Results: The mean overlap length was 5.8±1.8 mm. There were no quantitative changes in IVUS measurements within the overlap segment (Table), and there was no case of late malapposition.

Post 1-year FU P value
External elastic membrane volume (mm³) 61.8 ± 14.6 58.2 ± 13.6 0.6
Lumen volume (mm³) 38.1 ± 14.3 36.9 ± 14.5 0.8
Stent volume (mm³) 38.1 ± 14.3 38.1 ± 14.3 1.0
Plaque volume within the stent (mm³) 23.8 ± 5.6 20.1 ± 5.7 0.2
Intimal hyperplasia volume (mm³) 1.1 ± 0.4 NA
% intimal hyperplasia 3.4 NA

Conclusions: There appears to be no deleterious effect on the arterial wall - quantitative changes in vessel wall geometry or late malapposition - when overlapping Sirolimus-eluting stents are implanted to treat ISR lesions.

Methods: The expression of RANK, RANK-L and FKBP12 was detected by immunohistochemistry in rat carotid arteries at 7 and 28 days post balloon angioplasty (n=6 per time). Ox-62 and St100 immunostaining was used to identify DCs. In addition, neointimal cell types and apoptosis were characterized by transmission electron microscopy (TEM). Results: At day 7, the vast majority of neointimal cells were identified as DCs by Ox-62 and St100 immunostaining. TEM demonstrated cells with typical ultrastructural features of apoptosis which were co-localized with RANK-L. FKBP12 revealed similar expression profiles as those of Ox-62 and St100. In serial sections, extensive immunoreactivity for FKBP12 was frequently found with neointimal cells at this time point. However, at day 28, neointimal signaling for RANK and its ligand was present around the luminal surface, coincident with Ox-62 and St100 expression. Accordingly, TEM analysis revealed DCs exclusively in the apical neointima, while basal areas contained smooth muscle-like cells. Neointimal apoptosis was <1% at this time. Immunoreaction of FKBP12 was detectable in luminal neointima with DC aggregation. Of note, neointimal and adventitial cytokine profiles revealed no immunolabeling for RANK, RANK-L, FKBP12 or DCs at both time points.

Conclusions: The present data identify viable DCs as novel cellular component involved in early neointima formation, and suggest DC-specific RANK expression, co-localized with its ligand RANK-L, to promote their survival. Since neointimal DCs strongly express FKBP12, rapamycin may selectively interfere with neointimal DC accumulation post angioplasty.

**1031-175 Chronic Stent-Induced Injury and Inflammation Results in Sustained Activation of the Cell Cycle In the Porcine Model**

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The long-term processes of arterial repair are incompletely characterized though believed to be completed by 28 days in the porcine coronary model. The purpose of this study was to evaluate the ontogenic vascular response to balloon arterial injury on regulatory proteins of the cell cycle and to provide correlation with histological events. Methods: Fifty-three pigs underwent placement of 54 stents in the coronary arteries with histologic and Western blot (PCNA and CDC2) analysis at 3, 8, 12, 28 and 112 days. Histological evaluation included vessel injury, stented associated inflammation and morphometric analysis of the arterial wall. Results:

**POSTER SESSION**

1031 Restenosis Prevention

Sunday, March 30, 2003, Noon-2:00 p.m.
McCormick Place, Hall A
Presentation Hour: Noon-1:00 p.m.

1031-173 Receptor Activator of NF-kB (RANK) and Its Ligand RANK-L Coincide With Expression of Rapamycin Receptor FKBP12 in Neointimal Dendritic Cells

Alexander Jahn, Dirk Skowasch, Margaret Forney Presscott, Bernd Lüderitz, Gerhard Bauriedel, University of Bonn, Bonn, Germany, Cardiovascular Disease Research, Novartis Comp., Summit, NJ

Background: We recently reported the presence of dendritic cells (DCs) in postangioplasty neointima and suggested their circulatory origin. The aim of the present study was to assess the mode of DC survival at the angioplasty site. Specifically, we sought to analyze the presence of RANK whose activation by TNF-related ligands like RANK-L mediates anti-apoptotic effects promoting DC survival. In addition, we assessed the expression of FKBP12, the primary intracellular rapamycin receptor, as rapamycin may induce DC apoptosis.