a semiquantitative scale after the following stainings: hematoxylin eosin (for assessment of intra- and extracellular lipids and for assessment of thrombus formation), Elastica van Gieson, anti-CD31 for assessment of vessel density, anti-CD68, anti-CD3 and anti-CD15 for analysis of local inflammation, anti-SMC actin to stain smooth muscle cells, anti-CD61 for platelet depositions, TUNEL staining for apoptotic cells.

Results: Lipid and foam cell content was most pronounced in aortic lesions (P = 0.02, P = 0.04), calcification and SMC actin in femoral lesions (P = 0.001, P = 0.001), CD3+ cells in carotid lesions (P = 0.004) and TUNEL+ apoptotic cells in coronary lesions (P = 0.01). Thrombus was most prevalent in carotid and aortic lesions (P = 0.02). Neutrophil (CD15+) and monocyte (CD68+) cell infiltrations as well as vessel density were most pronounced in carotid and femoral lesions (P = 0.04, P = 0.02, P = 0.005). No significant changes were observed in vessel hemorrhage or platelet content. These results suggest more inflammatory lesions in carotid and femoral arteries with an increase in calcification and SMC content in femoral lesions as compared to more lipid rich lesions within the aorta and an increase in apoptotic cells within coronary lesions. Additionally, gene expression of atherosclerosis related genes in carotid and femoral endarterectomy specimen was measured. An overall increased metabolism was found in femoral plaques, especially genes concerning inflammatory response, response to stress, apoptosis, adhesion, whereas in carotid lesions, we detected overexpression of genes related to lipid/cholesterol transport and metabolism and negative transcription regulators.

Conclusions: Different arterial territories are differently affected by atherosclerosis: Our results suggest more inflammatory lesions in carotid and femoral arteries with an increase in calcification and SMC content in femoral lesions as compared to more lipid rich lesions within the aorta and an increase in apoptotic cells within coronary lesions. Concerning PAD, gene expression in atherosclerotic lesions shows significant differences. Understanding of the local differences of atherosclerosis may aid to improve prevention and lead to targeted treatment of atherosclerosis and its complications.

TCT-819
Healing of a Combination Sirolimus-Eluting, Endothelial Progenitor Cell Capture Stent Compared to an Everolimus-Eluting Stent in an Atherosclerotic Rabbit Model
Fumiya Otsuka1, Erica Pacheco1, Kenichi Sakakura1, Kazuyuki Yahagi1, TCT-819

Complications
Our results suggest more inflammation and stent thrombosis in carotid and femoral endarterectomy specimen was measured. An overall increased metabolism was found in femoral plaques, especially genes concerning inflammatory response, response to stress, apoptosis, adhesion, whereas in carotid lesions, we detected overexpression of genes related to lipid/cholesterol transport and metabolism and negative transcription regulators.

Conclusions: Different arterial territories are differently affected by atherosclerosis: Our results suggest more inflammatory lesions in carotid and femoral arteries with an increase in calcification and SMC content in femoral lesions as compared to more lipid rich lesions within the aorta and an increase in apoptotic cells within coronary lesions. Concerning PAD, gene expression in atherosclerotic lesions shows significant differences. Understanding of the local differences of atherosclerosis may aid to improve prevention and lead to targeted treatment of atherosclerosis and its complications.

TCT-820
First Approach to Stimulate Arteriogenesis using Monoclonal Antibodies: Blocking the Interferon-alpha/beta Receptor Subunit 1 Stimulates Restoration of Perfusion in a Murine Hindlimb-ischemia Model Without Affecting Atherosclerosis
Paul F. Toussaint1, Marieke C. Boshuizen1, Maurits Hollander1, Nina W van der Hoeven1, Marion Gijbels1, Menno P de Winther2, Anton J. Horrevoets1, Niels van Royen1

Background: Increased expression of interferon (IFN)-beta was shown in patients with insufficient coronary collaterals. Furthermore, mice treated with IFN-beta demonstrate inhibition of collateral artery growth (arteriogenesis). Interestingly, type 1 interferons (IFN-alpha and IFN-beta) have been identified as proatherosclerotic cytokines and in mouse models of atherosclerosis, IFN-beta treatment accelerated lesion formation and increased accumulation of macrophages in plaques. We hypothesized that arteriogenesis can be stimulated using monoclonal antibodies inhibiting IFN-beta signaling without accelerating atherosclerosis.

Methods: In an atherosclerotic murine hindlimb-ischemia model, LDLR-/– mice were treated during a 4-week period with monoclonal antibodies specific for mouse Interferon-alpha/beta Receptor subunit 1 (IFNAR-1) or a murine IgG1 isotype as control. Hindlimb perfusion was measured using laser Doppler perfusion imaging (LDP) directly after femoral artery ligation as well as at 2, 7, 14 and 28 days following ligation. We used a disease model to investigate effects of anti-IFNAR-1 on arteriogenesis, which was evaluated with histology to determine plaque area and composition.

Results: Hindlimb perfusion restoration after femoral artery ligation was improved in mice treated with anti-IFNAR-1 compared to controls as assessed by LDI at 7, 14 and 28 days (treatment vs. control; 7 days: 35.6 ± 16.5% vs. 23.6 ± 8.7%, p = 0.027; 14 days: 51.4 ± 17.2% vs. 35.0 ± 12.3%; p = 0.010; 28 days 71.5 ± 13.8% vs. 54.0 ± 16.3%; p= 0.005). Total plaque area (treatment vs. control: 118.8 ± 46.1±10 m2 vs. 139.3 ± 46.5 ±10 m2, p = 0.275) as well as composition were unaltered.

Conclusions: Blocking IFNAR-1 using monoclonal antibodies stimulates collateral artery growth in mice and has a neutral effect on atherosclerosis.