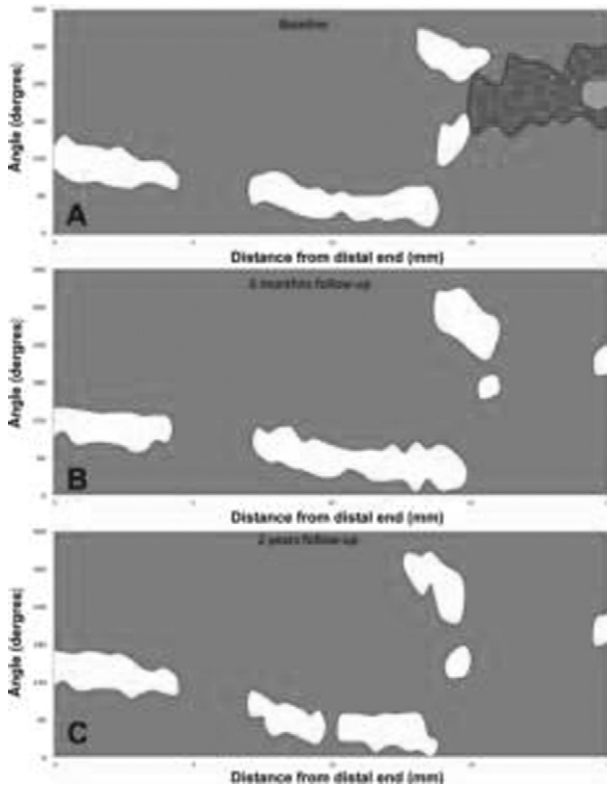

Results: The extent of the calcific tissue did not change at follow-up (4.08±4.31% at baseline vs. 3.41±4.05% at 6 months and 4.05±4.05% at 2 years, P=0.394) while the extent of the lipid component decreased (17.57±8.24% at baseline vs. 8.08±6.71% at 6 months and 2.69±3.62% at 2 years, P<0.001). The thickness of the overlaying tissue increased at follow-up in both the lipid cores (by 104μm at 6 months and by 157μm at 2 years comparing to baseline; P<0.001) and calcific spots (by 76μm at 6 months and by 105μm at 2 years comparing to baseline, P=0.001).
Conclusions: It appears that in BRSs the plaque type does not affect neointimal formation which can cover both lipid and calcific tissues.

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Impact of Stent Edge Plaque Burden on Lumen Preservation After DES Implantation: A 3D-IVUS Analysis from the J-DESsERT Trial

Hideki Kitahara[1], Katsuhisa Waseda[1], Kenji Sakamoto[1], Ryotaro Yamada[1], Ching-Chang Huang[1], Daisaku Nakatani[1], Kenji Sakata[1], Osami Kawarada[1], Paul Yock[1], Yutaka Matsuyama[2], Hiroyoshi Yokoi[3], Masato Nakamura[4], Toshiya Muramatsu[5], Shinsuke Nanto[6], Peter Fitzgerald[7], Yasuhiro Honda[8]
[1]Stanford University Medical Center, Stanford, CA, [2]University of Tokyo, Tokyo, Japan, [3]Kokura Memorial Hospital, Kitakyushu, Japan, [4]Toho university, Ohashi Medical Center, Tokyo, Japan, [5]Saiseikai Yokohama-city Eastern Hospital, Yokohama, Japan, [6]Osaka University, Suita, Japan, [7]Stanford University Medical Center, Stanford, California, [8]Stanford University, Stanford, USA

Background: Previous studies have recommended positioning the stent edge at a site of plaque burden <50%. The aim of this study was to investigate whether targeting less plaque burden beyond this threshold can further contribute to better lumen preservation at the stent edge after DES implantation.
Methods: Data were derived from the J-DESsERT trial, a prospective, randomized, multi-center study, comparing sirolimus- and paclitaxel-eluting stents in de novo native coronary lesions. IVUS was performed at pre-, post-intervention, and 8-month follow-up. Volume index (VI: volume/length, mm^3/mm) was measured for vessel, lumen, plaque within the 5-mm reference segments adjacent to the stent edges. % plaque volume (%PV) was calculated as plaque volume divided by vessel volume. Edge stenosis at follow-up was defined as the minimum lumen area (MLA) <4 mm^2 at the reference segment.
Results: %PV <50% at pre-intervention was achieved in 106 reference segments (61 distal: 45 proximal). Lumen, vessel, and plaque VI, and MLA at pre-intervention were significantly smaller in the edge stenosis group than the no edge stenosis. However, %PV was similar between the 2 groups (37.7±8.1 vs 37.1±8.2%, respectively, p=0.72). Univariate logistic regression analyses showed no contribution of %PV at pre-intervention to MLA at follow-up (OR: 0.99, p=0.72). These results were consistent, when the distal and proximal segments were separately analyzed.

	Edge Stenosis (n=33)	No Edge Stenosis (n=73)	p
Pre-intervention			
Lumen VI (mm^3/mm)	4.8 ± 2.0	8.6 ± 3.1	<0.001
Vessel VI (mm^3/mm)	7.6 ± 3.4	14.0 ± 5.5	<0.001
Plaque VI (mm^3/mm)	3.0 ± 1.6	5.4 ± 2.7	<0.001
Minimum lumen area (mm^2)	4.0 ± 1.7	7.5 ± 2.8	<0.001
% Plaque volume (%)	37.7 ± 8.1	37.1 ± 8.2	0.719
Follow-up			
Lumen VI (mm^3/mm)	4.1 ± 0.9	8.3 ± 2.8	<0.001
Vessel VI (mm^3/mm)	8.1 ± 2.7	14.5 ± 5.3	<0.001
Plaque VI (mm^3/mm)	4.0 ± 2.1	6.2 ± 3.2	<0.001
Minimum lumen area (mm^2)	3.1 ± 0.7	6.9 ± 2.3	<0.001
% Plaque volume (%)	47.0 ± 10.1	41.4 ± 9.2	0.006

Conclusions: In lesions with the stent edge positioned at a site of plaque burden <50%, %PV at pre-intervention did not predict stent edge stenosis at follow-up. This result suggests that aggressive lesion coverage strategy targeting further less plaque burden beyond 50% as the stent landing zone may not offer additional benefit on lumen preservation at the stent edge.

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Coronary Artery Plaque Regression and Change in Plaque Composition Associated with Statin Therapy Extend for a Long-Term -Results from the Extended TRUTH Study-

Tsuyoshi Nozue[1], Kazuki Fukui[2], Kiyoshi Hibi[3], Tomoyuki Kunishima[4], Ichiro Michishita[1], Yuko Onishi[5], Takashi Sozu[6], Mitsuyasu Terashima[7], Shingo Yamamoto[8]
[1]Yokohama Sakae Kyosai Hospital, Yokohama, Japan, [2]Kanagawa Cardiovascular and Respiratory Center, Yokohama, Japan, [3]Yokohama City University Medical Center, Yokohama, Japan, [4]Mizonokuchi Hospital, Teikyo University School of Medicine, Kawasaki, Japan, [5]Hiratsuka Kyosai Hospital, Hiratsuka, Japan, [6]Kyoto University School of Public Health, Kyoto, Japan, [7]Cardiovascular Imaging Center, Toyohashi, Japan, [8]Tsurumi Nishiguchi Hospital, Yokohama, Japan

Background: Recent trials using intravascular ultrasound (IVUS) have shown that statin produces regression and stabilization of coronary artery plaques. The TRUTH study was a prospective, open-labeled, randomized and multicenter trial to evaluate the effects of 8-month treatment with pitavastatin versus pravastatin on coronary artery plaque composition using virtual histology (VH)-IVUS. This study demonstrated that both statins altered coronary artery plaque composition by significantly decreasing the fibro-fatty component and increasing the dense-calcium component. However, there are no reports whether plaque regression or changes in plaque composition using statin could extend because no study has serially monitored coronary artery plaques for a long-term.
Methods: Among 164 patients who participated in the TRUTH trial, additional IVUS examination was performed in 39 patients (mean follow-up period 48±10 months). IVUS images qualifying for evaluation at baseline, at 8-month, and at 48-month were obtained in 30 patients.
Results: Mean age was 67±9 years and 27 patients (90%) were men. Twenty patients (67%) were treated with allocated statins without change in the dose at the TRUTH study, and the dose or type of statin was changed in another 12 patients (40%). Significant decrease in LDL-C (from 130 to 80 mg/dl, p<0.0001) and hs-CRP (from 3690 to 487 ng/ml, p<0.001) were observed at 48-month follow-up. HDL-C levels also increased significantly at 48-month (from 45 to 50 mg/dl, p<0.05). Significant decrease in external elastic membrane volume (-1.1% at 8-month and -5.9% at 48-month) and plaque volume (-0.5% at 8-month and -3.9% at 48-month) have extended. Furthermore, significant increase in dense-calcium component (from 0.56 to 0.65 mm^3/mm at 8-month and 0.77 mm^3/mm at 48-month) and decrease in fibro-fatty component (from 1.09 to 0.94 mm^3/mm at 8-month and 0.86 mm^3/mm at 48-month) have also extended.
Conclusions: Coronary artery plaque regression and change in plaque composition associated with statin therapy extend for a long-term. These changes in coronary atherosclerosis may lead to beneficial effects of statins on long-term clinical outcomes.