Atherosclerosis 263 (2017) e1 e28

Contents lists available at ScienceDirect

Atherosclerosis

journal homepage: www.elsevier.com/locate/atherosclerosis

EAS 2017 CONGRESS ABSTRACTS PRESENTATIONS

WORKSHOP 1.1 Progression and regression of atherosclerosis

W1.1:1.
LEUKOCYTE ABCA1 IMPEDES PROGRESSION OF ESTABLISHED ATHEROSCLEROTIC LESIONS AFTER DIETARY CHOLESTEROL LOWERING IN LDLR / MICE

Olga Snip, Janine Geerling, Miranda Van Eck. *Leiden Academic Centre for Drug Research, Leiden, The Netherlands*

Aim: The ATP binding cassette transporter A1 (ABCA1) facilitates the efflux of cholesterol and phospholipids to lipid free apolipoprotein A1 and small dense high density lipoproteins. Various studies have shown that leukocyte ABCA1 is an anti atherogenic factor. Dietary cholesterol lowering stabilizes atherosclerotic lesions, however the role of ABCA1 in this process is unknown. Therefore, this study aims to investigate the effect of leukocyte ABCA1 on diet induced atherosclerotic lesions after with drawal of the atherogenic diet.
Methods: Leukocyte ABCA1 was studied by transplanting bone marrow cells from donor mice that were either knock out (ABCA1 /) or wild type for ABCA1 or that overexpressed ABCA1 (ABCA1Tg) into low density li poprotein receptor knock out (LDLr /) mice. All three groups of chimeric mice were fed a Western type diet (WTD: 0.25%cholesterol, 15%cacao butter) for 6 weeks to induce atherosclerotic lesion development. After this period, a baseline group was sacrificed (ABCA1 / >LDLr / n=12; wild type >LDLr / n=14; ABCA1Tg >LDLr / n=9) for lesion assessment. The remainder of chimeric mice was switched to a chow diet (low fat, no added cholesterol) for 3 weeks to lower plasma cholesterol levels (ABCA1 / >LDLr / n=14; wild type >LDLr / n=14; ABCA1Tg >LDLr / n=12).
Results: Withdrawal of the atherogenic diet normalized plasma choles terol levels in all three groups. As a result, lesion development was sta bilized in both the wild type >LDLr / and ABCA1Tg >LDLr / mice, however not in the ABCA1 / >LDLr / mice, which displayed a 1.5 fold increase ($p<0.01$) in atherosclerotic lesion size.
Conclusions: Leukocyte ABCA1 is required to halt atherosclerotic lesion progression after dietary cholesterol lowering. Overexpression of ABCA1 did not result in any additional beneficial effects.

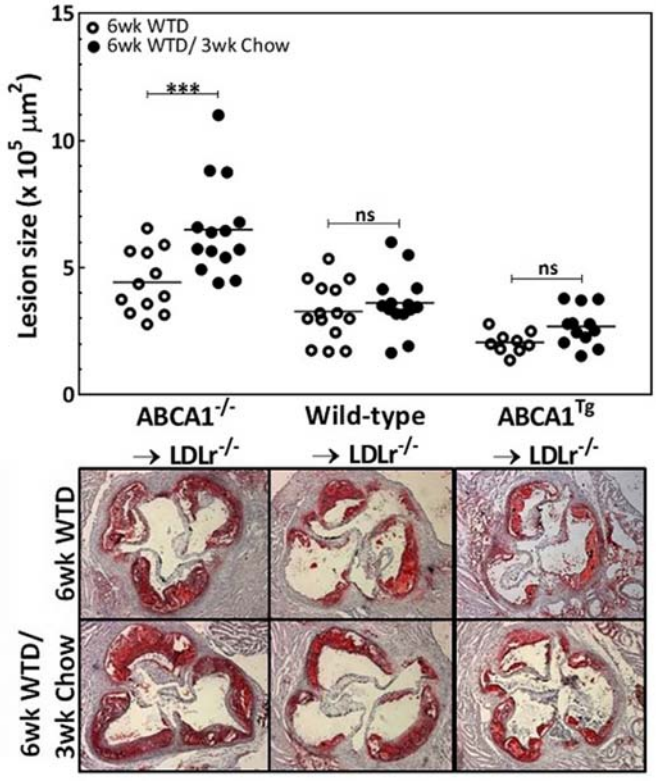

Fig. 1. Lesion size quantification of Oil Red O stained atherosclerotic lesions in the aortic roots.

W1.1:2.
NON TOXIC CONCENTRATIONS OF CADMIUM ACCELERATE SUBENDOTHELIAL RETENTION OF ATHEROGENIC LIPOPROTEINS IN HUMANIZED ATHEROSCLEROSIS SUSCEPTIBLE MICE

Siavash Kijani, Göran Bergström, Malin Lindbom, Malin Levin, Lars Barregård, Björn Fagerberg, Per Fogelstrand, Jan Borén. *Department of Molecular and Clinical Medicine, University of Gothenburg and Sahlgrenska University Hospital, Gothenburg, Sweden*

Aim: Cadmium is an important risk factor for cardiovascular disease (CVD). However, the underlying mechanism(s) are unclear. Cadmium has been shown to increase proteoglycan synthesis in vitro. Because pro teoglycans mediate a critical step in atherosclerosis by trapping apoB containing lipoproteins in the artery wall, we hypothesized that cad mium can increase proteoglycan mediated LDL binding and thereby promote subendothelial LDL accumulation. Here we tested the