ACTIVATED LIPID ENDOCANNABINOID SIGNALING IN ATHEROSCLEROSIS: DRIVING FORCE OR PROTECTIVE MECHANISM?

GUEST LECTURE by

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Potential therapeutic targets of CB2 activation in cardiovascular disorders.


CB2 antagonism inhibits accelerated neointima formation in FAAH-deficient mice.

Fatty acid amide hydrolase (FAAH) deficiency affects plaque quality.


CB2 antagonist inhibits accelerated neointima formation in ApoE-/- mice.