

## Protective Mechanism against Atherosclerosis Discovered

**Immune cells promoting inflammation play a crucial role in the development of atherosclerosis. Scientists at CeMM and the Medical University of Vienna in collaboration with the University of Cambridge showed that a survival factor for those cells has also anti-inflammatory functions and a protective role in atherosclerosis. The study, published in *Circulation*, provides valuable new insight for atherosclerosis research and suggests a hitherto unknown, inherited risk factor for atherosclerosis.**

(Vienna, June 12, 2018) Atherosclerosis, the pathological narrowing of blood vessels, is the underlying cause for the majority of strokes and heart attacks, the major causes of mortality worldwide according to the WHO. For the development of atherosclerosis, a special type of white blood cells called B2 lymphocytes have been suggested to play a crucial role. For their survival, they need the molecule BAFF. It has been shown, that deletion or blockade of the BAFF receptors at the surface of B2 lymphocytes reduces the development of atherosclerosis in mice. Hence, a similar effect was expected when BAFF is directly targeted.

With highly specific antibodies, BAFF can be bound and neutralized. Those antibodies were tested for their effects on the development of atherosclerosis in mice by scientists from CeMM Research Center for Molecular Medicine of the Austrian Academy of Sciences and the Medical University of Vienna in collaboration with the University of Cambridge. The results were surprising: instead of reducing atherosclerotic lesion formation in the arteries of the tested mice, the antibody treatment led to an increased plaque size. The findings were published in *Circulation* (DOI: 10.1161/CIRCULATIONAHA.117.032790).

“We were able to show that B2 lymphocytes were efficiently depleted upon anti-BAFF antibody treatment”, senior author of the study Christoph Binder, CeMM Principal Investigator and Professor for Atherosclerosis Research at the Medical University of Vienna, describes the results of the study. “Surprisingly, the plaque size increased, indicating a hitherto unknown role of the BAFF molecule in atherosclerosis. However, in further experiments, we were able to explain these findings.”

The researchers found BAFF to have anti-inflammatory properties, which has a positive effect on plaque size and atherosclerosis risk. The newly discovered mechanism is triggered by an alternative BAFF receptor (TACI) on the surface of macrophages, another type of immune cells. It was shown that these cells induce an anti-inflammatory process. This finding may provide

important implications for atherosclerosis research and prevention: For example, mutations in the gene for TAC1 may confer an increased cardiovascular risk.

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**The study** "BAFF Neutralization Aggravates Atherosclerosis" was published in *Circulation* on June 5, 2018 . DOI: 10.1161/CIRCULATIONAHA.117.032790

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**Christoph Binder** obtained his MD degree at the University of Vienna in 1997 and received his Ph.D. in Molecular Pathology at the University of California San Diego (UCSD) in 2002. Following a postdoctoral training period at UCSD, he joined the Institute for Laboratory Medicine of the Medical University of Vienna and became Principal Investigator at CeMM in 2006. In 2009 he was appointed Professor of Atherosclerosis Research at the Medical University of Vienna. Christoph Binder is dually affiliated with CeMM and the Medical University of Vienna.

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