Doctoral College
Metabolic & Cardiovascular Disease

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

GUEST LECTURE by

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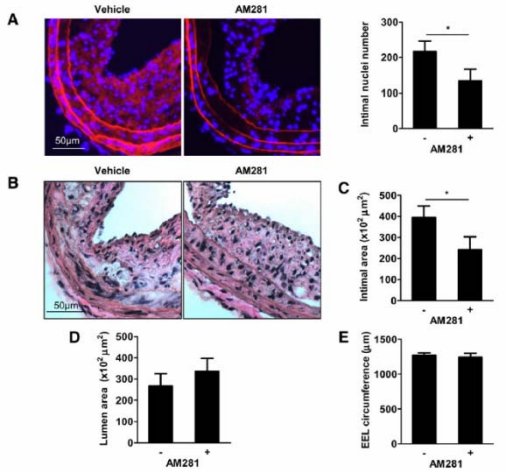
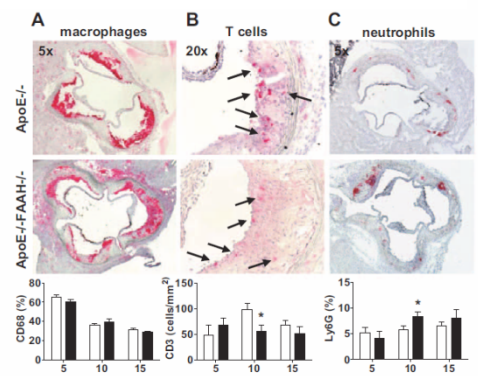
Monday, 13.10.2014
17:00

HS 07.03, Preclinics
Harrachgasse 21, MUG

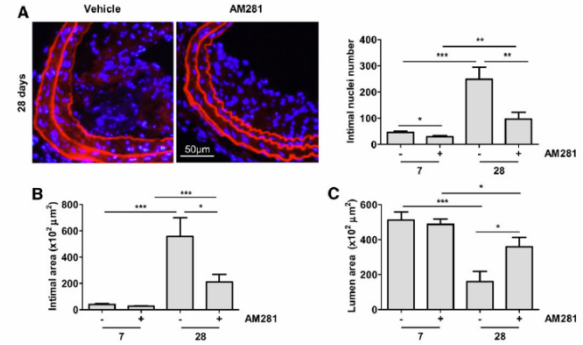


CB_1 antagonism inhibits accelerated neointima formation in FAAH-deficient mice.



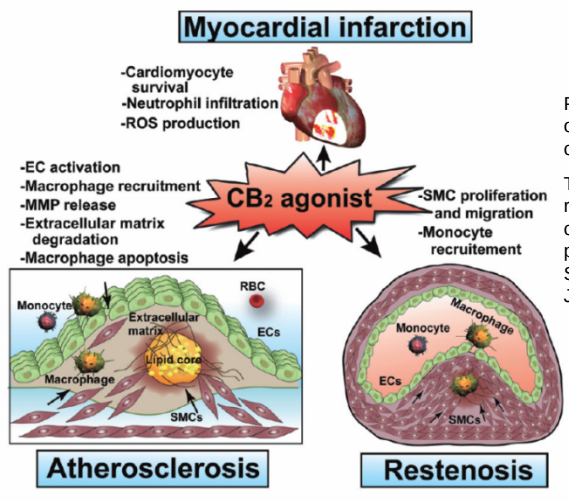
Fatty acid amide hydrolase (FAAH) deficiency affects plaque quality.

Fatty acid amide hydrolase deficiency enhances intraplaque neutrophil recruitment in atherosclerotic mice. Lenglet et al. (2013) Arterioscler Thromb Vasc Biol 33:215-23



CB_1 antagonism inhibits accelerated neointima formation in $ApoE^{-/-}$ mice.

Endogenous cannabinoid receptor CB_1 activation promotes vascular smooth-muscle cell proliferation and neointima formation. Molica et al. (2013) J Lipid Res 54:1360-8



Potential therapeutic targets of CB_2 activation in cardiovascular disorders.

Targeting cannabinoid receptor CB_2 in cardiovascular disorders: promises and controversies. Steffens & Pacher (2012) Brit J Pharmacol 167:313-23