| Press Release

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International Institute for Integrative Sleep Medicine, University of Tsukuba

Discovery of protein that promotes atherosclerosis

Researchers at the University of Tsukuba have discovered that the protein which regulates gene expression "MafB" exacerbates the pathogenesis of atherosclerosis by inhibiting the apoptosis of macrophages, a type of white blood cell. The team of investigators from the International Institute for Integrative Sleep Medicine, WPI-IIIS, and the Life Science Center, Tsukuba Advanced Research Alliance (TARA), University of Tsukuba, includes Michito Hamada, Megumi Nakamura and Satoru Takahashi

In atherosclerosis, lipids such as oxidized cholesterol accumulate where scratches have formed in the inside walls of arterial blood vessels. To remove the buildup, macrophages come from the blood to the site and gather. This condition leads to the narrowing of the arteries. However, until now, not much was known about the mechanism whereby macrophages remain in the vessel walls.

In their study published in Nature Communications, the research group succeeded in clarifying the inhibitor of macrophage apoptosis where MafB inside the macrophage receives a signal from the oxidized cholesterol. At the same time, by maintaining the function of MafB with atherosclerotic lesions in atherosclerosis mouse models, the atherosclerotic pathology has been significantly improved when macrophage apoptosis is induced. In addition, MafB was revealed to directly regulate the gene expression of the apoptosis inhibitor of macrophages (AIM), shedding light on this detailed mechanism. These results are expected to lead to the founding of new atherosclerosis treatment development.

In collaboration with the University of Tsukuba's Hitoshi Shimano, the University of Tokyo's Toru Miyazaki, and the University of California's Peter Tontonoz, this research was published online in Nature Communications on January 20, 2014 (19:00 Japan Time).

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Publication Information

“MafB promotes atherosclerosis by inhibiting foam cell apoptosis”
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