**ROLE OF IGE IN ANGIOTENSIN II-INDUCED ABDOMINAL AORTIC ANEURYSMS IN APOLIPOPROTEIN E-DEFICIENT MICE**

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Immunoglobulin E (IgE) binds to its high affinity receptor Fc epsilon receptor 1 (Fc&#949;R1) and activates mast cells, which have been suggested as important inflammatory cells in human and mouse abdominal aortic aneurysms (AAAs). The current study tested whether IgE has a role in angiotensin II (Ang II) perfusion-induced abdominal aortic aneurysms. After 28 days of Ang II perfusion in apolipoprotein

E-deficient (Apoe–/–) mice, these mice demonstrated three-fold increase of serum IgE levels. Absence of Fc&#949;R1 alpha-chain in Apoe–/– mice (Apoe–/–Fcer1a–/–) reduced both AAA incidence (from 83.3% to 33.3%) as well as aortic diameters (from 2.46±0.16 mm to 1.58±0.27 mm, P=0.006), compared with those from Apoe–/–Fcer1a+/+ littermate control mice, along with reduced inflammatory cell infiltration, cell proliferation, angiogenesis, apoptosis, and medial smooth muscle cell loss. Reduced AAA formation in Apoe–/–Fcer1a–/– mice can be greatly reversed after mice receiving peritoneal macrophages isolated from Apoe–/–Fcer1a+/+ mice, but not those from Apoe–/–Fcer1a–/– mice, suggesting a role of macrophage activation by IgE in mouse AAA pathogenesis. AAA formation and lesion inflammation, including macrophage and T cell accumulation and lesion chemokine expression, in Apoe–/–Fcer1a+/+ mice were fully abolished when mice received two doses of an anti-IgE antibody, implicating a potential therapy of human AAA by anti-IgE antibody treatment. Therefore, IgE may contribute to human and mouse AAA formation by activating not only mast cells, but also macrophages. Anti-IgE antibody therapy may be effective among AAA patients.