Results: PHC patients and controls have similar age, gender distribution, waist, BMI and blood pressure. Significant statistical differences were observed as expected in TC and LDL-C plasma values. We also found significant differences in fasting inflammatory markers (table 1).
During the OFLT significant differences in IL-8 and IL-1b persisted when comparing both studied groups. We observed a progressive decrease in IL-8 and IL-1b in PHC during the OFLT, although statistical significance was not achieved. However, the levels of IL did not change in the control group (table 2).

Table 1

	Controls (n=21)	PHC (n=14)
Age (y)	38.9 ± 10.6	40.3 ± 7.4
Gender Male/Female n (%)	10 (48)/11 (52)	8 (57)/6 (43)
BMI (kg/m^2)	24.5 ± 3.0	25.1 ± 3.2
Waist (cm)	86.1 ± 9.5	87.9 ± 9.2
Systolic blood pressure (mmHg)	112.6 ± 12.4	118.8 ± 13.0
Diastolic blood pressure(mmHg)	68.1 ± 7.0	73.0 ± 7.5
Fasting Glucose (mg/dl)	91.7 ± 7.5	94.5 ± 9.7
Total Cholesterol (mg/dl)	177.7 ± 26.5	376.7 ± 85.8*
Triglycerides (mg/dl)	67.9 ± 20.9	203.7 ± 142.8*
HDL-C (mg/dl)	59.7 ± 11.1	59.4 ± 10.4
LDL-C (mg/dl)	104.4 ± 22.3	280.4 ± 81.5*
IL-1β (pg/dl)	0.7 ± 0.7	2.1 ± 2.9*
IL-2 (pg/dl)	12.9 ± 10.2	13.2 ± 7.9
IL-8 (pg/dl)	3.3 ± 2.3	11.6 ± 14.6*

*p<0,05

Table 2

Time (h) oral fat load test		0	2	4	6	8
IL-1β	Controls (n=21)	0.7 ± 0.7	0.8. ± 0.6	0.7 ± 0.5	0.6 ± 0.5	0.6 ± 0.5
	PHC (n=14)	2.1 ± 2.9*	1.7 ± 2.5	1.9 ± 3.0	1.5 ± 1.8*	1.6 ± 1.9*
IL-2	Controls (n=21)	12.9 ± 10.2	13.8 ± 7.5	11.3 ± 8.0	9.9 ± 5.7	11.9 ± 8.9
	PHC (n=14)	13.2 ± 7.9	10.5 ± 8.5	11.4 ± 10.1	8.2 ± 6.1	10.7 ± 6.8
IL-8	Controls (n=21)	3.3 ± 2.3	3.4 ± 2.3	3.2 ± 2.4	3.3 ± 2.5	3.1 ± 2.0
	PHC(n=14)	11.6 ± 14.6*	13.1 ± 13.7*	11.9 ± 15.5*	10.9 ± 14.8*	10.3 ± 13.7*

Differences between PHC and control *p<0,05

Conclusions: PHC patients showed higher fasting inflammatory markers. We found a progressive reduction of IL levels in subjects with PHC (not present in controls). There were no significant differences. However, it is a pilot study with a few number of subjects included, but this trend suggests that unsaturated oral fat could be beneficial to reduce inflammation.

P1.1.028.
AN INHIBITOR TO MITOCHONDRIAL REACTIVE OXYGEN SPECIES INHIBITED PRO-INFLAMMATORY EFFECT IN ATHEROSCLEROTIC PLAQUES

M. Rahman [1], J. Steuer [2], P. Gilgren [2], A. Liu [1], J. Frostegård [1]. [1] *Karolinska Institutet, department of environmental medicine, Stockholm, Sweden;* [2] *Karolinska Institutet hospital, department of vascular surgery, Stockholm, Sweden*

Aim: Activated immune cells including macrophages and T-cells are abundant in atherosclerotic plaques and could play an important role in plaque erosions, rupture and cardiovascular disease (CVD). Oxidized low density lipoprotein (OxLDL) activates T-cells but role of different components in OxLDL are not uncovered. We therefore study oxidized phospholipid (Ox-PL) malondialdehyde conjugated with human serum albumin (MDA-HSA).
Methods: T-cells from human peripheral blood or atherosclerotic plaques stimulated with MDA-HSA or with Ox-PL in presence or absence of mitochondrial reactive oxygen species inhibitor (Mito-tempo) or anti-MDA antibodies. MDA-HSA or Ox-PL induced T-cells were co-cluttered with monocyte derived macrophages. Next, blood plasma from atherosclerotic patients` who were treated with or without lipid lowering drugs were added into the same patients` cells in presence or absence of mito-tempo or anti-MDA antibodies. Flow cytometry, cytokine measurement and transcription factors were analyzed for cellular activation profile.
Results: Both MDA-HSA and Ox-LP, also blood plasma induced pro-inflammatory T-cell activation. MDA-HSA, Ox-PL or blood plasma induced T-cell stimulated atherogenic M1 macrophages differentiation. MDA-HSA-induced T-cell activation was inhibited by mito-tempo or anti-MDA antibodies. Mito-tempo but not anti MDA inhibited Ox-PL or plasma induced T-cell activation.
Conclusions: MDA-HSA or Ox-PL promotes T-cells in plaques through direct mechanism without antigen presenting cells. T-cells activation was strongly induced with the plasma from the patients who were not treated with lipid lowering drugs. Specifically mitochondrial reactive oxygen species inhibitor can be a therapeutic possibility to prevent immune activation in plaques.

P1.1.029.
THE EFFECTS OF NORDIC WALKING TRAINING ON BLOOD ANTIOXIDANT DEFENCE IN PATIENTS WITH INTERMITTENT CLAUDICATION

E. Rosloniec [1], M. Wiecek [2], P. Mika [1]. [1] *Department of Clinical Rehabilitation, University of Physical Education, Krakow, Poland;* [2] *Department of Physiology and Biochemistry, University of Physical Education, Krakow, Poland*

Aim: Enhanced antioxidative protection can improve the condition of patients with intermittent claudication (IC). The goal was to evaluate changes in enzymatic and non-enzymatic blood antioxidant systems in patients with IC after a 12-week supervised Nordic Walking training.
Methods: The study included 38 subjects(12F, 26M) aged 66(±8)with IC, divided into two groups.Group NW: n=21 (5 F, 16 M) aged 66(±7) underwent a 12-week supervised Nordic Walking training conducted 3 times per week. A typical training session lasted approximately 50 minutes. Control group: n=17 (7 F, 10 M) aged 66 (±9) did not undergo any training. Before and after 12 weeks, plasma samples were used for analysis of the antioxidative enzymes activity: superoxide dismutase(SOD) and glutathione peroxidase(GPx) and uric acid concentration (UA). The total concentration of lipid peroxides was also determined. Additionally, walking ability was evaluated on the basis of a walking test according to Gardner protocol.
Results: Significant increases in SOD activity from 5.70 to6.46U/mL($P < 0.05$) and UA concentration from 235.2to330.6μmol/L($P < 0.01$), as well as a decrease in lipid peroxides from 926.3to568.1μmol/L($P < 0.01$) were observed in the group NW. After training, the time to claudication was extended from174sec(±98)on average by 94%, the maximum walking time was extended from 496 sec (±196) on average by 70% ($P < 0.001$). No significant changes in the evaluated parameters were observed in the control group.
Conclusions: A 12-week supervised Nordic Walking training leads to the improvement of walking ability and blood redox balance by increasing enzymatic and non-enzymatic antioxidant defence, resulting in a reduction in oxidative lipid damage.

P1.1.030.
THE ROLE OF LIPOPROTEIN METABOLISM AND INFLAMMATORY GENE POLYMORPHIMS ON THE CORONARY ARTERY DISEASE WITH TYPE 2 DIABETES MELLITUS

N. Coban [1], A.S. Ozuynuk [1], A.F. Erkan [2], B. Ekici [2], E. Vurgun [1], N. Erginel-unaltuna [1], I.Y. Diker [1]. [1] *Department of Genetics, Institute for Experimental Medicine, Istanbul University, Istanbul, Turkey;* [2] *Department of Cardiology, Medical Faculty, Ufuk University, Ankara, Turkey*

Aim: Coronary artery diseases (CAD) is one of the most important public health problems in Turks. In addition, CAD is the leading cause of morbidity and mortality in patients with type 2 diabetes mellitus (DM). Therefore, it is important to identify polymorphism of the candidate genes critical in the pathogenesis and treatment of CAD. We focused on six lipoprotein metabolism and inflammatory genes namely, MIF, APOC1, LDLR, APOE, APOD and APOA5 in CAD with type-2 diabetes (T2DM).

Methods: Unselected 493 Turkish CAD (coronary lesion with 50-100% stenosis) and control (coronary lesion with <30% stenosis) were genotyped for APOA5 rs662799, APOC1 rs11568822, APOD rs1568565, APOE rs7412/rs429358, and LDLR rs5930 polymorphisms using hydrolysis probes and MIF rs755622 polymorphism using hybridization probes in Real-Time PCR LC480 device. Blood samples were drawn before coronary angiography. Gensini and SYNTAX scores and myocardial blush grade (MBG) were assessed.
Results: When the case-control groups were stratified according to gender and T2DM, only in men, polymorphisms of APOC1, LDLR and APOE ε4 allele carriers were associated with T2DM in CAD ($p<0.05$). In contrast, in the same subgroups, compared to non-carriers, carriers of the MIF common allel and APOA5 rare allele show a protection against developing the T2DM in control group ($p<0.05$). In addition, LDLR gene polymorphism is associated with glucose level and HbA1C ($p<0.05$).
Conclusions: In CAD patients, the risk allele of CAD-related APOC1, APOE, LDLR gene polymorphisms were associated with T2DM.

P1.1.031.
RELATIONSHIP BETWEEN METABOLIC SYNDROME AND ORAL/DENTAL PATOLOGY

B. Gonzalez Navarro [1], E. Jané Salas [1], J. López López [1], A. Rodriguez Sánchez [2], X. Corbella [2], X. Pintó Sala [2]. [1] *Oral Health and Masticatory System Group, Institut Investigació Biomèdica de Bellvitge (IDIBELL), barcelona, Spain;* [2] *Bellvitge University Hospital, LHospitalet de Llobregat, barcelona, Spain*

Aim: In addition to abdominal obesity and insulin resistance, patients with metabolic syndrome (MS) often have a state of low-grade inflammation and the involvement of different organs, including the oral cavity. Whereas the association between periodontal disease and MS its well known, the association between MS and dental pathology, including caries and apical periodontitis, has not been well defined.
Methods: From October 2016 to June 2017, 100 consecutive patients with ischemic disease of atherothrombotic origin, between 35 and 60 years old, that attended a vascular risk unit of a tertiary hospital were studied. All patients underwent a general health examination, an analytical and an oral examination. The diagnosis of MS was defined according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria.
Results: The cohort was formed by 69 men and 31 women. Sixty-three patients met the metabolic syndrome criteria. 82,5% of the patients diagnosed with metabolic syndrome had a regular or deficient plaque index, against 69,4% of the control group. A statistically significant inverse correlation was observed between glucose levels and glycosylated hemoglobin with the number of teeth. In addition there is a statistically significant positive correlation between the CPO-D (index of decayed, lost by caries and filled teeth) with the two previous parameters.
Conclusions: Among patients who have had an ischemic disease of atherothrombotic origin, those with MS have a higher frequency of caries and oral pathology than those without MS. Further studies are needed to compare these data with those of the general non-ischemic population

P1.1.032.
ROLE OF CD4+ T CELLS IN ANGIOTENSIN II-INDUCED ATHEROSCLEROSIS AND PLAQUE VULNERABILITY IN 2-KIDNEY, 1-CLIP APOE-/-MICE

M. Pellegrin, K. Bouzourene, J. Aubert, L. Mazzolai. *Division of Angiology, University Hospital of Lausanne, Lausanne, Switzerland*

Aim: Angiotensin (Ang) II plays a pivotal role in atherosclerosis and vulnerable plaque development. CD4+ T cells are involved in atherogenesis; however their specific role in Ang II-induced plaque vulnerability remains unclear. This study therefore examined the effects of CD4 deficiency on Ang II-mediated vulnerable plaque in 2-kidney, 1-clip [2K1C] ApoE-/- mice.
Methods: ApoE-/- mice and ApoE-/- CD4-/- mice underwent left renal artery clipping to generate the 2K1C model (Ang II-dependent mouse model of renovascular hypertension and of vulnerable plaques).
Results: Four weeks after clipping, there was no significant difference in plaque size in both the aorta ($0.62\pm0.16\%$ vs. $0.80\pm0.11\%$) and aortic sinus ($132803\pm26377\mu m^2$ vs $139797\pm19414\mu m^2$) between ApoE-/- CD4-/- and ApoE-/- mice. Moreover, no significant differences were observed in plaque content of macrophages, smooth muscle cells and collagen as well as in area of necrotic core and fibrous cap between the 2 groups. Compared to ApoE-/- mice, aortic mRNA levels of pro-inflammatory cytokines IFN-γ, IL-2, IL-12p35, IL-1β, IL-18, IL-6 and TNF-α were unchanged in ApoE-/- CD4-/- mice. Further, no significant difference was found in aortic iNOS/Arg1 mRNA expression (M1/M2 macrophage balance marker) between the 2 groups. Splenic mRNA levels of markers for Th1 (T-bet and IFN-γ), Th2 (GATA3 and IL-4), Th17 (IL-17 and TGF-β) and Treg (IL-10 and Foxp3) cells did not significantly differ among the 2 groups. Mean arterial blood pressure, plasma renin activity and plasma total cholesterol were similar between the 2 groups.
Conclusions: CD4+ T cells might not be essential for triggering Ang II-dependent atherosclerosis in mice.

P1.1.033.
CYTOMEGALOVIRUS ACTIVATION IS ASSOCIATED WITH ENDOTHELIAL FUNCTION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

A. Lebedeva [1], E. Maryukhnich [1], W. Fitzgerald [2], E. Nikitskaya [1], N. Ryazankina [1], J. Grivel [2,3], A. Shpektor [1], L. Margolis [2], E. Vasilieva [1]. [1] *A.I. Evdokimov Moscow State University of Medicine and Dentistry, Moscow, Russia;* [2] *Eunice Kennedy Shriver National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, USA;* [3] *Sidra Medical and Research Center, Doha, QATAR*

Aim: It is well established that both inflammation and endothelial dysfunction play a significant role in atherosclerotic plaque destabilization and development of acute myocardial infarction (AMI). In spite of numerous studies, identification of infectious agents that may trigger these processes remains controversial, presumably due to the latency of most of the infections found in patients with atherosclerosis. Therefore, the objective of our work was to analyze the effect of productive infection of human cytomegalovirus (CMV) on endothelial function in patients with AMI.
Methods: We included 45 patients with AMI and 42 healthy volunteers, in whom we evaluated CMV infection by the detection of CMV DNA in plasma. Endothelial function was evaluated with brachial artery flow-mediated dilation (FMD)-test. Also, we analyzed a panel of 10 chemokines known to be upregulated by CMV infection.
Results: We found that in patients with AMI compared to healthy volunteers the production of CMV DNA was significantly elevated (1245.6 [0;2543.4] vs. 0 [0;910.8] copies/ml of plasma, $p = 0.011$) and the FMD-test results were markedly decreased (4.1% [0.0;10.3%] vs. 11.5% [7.5;15.2%], $p = 0.0001$). Furthermore, we found that the concentration of CMV DNA in plasma negatively correlated with the FMD-test results ($R = -0.42$, $p = 0.006$) and positively correlated with the level of macrophage inflammatory protein-1beta ($R = 0.31$, $p = 0.039$); the latter also negatively correlated with the FMD-test results ($R = -0.29$, $p = 0.009$).
Conclusions: Cytomegalovirus is activated in AMI patients and its production correlates with endothelial vasodilation and synthesis of pro-inflammatory chemokines.

P1.1.034.
THE STATES OF INTERLEUKIN-10 IN PLASMA IN PATIENTS WITH ACUTE CORONARY SYNDROME

A. Nizomov [1], K. Gulyamova [2], M. Mukhamedova [1,2]. [1] *Hospital National Holding Company UZBEKNEFTEGAS, Tashkent, Uzbekistan;* [2] *Republican Scientific Center for Emergency Medicine, Tashkent, Uzbekistan*

Aim: Study of interleukin-10 in acute coronary syndrome.