta-Interferon in mice lacking the gene for Beta-Interferon. These mice displayed poor immune responses. "Without those knock-out mice we would not have been able

to identify the impact of Beta-Interferon on the immune system," says Siegfried Weiß, leader of the Molecular Immunology group at the HZI. His research assistant, the scientist Natalia Zietara, investigated what Beta-Interferon is doing in immune cells. She found a molecular factor that is pivotal in producing the pathogen's profile and which is regulated by Beta-Interferon. The factor belongs to a group of proteins that is usually produced in conditions of stress. Without Beta-Interferon, no active stress protein - without stress protein, no wanted poster - without wanted poster, no immune response.

"We now have a far better understanding of how immune responses start, but also how diseases like autoimmune diseases may develop," says Weiß: without Beta-Interferon, the immune system may not be able to learn how to tolerate itself during the embryonic phase and that it should not fight against self-structures. "Our findings can help to develop or improve new therapeutics to combat autoimmune diseases such as multiple sclerosis or cancer."

More information: Zietara N, Łyszkiewicz M, Gekara N, Puchałka J, Dos Santos VA, Hunt CR, Pandita TK, Lienenklaus S, Weiss S. Absence of IFN-beta impairs antigen presentation capacity of splenic dendritic cells via down-regulation of heat shock protein 70. *J Immunol*. 2009 Jul 15;183(2):1099-109.

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