Table 1. Number of bleedings according to the scores

| Score | CHADS, n (%) | CHADSVASC, n (%) | HASBLED, n (%) |
|---|---|---|---|
| 0 | 4/79 (5.1) | 0/27 | 2/42 (4.8) |
| 1 | 18/247 (7.3) | 3/63 (4.8) | 13/207 (6.3) |
| 2 | 26/317 (8.2) | 9/144 (6.2) | 32/458 (7.0) |
| 3 | 12/224 (5.4) | 24/254 (9.4) | 31/276 (11.0) |
| 4 | 21/172 (12.2) | 20/241 (8.3) | 8/80 (9.1) |
| 5 | 7/39 (17.9) | 12/202 (5.9) | 2/14 (14.3) |
| 6 | 0/7 | 14/105 (13.3) | 0/0 |
| 7 | na | 6/42 (14.3) | 0/0 |
| 8 | na | 0/6 | na |
| 9 | na | 0/1 | na |
| Total | 88/1085 (8.1) | 88/1085 (8.1) | 88/1085 (8.1) |

**Results:** We prospectively studied 1085 AF patients (37.8%female; median age 75 years, follow-up 4697 patient-years). We stratified patients in relation to the 3 indicated scores. The average CHADS2 score was 2.2±1.3; CHA2DS2-VASc was 3.7±1.6 and HASBLED was 2.2±1.0; 88 major bleeds were recorded (rate 1.78×100 patient-years) and stratified according to the scores (Table 1). Predictive ability of HASBLED was 0.569 (0.507–0.631; p=0.030), of CHADS2 was 0.559 (0.494–0.623; p=0.041), of CHA2DS2VASc was 0.555 (0.493–0.616; p=0.047).
**Conclusions:** The number of bleeds increases with the increase of the score for all models, with similar predictive ability for bleeding risk. HASBLED seems not to add useful information to identify high risk patients, and stroke stratification scores could be sufficient for tailoring treatment.

### OC-128
### Stroke in APS patients: clinical and laboratory characteristics

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**Background:** Laboratory aPL profile and clinical characteristics of APS patients presenting with stroke are still lacking. Patients with APS and ischemic stroke as index event were re-evaluated in a multidisciplinary, centralized board. Clinical history, risk factors, radiological imaging (MRI and CT), and laboratory aPL profile were evaluated to assess characteristics of APS stroke patients.
**Patients and methods:** 60 (14 males and 46 females) APS patients with stroke as index event have been studied either for the aPL profile and for radiologic imaging. The centralized board, including a neurologist, adjudicated and classified the APS syndrome and the radiological imaging. Ongoing guidelines for APS classification TOAST criteria for stroke classification and were strictly observed.
**Results:** Median age at diagnosis was 49 years. aPL profile: 88% had "high risk" (Miyakis 1 and 2a) aPL profile. Stroke characteristics: 31 (52%) patients had a lacunar stroke, 48% had cardioembolic like lesion. Cardiac US abnormalities: 9 (15%) patients had a patent foramen ovale, 8 (13%) had mixomatous thickening of mitral valve. Risk factors: hypertension was found in 32 patients (53%), dyslipidemia in 22 (36%), smoking in 18 (30%), migraine in 11 (18%), obesity in 7 (12%), oestro-progestinic pill in 7 (12%).
**Conclusions:** APS patients had the stroke at a younger age with respect to the general population in Italy (49 vs 74 years). A high-risk aPL profile was found in about 90% of patients. Lacunar stroke is the most represented type of ischemic stroke.

### OC-129
### Enhanced fibrinolysis in patients treated with dabigatran etexilate

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In vitro, we showed that the direct thrombin inhibitor dabigatran facilitates fibrinolysis through TAFI-dependent and TAFI-independent mechanisms. Here, we evaluated the effect of dabigatran treatment on plasma fibrinolysis and the possible involvement of the TAFI pathway.

Plasma from 15 patients with atrial fibrillation receiving therapeutic doses of dabigatran etexilate (Pradaxa®) was collected 2–3h after administration ("peak"), and ≈ 20 h later, right before the next dose ("trough"). Dabigatran levels at "peak" and "trough" were (median [IQR]): 151 [53–435] and 63 [12–156] ng/mL, respectively (p=0.0001). The lysis time of clots exposed to t-PA was significantly shorter in "peak" than in "trough" samples (65 [42–109] and 72 [45–124] min, respectively; p=0.0004) and inversely correlated with dabigatran concentration (rho=−0.64, p=0.0001). Moreover, thrombin (ETP) and TAFIa generation in clotting plasma showed an inverse correlation with dabigatran concentrations ($p \leq 0.0003$) and were significantly lower at "peak" ($p \leq 0.002$), suggesting a reduction of thrombin-mediated TAFI activation. Upon addition of PTCI, a specific inhibitor of TAFIa, the difference in lysis time between "peak" and "trough" samples became smaller but was still statistically significant (p=0.005). Thus, consistent with previous in vitro findings, our data suggest that dabigatran treatment accelerates fibrinolysis by impairing thrombin-mediated TAFI activation as well as by other mechanisms. Concerning the in vivo effect of dabigatran treatment, the plasma levels of plasmin-antiplasmin complexes were significantly higher at "peak" (261 [121–726] vs 223 [105–702] ng/mL, p=0.0004), hinting to enhanced in vivo fibrinolysis. However, the circulating levels of TAFIa/ai (a marker of in vivo TAFI activation) did not differ between "peak" and "trough" samples, suggesting that enzymes other than thrombin may activate TAFI in vivo, under basal conditions.
In conclusion, dabigatran administration enhances "ex vivo" plasma fibrinolytic potential and is associated with increased "in vivo" fibrinolysis markers, suggesting that hyperfibrinolysis may be an additional mechanism of the antithrombotic activity of dabigatran.

## Oral communications

### Atherosclerosis, diabetes, MS and arterial occlusive diseases II

### OC-130
### Dietary inflammatory index in the Italian population: findings from the INHES project

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**Background:** Reducing inflammation may help prevent cardiovascular disease. Diet has consistently been shown to modulate inflammation. Recently, a Dietary Inflammatory Index (DII) designed to assess the inflammatory potential of individuals' diets has been proposed.
The study's aim was to evaluate how this DII is distributed in a large sample of Italians.
**Methods:** We included 6,933 persons (3,177 men, age range 5–96) recruited in the Italian Nutrition & Health Survey (INHES) Project*, a program specifically designed to collect current information on dietary habits and lifestyle of the Italian population. Subjects with cardiovascular diseases, diabetes, under dietary therapy or with alimentary allergies were excluded. The DII was based on data from 30 foods and nutrients positively or negatively associated with inflammation, in relevant peer-reviewed journal articles. Food- and nutrients-specific scores were multiplied by the intake for each participant, and then summed to create the overall Inflammatory Index score. Major determinants with a negative weight are energy, carbohydrates and fats, whereas those with a positive load are tea, alcohol, fiber, (n-3) fatty acids and vitamins. The score was normalized to mean zero and standard deviation one and its positive values represent movement toward an anti-inflammatory diet.
**Results:** The DII ranged from −3.0 to 8.5 in the Italian population. It was higher in women, at increasing age or educational level and in North regions of Italy as compared with the South (Table 1; all differences $P<0.0001$; multivariable analysis of variance adjusted for sex, age and geographic location). No differences were found according to BMI.

Table 1. Dietary Inflammatory Index according to sex, age, geographic location and education level

| | N | Mean | SD | P-value |
|---|---|---|---|---|
| Sexes | | | | <0.0001 |
| Women | 3,756 | 0.08 | 1.00 | |
| Men | 3,177 | −0.09 | 0.99 | |
| Age (years) | | | | <0.0001 |
| ≤ 14 | 165 | −0.47 | 0.54 | |
| >14–19 | 210 | −0.66 | 0.70 | |
| >19–35 | 427 | −0.29 | 0.78 | |
| >35–65 | 4,159 | 0.03 | 1.02 | |
| ≥65 | 1,972 | 0.10 | 1.01 | |
| Geographic area | | | | <0.0001 |
| North | 2,759 | 0.14 | 1.15 | |
| Center | 1,170 | 0.02 | 0.94 | |
| South/Islands | 3,004 | −0.13 | 0.84 | |
| Education level (years of education) | | | | <0.0001 |
| ≤8 | 3,171 | −0.07 | 0.96 | |
| 8 to 13 | 2,682 | 0.04 | 1.04 | |
| >13 | 1,080 | 0.10 | 1.00 | |

**Conclusions:** Consumption of foods with anti-inflammatory proprieties is limited among young Italians, especially in the range 14–19 years, in men, in poorly educated people and in subjects living in Southern Italy. Our findings could be used for guiding individuals in setting dietary goals to help decreasing levels of inflammation.

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## OC-131

## Release of MMP-2 by platelets in patients with acute coronary syndromes undergoing percutaneous trans-coronary angioplasty

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The rupture of an atherosclerotic plaque with subsequent thrombus formation is the event underlying acute coronary syndromes (ACS). Matrix metalloproteinases (MMPs), present in atherosclerotic plaques are the enzymes implicated in their rupture. Several MMPs, including MMP-2, are contained also in platelets and released upon activation. We have previously shown that MMP-2 is released in the culprit coronary artery of medically-treated patients with ACS and contributes to sustain platelet activation. An increase in the circulating levels of MMP-2 has also been reported in patients undergoing percutaneous transluminal coronary angioplasty (PTCA), but its origin remains unclear. Aims of the current study were to assess whether plasma MMP-2 increases in patients with coronary artery disease undergoing PTCA, to evaluate the time-course of this release and to define its origin. Patients with ACS (NSTEMI or STEMI, n=32) or with SA (stable angina, n=21) were enrolled. Peripheral blood samples were drawn immediately before, immediately after, and 4 and 24 h after PTCA. Platelet-poor plasma and washed platelet lysates were prepared and stored (−80°C) for subsequent analyses. β-thromboglobulin (β-TG), a platelet-specific protein released upon activation, and MMP-2 levels were assayed by ELISA and by zymography, respectively. Plasma MMP-2 increased significantly 4h after PTCA and returned to baseline at 24h in ACS patients, while it did not change in SA patients. Plasma β-TG also increased, with a pattern similar to that of MMP-2. Platelet content of MMP-2 and β-TG decreased significantly 4h after PTCA in patients with ACS, compatible with ongoing platelet activation, while they did not change in patients with SA. Therefore, the characteristics of the angioplasted coronary plaques are crucial in generating in vivo platelet activation and subsequent MMP-2 release. In turn, released MMP-2 may contribute to sustain platelet activation and possibly to unstabilize downstream-located plaques. These data may encourage the search for therapeutic agents blocking MMP-2 release or activity.

## OC-132

## Impairment of peripheral vascular function in Systemic Lupus Erythematosus patients. Differences from the vascular pattern of patients at high cardiovascular risk

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**Purpose:** We investigated vascular function of peripheral arteries in Systemic Lupus Erythematous (SLE) patients, comparing results obtained in patients with acute coronary syndromes (less 1-year previous event) and in healthy control subjects (no cardiovascular events or autoimmune disease).
**Methods:** In 170 patients (controls: 76, ACS: 74, SLE: 20) peripheral arterial tonometry (PAT) and augmentation-index (Aix) by EndoPAT (Itamar, Caesarea, Israel), intima-media-thickness and pulse-wave-velocity at common carotid (c-IMT, c-PWv) and femoral arteries (f-IMT, f-PWv) by ultrasound technology (Esaote MyLab70) were assessed.
**Results:** Among SLE group, 8 (40%) had a previous vascular event. ACS showed higher cardiovascular risk profile. ACS showed a marked atherosclerotic damage, because of higher c- and f-IMT values in comparison to others (ACS: c-IMT=1.9±0.6mm, f-IMT=1.7±0.8mm; SLE: c-IMT=1.6±0.8mm, f-IMT=1.5±0.7; controls: c-IMT=1.3±0.9mm, f-IMT=1.2±0.4); SLE and ACS data did not significantly differ, but both resulted to be significantly higher than data found in controls (p=0.02 and p=0.03, respectively).
Vascular compliance was significantly impaired in SLE (c-PWv=10.5±2.6m/s, f-PWv=10.2±1.3 m/s), in comparison to ACS (c-PWv=9.2±2.3 m/s, f-PWv=9.4±3.1 m/s; $p<0.05$) and controls (c-PWv=7.1±2.1 m/s, f-PWv=7.2±1.4 m/s; p=0.001). Aix was higher in SLE (17.2±6.3%), in comparison to ACS (15.3±1.4%, p=0.2) and controls (8.1±4.2%; p=0.002).
SLE showed a significantly lower endothelial function, expressed as natural logarithm of reactive hyperaemia index (LnRHI), (0.56±1.2), compared to controls (0.79±1.7; p=0.03) and these values minimally differed between SLEs and ACS groups (0.50±2.3, p=0.3). LnRHI and PWv values were significantly correlated with number of CRFs. At univariate and multivariate analyses, presence of SLE and a previous vascular event were significantly associated to endothelial dysfunction (p=0.02 and 0.03, respectively) and compliance impairment (p=0.02 and 0.03, respectively).
**Conclusions:** Our data suggested that SLE showed a marked atherosclerotic peripheral vascular involvement, similar to patients at very high CV as ACS, because of impairment of local compliance at common carotid and femoral arteries and a marked endothelial dysfunction.

## OC-133

## Role of platelets in the increased cardiovascular risk of non-alcoholic fatty liver disease

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**Introduction:** Non-alcoholic fatty liver disease (NAFLD) and Non-alcoholic steatohepatitis (NASH) are the commonest cause of chronic liver disease. NAFLD and NASH are associated with a significant increase of cardiovascular events, which represent the major cause of mortality, most probably caused by a systemic proinflammatory/prothrombotic condition. The platelet transcriptome might change into a pro-inflammatory and a prothrombotic phenotype in diseases at high risk of cardiovascular mortality.
**Aim of the study:** The aim of the study is to assess whether the changes induced by chronic inflammatory stimulus in NASH induces a proinflammatory/prothrombotic phenotype in platelets.
**Materials and methods:** Nine patients with histologic diagnosis on liver biopsy of NASH and nine healthy volunteers were recruited in this pilot study. mRNA was extracted from purified platelet and leukocyte populations and subsequently reverse transcribed in cDNA. The differential expression of transcripts encoding receptors relevant for the platelet hemostatic function (GPVI, GPIB, F2R, F2RL3, P2Y1, P2Y12, TBXA2R) and inflammatory transcripts (TLR2, TLR4, IL6, IL1R1, MPO, CXCR1, ICAM1, TNF) was evaluated through comparative analysis of real-time PCR.
**Results:** Increased expression of some inflammatory transcripts, such as ICAM1, MPO, IL6, IL1R1 and CXCR1, was found in platelets of NASH subjects compared to controls, whereas the "hemostatic" transcripts of platelets were unchanged in the two populations. In contrast, the inflammatory