# Platelets block HIV

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Scientists of the DPZ have shown that platelet activation inhibits the host cell entry of HIV

Infection biologists of the German Primate Center (DPZ) under the direction of Stefan Pöhlmann have found evidence that platelets (thrombocytes) might constitute an innate defense against infection with the human immunodeficiency virus (HIV). HI-viruses are the cause of the immune deficiency disease AIDS.

In cooperation with colleagues from the Hannover Medical School and the Institute of Molecular Virology, Ulm University Medical Center, the scientists of the DPZ have shown in a recent study that platelet activation suppresses HIV type 1 (HIV-1) infection of cell cultures and might thus reduce viral spread in patients. The paper was published in the scientific journal *Retrovirology*.

Platelets, the smallest particles of the blood, are activated through contact with the vascular connective tissue, which leads to a change of their shape and the release of biological active substances from inside the platelets. One of these substances is the messenger protein CXCL4, which blocks the host cell entry of HIV-1. This finding was reported in 2012 by Auerbach and colleagues, National Institute of Allergy and Infectious Diseases, Bethesda, USA, for purified CXCL4. The efficiency of HIV-1 inhibition by platelets in patients is currently unclear and might depend on platelet numbers and activation status.

There are two types of the HI-virus: HIV-1 and HIV-2. Additionally,

there is a closely related virus, which occurs in monkeys and apes – the simian immunodeficiency virus (SIV). The current study has demonstrated that HIV-1 is inhibited upon platelet activation while HIV-2 and SIV are not. HIV-1 is the most frequent and also the most aggressive type of HI-virus.

"Our research indicates that platelets might constitute an innate barrier against HIV-1 infection, a function that was largely unknown", says Stefan Pöhlmann, head of the Infection Biology Unit at the German Primate Center and senior author of the new study. "Further research needs to uncover how efficiently CXCL4 released by platelets inhibits HIV-1 spread in patients. Another goal should be to identify substances with a CXCL4-like antiviral activity and to develop them as novel therapies against HIV-1 infection."

**More information:** *Retrovirology* 10: 48.

Provided by German Primate Center