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 of 1,1-alkyl tocopherol and 15 mg of β-carotene per 100 mL of beverage and 80 mg per bar) and catechinhes (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 Mediasediated Distraction (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 vasodilatation during FMD test decreased from 6.4±1.3% to baseline at 4.9±2.2% 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±1.3% to baseline at 6.6±3.4% after 2 and 6.3±3.5% after 3 hours. In addition, we observed the appearance of measurable concentrations of epigallocatechin gallate at 2 and 3 hours (29.8±2.21 and 27.8±2.14 mL/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 vasodilatation. 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.

51 IMPACT OF TC7FL2 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 (TC7FL2) polymorphism is an emerging risk factor for diabetes by modulating beta-cell function. Recent data suggested TC7FL2 polymorphism modulates fasting triglyceride levels in familial hyperlipidemia and it is differentially expressed in adipocytes of diabetic and dyslipidaemic subjects, suggesting it may regulate adipocyte secretion and lipoprotein metabolism. The impact of TC7FL2 polymorphism on lipoprotein metabolism and adipokines response to fat ingestion is unknown. This study assess the relationship of TC7FL2 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 concentrations were measured at 2, 6, 12, 24, 48 and 72 h after the test for TC7FL2 rs7903146 C/T and ApoC polymorphisms. The area under the curve (AUC) and incremental area under the curve AUC (AIAUC) of different plasma lipoproteins, adipokines and resistin were computed by the trapezoidal method. Data from the oral fat load were compared by ANOVA and Scheffe post hoc test (after log transformation), vitamin D (25(OH)D) and ApoC polymorphisms were assessed by Student’s t-test. Data were expressed as mean±SEM. Differences were considered statistically significant at p < 0.05.

Results: Within each group, plasma Tg, FFA, and VLDL subfraction responses were higher in TC7FL2 CT/TT than in CC carriers. LDL-C and Insulin changes 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 TC7FL2 significantly predicted postprandial resistin and adiponectin responses, with TC7FL2 CT/TT carriers displaying a more favorable adiponectin profile than the CC homozygotes. In the current study, postprandial expression analysis, TC7FL2 polymorphism was an independent predictor of postprandial Tg (β = 0.49; p = 0.004), adiponectin (β = -0.40; p = 0.01) and resistin (β = 0.43; p = 0.002) responses.

Conclusions: TC7FL2 polymorphism enhances modulates postprandial lipoprotein metabolism and adipokine responses in both insulin sensitive and insulin resistant nondiabetic normolipidemic normocholesterolemic subjects. Targeting postprandial lipemia may improve lipid dysmetabolism in high-risk TC7FL2 genotypes.

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

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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 ATENEA). Serum cholesterol, HDL-cholesterol, triglycerides, and glucose were measured in 3,083 women and 4,491 men (ratio 0.74). A dose-response relationship between Apo B and IMT at bifurcation was evaluated. In both groups TC7FL2 significantly predicted postprandial resistin and intima-media thickness (IMT) has been calculated. The association between carotid IMT, BMI, Apo B, hs-CRP and Metabolic Syndrome was analyzed taking into account different adjustment models. BMI 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 <0.001; adjusted for age, apolipoproteins and hs-CRP, comparable finding were observed whenhs 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 = 0.001); 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 ATENEA


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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 participating to a population-based cohort study, aged 30-69, living in the metropolitan area of Naples, Southern Italy (Progetto ATENEA).

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 (5% 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 biconfounding for age, Apo A and Metabolic Syndrome (OR 2.73; 95% CI 1.04-7.44; 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 1.04-3.99; p = 0.035 for mean LDL particle size). After controlling for age and HDL, bifurcation IMT remained as related to mean LDL particle size (OR 1.86; 95% CI 1.02-3.40; p = 0.041 for mean LDL particle size).