blot, respectively. In vitro, the expression of NFATc1 mRNA and protein in cultured lymphocytes of ApoE $^{-/-}$  mice was measured by RT-PCR and flow cytometry, respectively. The level of IL-2, IL-6, TNF- $\alpha$  and IFN- $\gamma$  was measured by RT-PCR.

**Results** We found that the expression of NFATc1 was significantly increased both in atherosclerotic lesion and in leukocytes from ApoE $^{-/-}$  mice. In vitro and vivo, after stimulating CD137-CD137L interaction, the expression level of NFATc1 mRNA and protein was significantly increased in lymphocytes, while anti-CD137L mAb significantly suppressed the expression of NFATc1 in leukocytes. Moreover substantially elevated levels of IL-2, IL-6, TNF- $\alpha$  and IFN- $\gamma$  were induced by anti-CD137 mAb, while NFATc1 inhibitor markedly suppressed production of IL-2, IL-6, TNF- $\alpha$  and IFN- $\gamma$ .

**Conclusions** This study suggested that CD137-CD137L interactions can regulate the expression of NFATc1 in Apo $E^{-/-}$  mice, which may play an important part in atherosclerotic plaque formation.

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## THE EFFECT OF CD137-CD137 LIGAND INTERACTION ON THE EXPRESSION OF NFATC1 IN APOLIPOPROTEIN E-DEFICIENT MICE

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**Objectives** Increasing evidence shows that the interaction of CD137-CD137L plays an important role in atherosclerosis. However, the mechanism of CD137-CD137L interaction contribution towards pathogenesis has been poorly understood. The aim of this study was to investigate the effect of CD137-CD137L interaction on the nuclear factor of activated T cells c1 (NFATc1) in  $\rm ApoE^{-/-}$  mice.

**Methods** Atherosclerotic plaque model was produced by rapid perivascular carotid collar placement in ApoE<sup>-/-</sup> mice. In vivo, the expression of NFATc1 in mice plaque and lymphocytes was detected by immunohistochemical, flow cytometry and Western

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