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in a randomized order, with a week of wash out between the two tests. The antioxidant-enriched snack was composed by two products – a beverage (green tea or berries juice) and a cereal bar – supplemented with vitamin E (12 mg per serving of beverage and 15 mg per serving of bar), vitamin C (90 mg per beverage and 80 mg per bar) and catechins (35 mg per serving of both). Placebo snacks were prepared to be identical to the active ones in terms of appearance and organoleptic properties. All subjects came in the morning, after overnight fasting, and underwent to a Flow Mediated Dilatation (FMD) test fasting, 2, and 3 hours after snack ingestion. Blood was drawn with the same time-course to evaluate changes in plasma antioxidant concentrations.

Results: We observed a significant and sustained decline in EF in the placebo meal 2 and 3 hours after snack ingestion (maximal post-ischemic vasodilation during FMD test decreased from $6.4 \pm 3.5\%$ at baseline to $4.9 \pm 2.6\%$ and $4.9 \pm 2.8\%$ respectively; $p < 0.05$ for both), whereas the assumption of the antioxidant-enriched snack was associated with a significant improvement ($p < 0.05$) of FMD values ranging from $5.4 \pm 3.3\%$ at baseline to $6.6 \pm 3.4\%$ after 2 and $6.5 \pm 3.5\%$ after 3 hours. In addition, we observed the appearance of measurable concentrations of epigallocatechin gallate at 2 and 3 hours (29.8 ± 22.1 and 27.8 ± 24.8 nMol/L, respectively) after the assumption of the antioxidant-enriched meal.

Conclusion: In a population of apparently healthy subjects, the supplementation of a mixed meal with antioxidants is able to reverse the temporary decline in endothelial function usually observed in the post-prandial phase, inducing an acute increase in endothelial-dependent vasodilation. These data suggest that functional foods supplemented with antioxidants (like snacks and beverages) could have a favourable effect on post-prandial EF, raising the speculation that their introduction in a regular diet might produce positive long term effect on atherosclerosis development.

50 SMOKING HABITS AS DETERMINANT OF CAROTID IMT IN PATIENTS IN PRIMARY AND SECONDARY PREVENTION

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Objective: To evaluate whether the contribution of smoking habits as a determinant of carotid artery IMT is dependent on the patient's atherosclerotic burden.

Methods: Patients in secondary prevention (SP) were matched for gender and smoking habits with patients classified in primary prevention (PP) because free of vascular events, even if ten years older ($n = 180$ per group). In both groups there were 87 never, 68 former and 25 current smokers.

Results: Years of smoking, cigarettes/day (cig/die) and packyears correlated with IMT better in SP than in PP patients. In former smokers, a stronger negative correlation with years elapsed since smoking cessation was observed in SP than in PP patients. Although selected to be 10 years older, patients in PP showed a lower IMT than those in SP (0.93 ± 0.33 vs 1.06 ± 0.34 ; $p = 0.009$). Similar results were obtained after patients stratification in never, former and current smokers. A general linear model confirmed, after data adjustment for age, cig/die and conventional vascular risk factors, that both prevention level ($p = 0.012$) and smoking habits ($p = 0.017$) were independently associated with carotid IMT. No additive effect between prevention level and smoking habits was observed.

Conclusions: Prevention level is associated with a thickened carotid intima media complex independently of variables descriptive of smoking behaviour and other conventional risk factors. Smoking habit is an important determinant of carotid IMT both in primary and secondary prevention patients. Funding: Research supported in part by Philip Morris USA Inc. and Philip Morris International.

51 IMPACT OF TCF7L2 POLYMORPHISM ON POSTPRANDIAL LIPOPROTEIN METABOLISM AND ADIPOKINE RESPONSES IN INSULIN RESISTANT AND INSULIN SENSITIVE SUBJECTS

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Background and Aims: Genetic factors predisposing to diabetes and atherosclerosis are unknown. Transcription factor-7-like 2 (TCF7L2) polymorphism is an emerging risk factor for diabetes by modulating beta-cell function. Recent data suggested TCF7L2 polymorphism modulates fasting triglyceride levels in familial hyperlipidemia and it is differentially expressed in adipocytes of diabetic and dyslipidaemic subjects, suggesting it may regulate adipokine secretion and lipoprotein metabolism. The impact of rs7903146C/T polymorphism on lipoprotein metabolism and adipokine response to fat ingestion is unknown. This study assess the relationship of TCF7L2 polymorphism on postprandial lipoprotein metabolism and on adipokines responses both in insulin resistant and in insulin sensitive subjects.

Methods: Thirty nondiabetic normolipidemic non-obese insulin resistant subjects and 30 age-, BMI-, sex-matched healthy insulin sensitive controls underwent a 10-hr oral fat test and circulating lipoprotein subfractions and adipokines were assessed at 2-hr intervals. Subjects were genotyped for TCF7L2 rs7903146 C/T and ApoE polymorphisms. The area under the

curve (AUC) and incremental area under the curve AUC (IAUC) of different plasma lipoproteins, adiponectin and resistin were computed by the trapezoid method. Data from the oral fat load were compared by ANOVA and Scheffé post hoc test after log normalization of skewed variables. Data were expressed as mean \pm SEM. Differences were considered statistically significant at $p < 0.05$.

Results: Within each group, plasma Tg, FFA, and VLDL subfraction responses were higher in TCF7L2 CT/TT than in CC carriers. LDL-C did not change throughout the test. Fasting plasma resistin was comparable between groups and increased significantly postprandially in both groups. Fasting plasma adiponectin was lower in insulin resistant than in insulin sensitive subjects, and significantly rose in the latter group, while it decreased in the former. In both groups TCF7L2 significantly predicted postprandial resistin and adiponectin responses, with TCF7L2 CT/TT carriers displaying a more unfavorable adipokine profile than TCF7L2 CC carriers. On multiple regression analysis, TCF7L2 polymorphism was an independent predictor of postprandial Tg ($B = 0.49$; $p = 0.004$), adiponectin ($B = -0.40$; $p = 0.01$) and resistin ($B = 0.43$; $p = 0.009$) responses.

Conclusions: TCF7L2 polymorphism enhances modulates postprandial lipoprotein metabolism and adipokine responses in both insulin sensitive and insulin resistant nondiabetic normolipidemic nonobese subjects. Targeting postprandial lipemia may improve lipid dysmetabolism in high-risk TCF7L2 genotypes.

52 EARLY CAROTID ATHEROSCLEROSIS IN OBESITY AND OVERWEIGHT IS INDEPENDENT OF HS-CRP IN A COHORT OF MEDITERRANEAN WOMEN: FINDINGS FROM PROGETTO ATENA

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The relationship between BMI and common carotid intima media thickness (IMT) or Apo B and bifurcation intima media thickness (IMT) has been evaluated within the framework of a population-based cohort study in women, aged 30–69, living in the metropolitan area of Naples, Southern Italy (Progetto ATENA). Serum cholesterol, HDL-cholesterol, LDL-cholesterol, Triglycerides, Insulin, HOMA, Apo B and hs-CRP have been measured in 390 women as a part of 5,062 participants of the cohort. In this group carotid ultrasound examination (B-Mode imaging) was performed in 3 sites for each artery, and intima-media thickness (IMT) has been calculated. The association between carotid IMT, BMI, Apo B, hs-CRP and Metabolic Syndrome has been evaluated taking into account different adjustment models. Women in the second and third tertile of BMI, compared with those in the first tertile, show the following OR for presence of increased IMT at common carotid site: II vs I tertile 2.16 ($p = 0.018$), III vs I tertile 1.95 ($p = 0.037$), p trend < 0.0001 ; adjusted for age, apoB tertiles and hs-CRP. Comparable finding were obtained when the model included age, Apo B tertiles, hs-CRP and Metabolic Syndrome. Women in the second and third tertile of Apo B, compared with those in the first tertile, show the following OR for presence of increased IMT at bifurcation site: II vs I tertile 1.63 ($p = 0.077$), III vs I tertile 2.32 ($p = 0.003$), p trend < 0.0001 ; adjusted for age, BMI tertiles and hs-CRP. Comparable finding were obtained when the model included age, BMI tertiles, hs-CRP and Metabolic Syndrome. These findings show that relationship between BMI and common carotid artery wall thickening or Apo B and bifurcation artery wall thickening are independent of hs-CRP concentration in a population of middle-aged women.

53 ASSOCIATION BETWEEN SMALL DENSE LDL PARTICLES AND EARLY ATHEROSCLEROSIS IN A SAMPLE OF MIDDLE-AGED WOMEN. FINDINGS FROM PROGETTO ATENA

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Background: The association between small dense LDL particles (sd-LDL) and early atherosclerosis has been evaluated in a sample of middle-aged women participated to a population-based cohort study, aged 30–69, living in the metropolitan area of Naples, Southern Italy (Progetto ATENA).

Patients and Methods: We analyzed the relation between sd-LDL and common carotid/bifurcation Intima Media Thickness (IMT) in 210 women. LDL particle separation was performed by Lipoprint System: 7 LDL subfractions were obtained, mean LDL particle size and LDL score (% of sd-LDL) were calculated.

Results: Multivariate analysis showed a significant association between common carotid IMT (upper 1.2 mm) and mean LDL particle size after controlling for age, Apo B and Metabolic Syndrome (OR 2.73; 95% CI 1.00–7.44; $p = 0.048$ for mean LDL particle size). In a subsequent multivariate analysis, after controlling for age and systolic pressure a significant association between bifurcation IMT (upper 1.29 mm) and mean LDL size was found (OR 1.94; 95% CI 1.04–3.59; $p = 0.035$ for mean LDL particle size). After controlling for age and HDL, bifurcation IMT remained related to mean LDL particle size (OR 1.86; 95% CI 1.02–3.40; $p = 0.041$ for mean LDL particle size).