Conclusions: Rate of MACEs was significantly lower in the EES group when compared with the SES, FES, and ZES groups, mainly due to the lower rate of TLR during the 3-year follow-up.

TCT-646
Primary Results Following Percutaneous Coronary Intervention with the 38 mm Resolute Zotarolimus-eluting Stent
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Background: Implantation of drug-eluting stents in long coronary artery lesions is associated with a higher risk for restenosis and stent thrombosis related to the need for multiple and overlapping stents. The Resolute zotarolimus-eluting stent (R-ZES) is manufactured in a 38 mm length to accommodate longer lesions but clinical data to demonstrate efficacy and safety of the longer stent have not been reported.

Methods: A sub-study of 2 prospective, multicenter clinical trials; RESOLUTE-US and RESOLUTE-Asia enrolled patients with de novo coronary artery lesions amenable to treatment with the 38-mm-length R-ZES. The target lesion had to be > 35 mm long and the primary endpoint was target lesion failure (TLF) defined as the composite of cardiac death, target vessel myocardial infarction (TVMI), or clinically-driven target lesion revascularization (TLR) by percutaneous or surgical methods. The rate of TLF at 1 year was compared to a performance goal of 19.0% based on literature suggesting an expected TLF rate of 15.1%. All patients were prescribed dual antiplatelet therapy for a minimum of 6 months.

Results: There were 223 patients treated with a 38 mm R-ZES; mean age was 61 years, 78.9% were men, 44.6% were white, 3.6% were black, and 50.9% were Asian. Prior PCI was noted in 27.4% of patients, 37.7% had diabetes mellitus and 53.8% had multivessel disease. Lesions were located in the LAD (52.0%), LCX (20.2%) and RCA (44.4%) and 3.4% were moderately or severely tortuous. TIMI flow was 0 or 1 in 61% of lesions and 47.9% were branch vessel disease with mean sidebranch stenosis of 30.4%; 91.2% were lesion class B2/C. The primary endpoint was met with a 12-month TLF rate of 4.5% (upper one-sided 95% confidence interval 7.5%). Rates of cardiac death, TVMI, and TLR at 12 months were 0.9%, 3.6%, and 1.4%, respectively. Early stent thrombosis (<30 days) was 0.9% and there were no stent thrombosis events after 30 days.

Conclusions: At 12 months the rate of TLF was significantly less than the performance goal of 19% and clinical events rates were low with no late stent thrombosis. The 38 mm R-ZES demonstrated efficacy and safety of the longer stent have not been reported.

TCT-647
Impact of Stent Structural Design and Deployment Pressure on Strut Apposition and Recoil
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Background: There are major differences in the structural design of currently available coronary stents. Differences in radial forces have been reported between the different stent platforms, but it is still unclear how stent material and strut design may affect stent mechanical performances in clinical settings.

Methods: To evaluate coronary stent designs in a reproducible environment, we created a compliant model of a focal coronary lesion, representing a 50% reduction in lumen diameter. The stents evaluated were the Cobalt Chromium (CoCr) Xience V (Abbott Vascular) and Integrity (Medtronic), 316L Stainless Steel (316L SS) Taxus Liberte (Boston Scientific) and BioMatrix Flex (Biosensors Int.), and the Platinium Chromium Vascular and Integrity (Medtronic), 316L Stainless Steel (316L SS) Taxus Liberte (Boston Scientific) and BioMatrix Flex (Biosensors Int.), and the Platinum Chromium (PtCr) Promus Element (Boston Scientific). The 3.0 stents (n=14) were deployed across the model lesion at their Nominal Pressure (NP). Minimal Lumen Area (MLA), residual % area stenosis and strut apposition were assessed at NP from micro-CT. After NP assessment, the same 3.0 delivery balloon was then inflated at 18 ATM to evaluate impact of deployment pressure on plaque recoil and residual stenosis. A total of 28 experiments were performed.

Results: MLA was significantly increased with pressure deployment: from 5.0 ± 0.5 mm2 at NP to 6.8 ± 0.1 mm2 with 18 ATM dilatation (p<0.0001). Higher pressure also eliminated the risk of malapposed strut from a maximal cross-sectional rate of malapposed strut observed of 25.6 % at NP compared to virtually zero malapposition at 18 ATM pressure. At NP, CoCr stents resulted in overall higher MLA and lower residual stenosis than 316L SS and PtCr stents. MLA was respectively 5.4 mm2 for CoCr, 4.9 mm2 for 316L SS and 5.0 mm2 for PtCr (p<0.0001). Such differences were markedly eliminated with higher pressure inflation: at 18 ATM, MLA was respectively 6.7 mm2 for CoCr, 6.6 mm2 for 316L SS and 6.8 mm2 for PtCr.

Conclusions: Initial results with 5 different DES designs underline the importance of deployment pressure in the overall ability of a stent to scaffold a lesion and restore lumen area.

TCT-648
Value Of High-Density Lipoprotein Cholesterol In Predicting Future Cardiovascular Events Of Patients With Low-Density Lipoprotein Cholesterol At The Time Of Percutaneous Coronary Intervention
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Background: Higher levels of high-density lipoprotein cholesterol (HDL-C) have been associated with decreased cardiovascular risk in epidemiologic studies, although a causal role is in question. Dysfunctional HDL has been identified in humans with metabolic syndrome (MSx) or coronary artery disease (CAD). Additionally, current determinations of HDL-C may not correlate with the true anti-atherosclerotic properties, casting doubts on its prognostic value. Therefore, the significance of HDL-C on the outcome of patients with CAD requiring percutaneous coronary intervention (PCI) is unclear.

Methods: Patients treated with PCI from 01/2004 to 12/2011 were prospectively enrolled. The lipid panel of those on statin therapy prior to PCI and who had attained low-density lipoprotein cholesterol (LDL-C) <100 mg/dl was analyzed. Major adverse cardiac events (MACE), including all-cause death, Q-wave myocardial infarction, and target vessel revascularization at 1 year were evaluated in relation to the lipid profile at the time of index PCI. Multivariable Cox proportional hazards regression was employed for the entire cohort and for the subgroup of patients with diabetes mellitus (DM) or MSx.

Results: 2789 patients were included. The population’s mean age was 66 years and 68% was male. 53% had a history of CAD, 39.8% had DM and 42.5% had MSx. At 1 year follow-up, a total of 279 patients (10.1%) experienced MACE. Death occurred in 5.2% of the population. HDL-C after adjustment for baseline characteristics did not demonstrate an independent association with MACE in either the main population or the subgroup with MSx (HR 1.00, 95% CI 0.99-1.01, p=0.98 and HR 1.00, 95% CI 0.99-1.01, p=0.93, respectively). However, the evaluation of non-HDL levels at the time of PCI demonstrated a trend for the subgroup of patients with DM or MSx (HR 0.99, 95% CI 0.99-1.00, p=0.07).

Conclusions: In patients treated with statin therapy for CAD, in whom an LDL-C level <100 mg/dl has been achieved at the time of PCI, HDL-C did not independently correlate with MACE at 1 year. These data suggest that HDL-C for patients on statin therapy and controlled LDL may not be an effective biomarker for future clinical events.