results to be generalisable to the entire target population. An adjusted model was created to analyse death as an outcome at 30 days using a treatment and inclusion to VALIDATE trial interaction term.

Results: The propensity-score differences did not meet the criteria of 0.25; thus reflecting that the patient populations between the randomised and screened not enrolled group were not similar. The adjusted model showed that the randomised population had lower mortality than the screened not enrolled group irrespective of the received treatment, OR 0.50, 95% CI [0.330–0.772]). However, the neutral efficacy of bivalirudin versus heparin was maintained in the non-randomised group, which matches the results of the VALIDATE trial (OR 1.12, CI [0.697–1.801]).

Propensity-score difference of Validate

Randomised and screened not enrolled population	n	Propensity-score difference
Total population	12561	
All patients with no systematic missing values for adjustment variables*	10263	0.47
All patients with fulfilled inclusion criteria*	7640	0.76
No killip class >2 & no CPR/No exclusions and fulfilled adjustment criteria*	7739	0.74

*Analyses were adjusted for STEMI, weight <65, gender, age, smoking, diabetes, hypertension, hyperlipidaemia, previous heart attack, previous MI, previous coronary artery bypass, previous stroke, ticagrelor before PCI, clopidogrel before PCI, CPR, kidney failure, trombectomy and killip class.

Conclusions: Patients enrolled in the randomised VALIDATE trial showed significantly lower mortality rates compared to patients screened not enrolled, which can be explained by patients populations' not being similar. However, in multivariable analysis, the results show that there were no differences in mortality between bivalirudin versus heparin in the screened not enrolled population thus reflecting the results from the original VALIDATE trial.

P3644
Independent predictors of very long-term outcomes of patients with stable angina - insights from The Prospective REgistry of Stable Angina manaGment and trEatment (PRESAGE) Registry

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Background: Changes in clinical presentation and modification of treatment of patients with stable angina (SA) prompt to continuous monitoring of the results of treatment in the real-world practice. Accurate estimation of predictors of very long-term outcomes may improve and clarify the therapeutic decisions for patients.

Aim: The aim of the study was to assess the very long-term independent predictors of all-cause mortality in patients with SA.

Methods: Data of patients from the PRESAGE Registry were analyzed. In brief, the PRESAGE Registry is an ongoing, single-center, prospective observational study recruiting consecutive patients with SA. Independent predictors of 5-year all-cause mortality were calculated by Cox proportional hazard models.

Results: Between 2006 and 2014, a total of 9379 consecutive patients were enrolled to PRESAGE registry. The average age was 65.1±9.5, the percentage of female constituted 33.3% of study population. At 5 years the cumulative rate of all-cause death was 16.9%, non-fatal MI occurred in 6.9% and ACS-driven revascularization was performed 8.1%. Independent predictors influencing 5-year mortality were lower left ventricular ejection fraction (per 5%; hazard ratio 1.29; 95% confidence interval 1.24–1.34; P<0.0001), higher serum creatinine on admission (per 10 umol/L; HR 1.04; 95% CI 1.03–1.05; P<0.0001), peripheral artery disease (HR: 1.63; 95% CI 1.36–1.96; P<0.0001), diabetes mellitus (HR 1.45; 95% CI 1.23–1.71; P<0.0001), history of cancer disease (HR 6.65; 95% CI 2.92–15.12; P<0.0001), lower hemoglobin on admission (per 1 mmol/L; HR 1.23; 95% CI 1.12–1.36), chronic obstructive pulmonary disease (HR 1.64; 95% CI 1.28–2.11; P<0.0001), older age (per 5 years; HR 1.11; 95% CI 1.05–1.16), atrial fibrillation (HR 1.36; 95% CI 1.11–1.67; P=0.0045), chronic total occlusion (HR 1.32; 95% CI 1.08–1.62; P=0.0062) and revascularization (HR 0.81; 95% CI 0.66–0.98; P=0.032).

Conclusions: The PRESAGE Registry provides evidence for independent predictors of very long-term outcomes in SA population. Further study with validation cohort and advanced statistical models should be performed to confirm our findings.

P3645
Interleukin 8 (IL-8), but not GRO-a, associates with carotid intima-media thickness. Results from the IMPROVE study

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Background and aim: We investigated the causality of interleukin 8 (IL-8) and GRO-α, two chemokines known to participate in the inflammatory response in atherosclerosis, on carotid intima-media thickness (c-IMT), in a multicenter European study the c-IMT and c-IMT Progression as Predictors of Vascular Events in a High Risk European Population (IMPROVE) (n=3711).

Methods: At baseline c-IMT measures were recorded (mm). IL-8 and GRO- α (available in 3367 study participants) were measured by Proximity Extension Assays and expressed in arbitrary units. Genomic DNA was genotyped with the CardioMetaboChip 200K and the Immunochip arrays. After exclusions, a total of 251108 SNPs and 3325 study participants were available for analysis. The association of IL-8 and GRO-a with c-IMT measures was estimated by quantile regression, expressed as beta coefficients (b) and standard error (SE) and adjusted by age, gender, latitude and the common cardiovascular (CV) risk factors. Linear regression was used to identify SNPs associated with IL-8. IL-8 SNPs were also tested for association with c-IMT measures. Estimates expressed as b (SE) were adjusted by age, gender, multi dimension scale as well for the c-IMT and IL-8 lead SNP, respectively. A IL-8 SNP score was created summing the effect of the IL-8 increasing alleles and used as instrumental variable to assess causality of the association of IL-8 with c-IMT measures using the 2 stages least squares method.

Results: Each unit increase in plasma IL-8, but not GRO- a, levels was associated with an increase in the median c-IMT measures after adjustments (all p<0.006). Two loci, on chromosome 8 (lead SNP rs117518778) and on chromosome 16 (lead SNP rs8057084) were associated with IL-8 plasma levels. The effect allele (EA) at rs8057084 associated with lower IL-8 levels (b: -0.023, SE 0.005, p=5.72*10–6). Rs8057084 was in moderate linkage disequilibrium, (r2=0.7) with rs4888378, a SNP previously associated with lower c-IMT in the IMPROVE. Inclusion of rs4888378 in the regression model did not significantly change the estimates (b: -0.031, SE 0.0009 p=0.0017). Instrumental variable analysis, using the IL-8 SNP score as instrumental for IL-8 confirmed the causality of the association.

Conclusions: IL-8 is causally associated with c-IMT, a measure of arterial remodeling and subclinical atherosclerosis. These findings generate novel hypothesis of target molecules for early primary and secondary cardiovascular prevention.

P3646
Controlling nutritional status (CONUT) score predicted poor outcome better then prognostic nutritional index(PNI) in patients with acute myocardial infraction

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Background: Nutritional status has been related to clinical outcomes in patients with acute myocardial infraction. Prognostic nutritional index (PNI) and controlling nutritional status (CONUT) score has a prognostic impact in patients with severely decompensated acute heart failure and coronary artery disease. However, the relationship between two nutritional status score and clinical outcomes in patients with acute coronary syndrome (ACS) remains unclear.

Purpose: We want to understand the associations of nutritional status via PNI and CONUT score level with the development long-term mortality in patients with ACS treated with intervention including percutaneous coronary intervention (PCI), coronary artery bypass grafting (CABG) alternatively, and conservative medical treatment.

Methods: From August 2003 to December 2012, data for patients hospitalized due to ACS were drawn from an intramural registry and measured the PNI and CONUT score on admission (PNI was calculated as 10*serum albumin (g/dL)+ 0.0059 total lymphocyte count (per mm^3); CONUT was calculated as serum albumin (g/dL), total cholesterol level (mg/dl) and total lymphocyte count (/ml); range 0–12, higher score reflect worse nutritional status). The multivariate Cox-regression model calculated predictors of all- course mortality and cardiovascular mortality. Akaike Information Criteria and Schwarz Bayesian criterion compare the effect between PNI and CONUT. Net reclassificantion improvement used for quantify CONUT score. National Death Registry was linked for the identifications of mortality data.

Results: Among 1714 participants (age 73.1±13.1 years, 75.9% male), 1153 patients received coronary angiography, 884 patients perform percutaneous coronary intervention (PCI), 202 patient had CABG surgery (17 cases had received PCI before) during a mean follow-up of 41.4±29.0 months. The Kaplan-Meier curves revealed that patients with low PNI or high CONUT scores had higher rates of the major event such as mortality and cardiovascular death (log-rank p<0.001). The PNI and COUNT score were an independent predictor for long-term (5 year) [Hazard ratio and 95% confidence intervals per-1 point: [1.09, 1.03–1.15] all-course mortality and [1.11 1.02–1.20] cardiovascular mortality with ACS. AIC and SBC are smaller between PNI and CONUT score and C-statistics is the same as that of PNI. Utilized CONUT, the net proportion of event and non-event assigned correctly increases 28% and 3% respectively

Conclusion: Nutritional status assessed by PNI and CONUT score helps predict survival in patients with ACS. CONUT score may provide more accurate prognostic information than PNI