

	1st angio (%)	2nd angio (%)	Δ (%)	1st angio (mm)	2nd angio (mm)	Δ (mm)
IRS	66.2 ± 6.6	72.8 ± 16.9*	6.6 ± 15.8	1.15 ± 0.28	0.91 ± 0.52*	0.24 ± 0.47
non-IRS	44.6 ± 14.5	48.0 ± 15.8*	3.4 ± 12.2 <sup>‡</sup>	1.71 ± 0.50	1.61 ± 0.53*	0.10 ± 0.31 <sup>‡</sup>
Com IRS	66.9 ± 7.1	76.5 ± 16.3*	9.6 ± 17.2	1.13 ± 0.28	0.79 ± 0.54*	0.34 ± 0.51
Smo IRS	64.8 ± 5.3	65.5 ± 12.1	0.71 ± 10.6 <sup>†</sup>	1.18 ± 0.27	1.13 ± 0.38	0.05 ± 0.30 <sup>†</sup>

Data are mean ± SD. \*p < 0.01 vs. 1st angio by paired t-test; <sup>‡</sup>p < 0.05 vs. IRS, and <sup>†</sup>p < 0.01 vs. Com IRS by unpaired t-test. Com = complex, Smo = smooth

Despite clinical stabilization, IRS are unstable and at high risk for rapid progression to total occlusion, which is usually associated with coronary events.

5:15

**768-6 Lipid Rich Plaques with Thrombus are Common in Unstable Rest Angina: Observations from Atherectomy Tissue Analysis**

Samin K. Sharma, Billie Fyfe, Ram Bongu, Jeremy Asnes, Jonathan D. Marmor, Thomas Cocke, Orlando D. Almeida, John A. Ambrose. *Mount Sinai Hospital, New York, N.Y.*

Several autopsy studies indicate that disrupted atherosclerotic plaques particularly those rich in lipid with associated thrombus, are the cause of acute MI. Other plaques without disruption are fibrocellular without lipid. Although unstable rest angina (UA) shares a common pathogenesis to acute MI, there are little pathological data on plaque composition in UA. Directional Coronary Atherectomy (DCA) provides the opportunity to study plaque composition in UA. *Methods:* We prospectively analyzed the DCA tissue specimens of 60 pts with *de novo* culprit lesions for fibrocellular and lipid constituents by hematoxylin & eosin and oil red O stains. A fibrous plaque (FP) was defined as one which had predominantly fibrous and sclerotic tissue with no or minimal lipid (0 or 1+ on scale of 3). A lipid plaque (LP) was defined as one with moderate or high lipid (2 or 3+) content. The presence of inflammatory cells and thrombus were also noted. Histopathology was analyzed independent of clinical presentation.

Clinical syndrome	FP (%)	LP (%)	p
Stable/asymptomatic (10)	9 (90)	1 (10)	NS
New or Crescendo (16)	9 (56)	7 (44)	
Rest Angina (21)	4 (19)	17 (81)	NS
Post-MI (13)	2 (15)	11 (85)	

} <0.01

Coronary thrombus on histologic analysis was present in 23/36 (64%) of LP vs. in 5/24 (21%) of FP (p < 0.01). Coronary thrombus was present in 88% and 82% of LP in rest angina and post MI respectively. Inflammatory cells were noted in 12 LP and 2 FP (p = 0.02) — in 38% of both rest angina and post MI specimens. *Conclusions:* Lipid rich plaque are very common in rest angina and post MI, moderately common in crescendo or new onset and rare in stable angina. LP are usually associated with thrombus and inflammation particularly in rest angina and post MI pts. These DCA tissue analyses confirm and expanded on prior autopsy studies in acute syndromes and support the pathogenetic link between unstable rest angina and acute MI.

**769 The Effect of Lipid-Lowering Drugs on Coronary Anatomy and Myocardial Ischemia**

Tuesday, March 21, 1995, 4:00 p.m.–5:30 p.m.  
Ernest N. Morial Convention Center, Room 102

4:00

**769-1 The Influence of Pravastatin on Progression and Regression of Coronary Atherosclerosis in Men with Normal or Mildly Raised Serum Cholesterol. Results of the Regression Growth Evaluation Statin Study (REGRESS)**

Albert V.G. Brusckha, J. Wouter Jukema, Ad J. van Boven, Johan H.C. Reiber, Egbert T. Bal, Aeiko H. Zwinderman, Kong I. Lie, REGRESS study group *Interuniversity Cardiology Institute, Utrecht, The Netherlands*

REGRESS is a double blind, placebo controlled multicenter study to assess the effect of 2 year treatment with pravastatin 40 mg once daily on pro- and regression of angiographically documented coronary artery disease in 885 patients with a serum cholesterol between 4–8 mmol/l (155 and 310 mg/dl). The REGRESS study comprises three blocks of patients: a percutaneous coronary angioplasty, a coronary artery bypass grafting and a medical management group. Analysis of baseline and follow-up coronary arteriograms was performed visually and by quantitative computer analysis (QCA). Primary

endpoints were QCA assessments of 1. change in Mean Segment Diameter (MSD) averaged per patient and 2. change in Minimum Obstruction Diameter (MOD) averaged per patient.

*Results:* 778 patients (88%) of the patients had an evaluable final angiogram. Mean Segment Diameter decreased with 0.10 mm in the placebo group versus 0.06 mm in the pravastatin group (p = 0.019): the mean difference between treatment groups was 0.04 mm with 95% confidence interval (ci) of 0.01–0.07 mm. The median Minimum Obstruction Diameter decreased with 0.09 mm in the placebo group versus 0.03 mm in the pravastatin group (p = 0.001): the difference of the medians between the treatment groups was 0.06 mm with ci of 0.02–0.08 mm. At the end of the follow-up period 89% (ci 86–92%) of the pravastatin patients and 81% (ci 77–85%) of the placebo patients were without new cardiovascular events (p = 0.002).

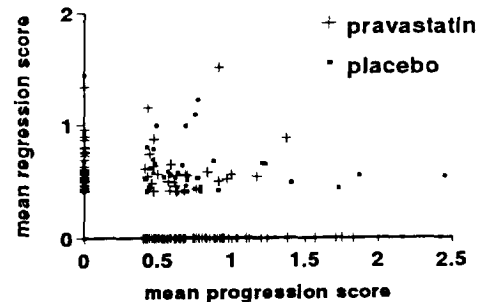
*Conclusions:* in symptomatic men with normal or moderately raised serum cholesterol, pravastatin slows progression of coronary atherosclerosis and reduces the number of cardiovascular events.

4:15

**769-2 Progression and Regression of Coronary Atherosclerosis Occur within the Same Patient During Placebo Treatment and During Lipid-Lowering Therapy with Pravastatin**

J. Wouter Jukema, Ad J. van Boven, Johan H.C. Reiber, Aeiko H. Zwinderman, Albert V.G. Brusckha, REGRESS study group *Interuniversity Cardiology Institute, Utrecht, The Netherlands*

REGRESS (Regression Growth Evaluation Statin Study) is a placebo controlled multicenter study to assess the effect of 2-yr treatment with Pravastatin (PRAV) on progression and regression of angiographically documented coronary atherosclerosis (CA) in patients with a serum cholesterol between 4–8 mmol/l (155–310 mg/dl). Analyses of the coronary arteriograms were performed by quantitative computer analysis. The primary endpoints of the study, change in Mean Segment Diameter and Minimum Obstruction Diameter (MOD) averaged per patient, showed significant retardation of mean progression of CA in the PRAV-group as compared to the placebo (PLAC)-group. However, these mean changes per treatment group are hardly informative about individual CA-behavior. Therefore we determined for all 641 patients included in the primary MOD-analysis: 1. a mean progression score (MPS)-cumulative value of all >0.4 mm progressing obstructions divided by the number of contributing obstructions-, and 2. a mean regression score (MRS)-cumulative value of all >0.4 mm regressing obstructions divided by the number of contributing obstructions. Obstructions changing ≤0.4 mm were considered stable and do not contribute to the scores. Thus, each patient is characterized by a MPS and a MRS. An overview of the patient MPS and MRS is presented in the figure below.



*Conclusion:* significant progression and regression of CA within the same patient occurred in 41 (13%) PRAV-patients and in 27 (9%) PLAC-patients. Thus, although pravastatin slows mean progression of CA, progression and regression of CA within the same patient still occurs in a considerable number of patients during lipid lowering therapy.

4:30

**769-3 Lipid Intervention and Progression of Coronary Atherosclerosis**

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The *Coronary Intervention Study, CIS*, is a multicenter, randomized, double-blind, placebo-controlled study to investigate the effects of lipid-modifying therapy on progression in 254 men with documented coronary artery disease (CAD) and hypercholesterolemia. *Entry criteria* were the presence of at least 3 coronary stenoses of ≥25% and a mean serum total cholesterol

TUESDAY PM