Mechanism of AAA rupture and its potential prevention strategy

Abdominal aortic aneurysm (AAA) is a fatal disease that involves gradual dilation of the abdominal aorta followed by its rupture. However, the mechanism of AAA rupture is not fully understood yet. Although AAA is one of the major causes of death in many countries, there is no drug for its treatment. Previous studies demonstrated that inflammation in the vascular wall is associated with AAA rupture. However, at present, drugs used for the treatment of vascular inflammation are not effective in preventing AAA rupture.

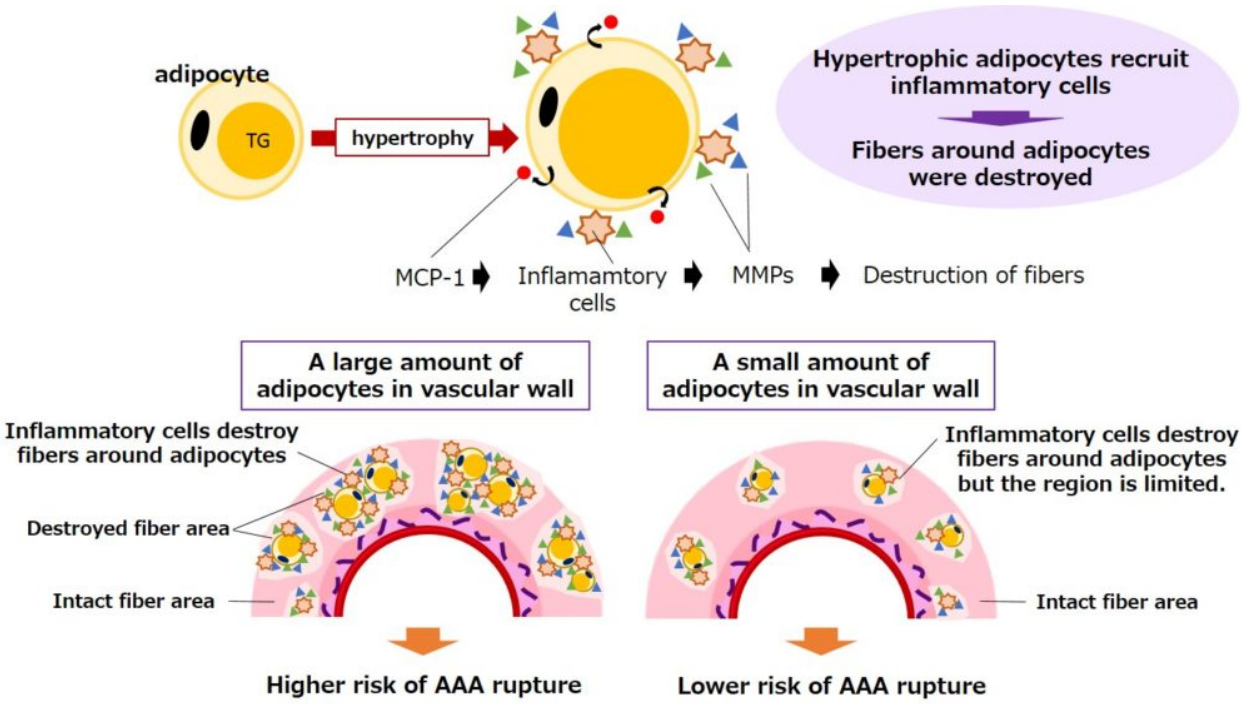

Fig. 1. Potential mechanism of AAA rupture induced by adipocytes

Using our newly established AAA animal model (vascular hypoperfusion-induced model), we recently showed that the abnormal appearance of adipocytes in the vascular wall can cause AAA rupture. The AAA formed in this animal model spontaneously rupture. When triolein, a triglyceride (TG), was administrated to this AAA animal model, the incidence of AAA rupture was significantly increased and this was due to an abnormal increase in the number of adipocytes in the vascular wall. Figure 1 represents our hypothesis for the mechanism of AAA rupture. The collagen fiber in vascular adventitia plays a role in maintaining the normal vascular diameter. Replacement of collagen fiber or collagen-producing cells with adipocytes causes weakening of the arterial wall. In addition, inflammation can be induced around adipocytes by factors secreted by them (known as

adipocytokines or adipokines). This induced inflammation can cause the degradation of collagen fiber around adipocytes. Thus, this vicious circle can increase the risk of AAA rupture. We found that in human AAA tissue, the abnormal appearance of adipocytes and the number of adipocytes in vascular wall were correlated with AAA diameter. This suggests that abnormal adipocytes can cause AAA rupture in humans as well as in the AAA animal model. Eicosapentaenoic acid (EPA)-rich fish oil (a TG) has suppressive effects on de novo TG synthesis and inflammation. We found that EPA-rich fish oil administered group had a decrease in the number of adipocytes in the vascular wall as well as a decrease in the risk of AAA rupture ratio by 0.23 compared to the triolein administered group.

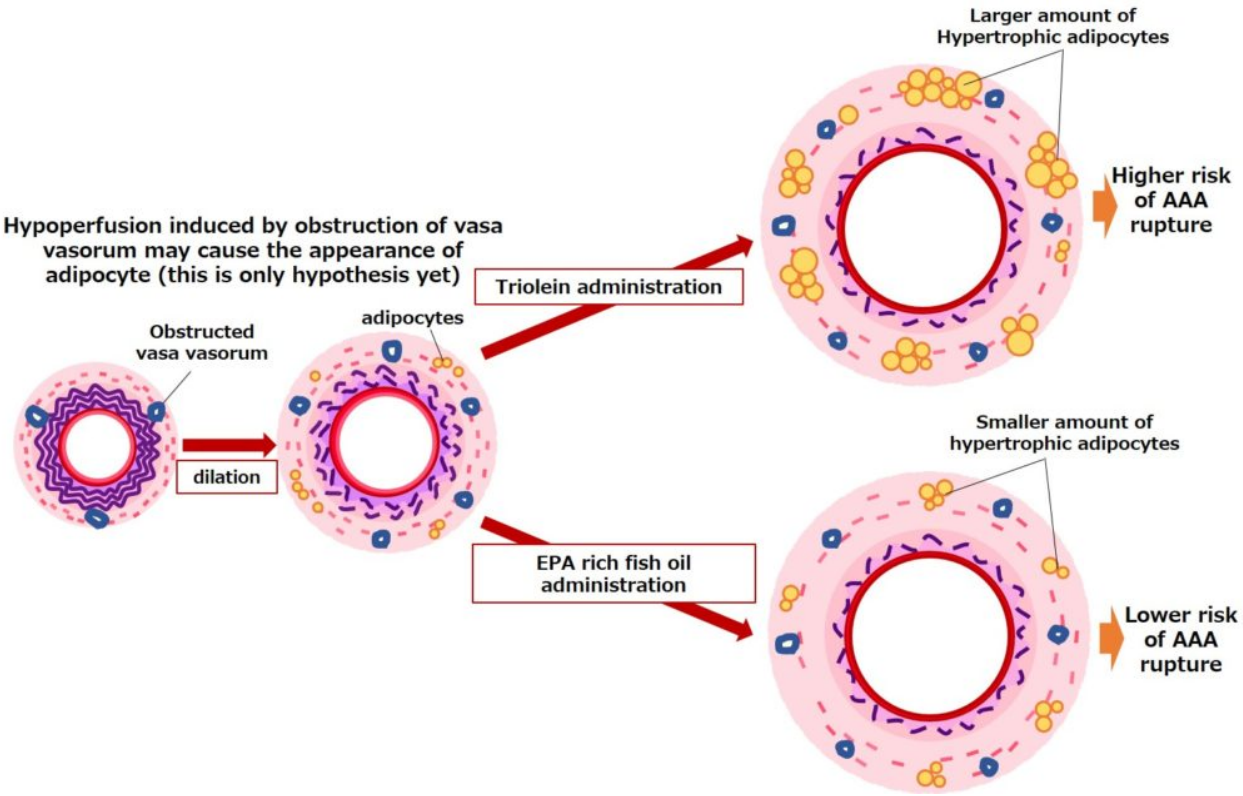

Fig. 2. The relationship between adipocytes and AAA rupture.

Thus, our studies raise the possibility that adipocytes in vascular adventitia can become an important target of drugs or functional food factors aimed at preventing AAA rupture.

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Publication

Adipocyte in vascular wall can induce the rupture of abdominal aortic aneurysm.
Kugo H, Zaima N, Tanaka H, Mouri Y, Yanagimoto K, Hayamizu K, Hashimoto K, Sasaki T, Sano M, Yata T, Urano T, Setou M, Unno N, Moriyama T
Sci Rep. 2016 Aug 8