tail cuff method (SBP), and left ventricular (LV) dimensions (M-mode echo) were simultaneously recorded while rats were awake.

Results: After 6 months, K weighed 685.9±21 g vs 617.1±49.9 (C) and 630.7±42.6 (L) ($p<0.05$) and showed SBP of 146.6±10.6 mmHg vs 136.9±7.3 and 135.1±8.2, respectively ($p<0.05$). Plasma glucose was 149.4±16.3 mg/dl (K) vs 119.3±13.5 (C), and 115.0±6.5 (L) ($p<0.001$); triglycerides were 182.6±86.6 mg/dl (K) vs 72.6±26 (C), and 84.5±34.2 (L) ($p<0.01$); while plasma concentration of HDL-Cholesterol showed no differences among groups. LV diastolic diameter was 6.78±0.35 mm (C) vs 7.46±0.3 (K; $p<0.05$), and vs 7.1±0.55 (L; ns). LV wall thickness was 0.42±0.03; 0.37±0.03; and 0.38±0.03 mm, respectively (C vs K and L, $p<0.05$). LV diastolic volume (ml) was 0.27±0.04 (C) vs 0.35±0.04 (K; $p<0.01$); and vs 0.3±0.07 (L; ns). Cardiac output (ml/min) was 113.96±22.8 in C vs 161.15±28.87 in K ($p<0.01$), and vs 129.85±38.09 in L (ns).

Conclusions: In rats, chronic consumption of coke affects body weight, blood pressure, glucose, and tryglicerides, thus reproducing most of the features of metabolic syndrome. Furthermore, these animals showed LV dilatation and remodeling. These deleterious effects on metabolism and cardiac geometry were not seen in animals drinking light coke, thus indicating that they were largely due to the high calorie intake from sucrose in regular drink.

3A.3 EFFECT OF FOLIC ACID TREATMENT ON OXIDATIVE STRESS AND NITRIC OXIDE ACTIVITY IN PATIENTS WITH METABOLIC SYNDROME

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Objectives: Alterations in microvascular function play an important role for the development of diabetic nephropathy and retinopathy. Experimental data suggest increased vascular production of superoxide and of nitric oxide (NO), both subsequently reacting to highly toxic peroxynitrite. We hypothesized that folic acid treatment may reduce oxidative stress and microvascular NO activity in subjects at high risk for diabetes.

Research Design and Methods: Male subjects with the metabolic syndrome (MS, n=49) and controls with similar body mass index (BMI) (n=26) were included in a randomized, double-blind, cross-over designed trial. After treatment with placebo or folic acid (5mg/day) for four weeks, oxidative stress was determined as reduced to oxidized glutathione levels in erythrocytes (GSH/GSSG ratio). Renal and retinal NO activities were assessed *in vivo* as renal plasma flow (clearance-technique) and retinal blood flow (laser doppler scanning flowmetry) responses to NO synthase inhibition with N(G)-monomethyl-L-Arginine (L-NMMA).

Results: BMI was similarly elevated in subjects with and without the MS (34±4 vs 32±3 kg/m^2, n.s.). In all subjects, GSH/GSSG ratio increased from 42±80 to 100±198 ($p=0.04$). Only in subjects with MS, folic acid reduced the responses of renal plasma and retinal blood flow to L-NMMA compared to placebo (−48±40 vs −66±48 ml/min/1.73 m^2, $p=0.03$; and −9±35 vs −27±44 U, $p=0.04$).

Conclusions: Folic acid reduces oxidative stress in obese subjects, and reduces NO activity in renal and retinal circulations in those with the MS. We hypothesize that folic acid treatment may be useful in this setting to prevent microvascular damage, e.g. *via* reduced generation of peroxynitrite.

3A.4 GLOMERULAR HYPERFILTRATION AND INCREASED PROXIMAL SODIUM REABSORPTION IN TYPE 2 DIABETES IN A MIDDLE-INCOME COUNTRY IN THE AFRICAN REGION

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Objective: Glomerular hyperfiltration is a well-recognized early renal event in diabetic patients. However, so far, very few studies have examined renal function and renal sodium handling in a large population of type 2 diabetics of African origin. Therefore, the aim of this study was to measure renal hemodynamics and renal sodium handling and to establish the prevalence of glomerular hyperfiltration (GHF) in type 2 diabetics as compared to non-diabetic Africans.

Design and Method: This cross sectional study conducted in the Seychelles islands included families of East African descent, with high prevalence of hypertension. Glomerular filtration rate (GFR), effective renal plasma flow (ERPF) and proximal tubular sodium reabsorption were measured using inulin-, PAH- and endogenous lithium clearance, respectively; 24-hour urine was collected on the preceding day.

Results: Of the 363 participants, 24 (6.6%) had impaired glucose tolerance (IGT) and 36 (9.9%) type 2 diabetes (DM); 230 participants (63.3%) had hypertension. Compared to participants with normal fasting glucose (NFG), participants with IGT or DM were older (median age 47 vs. 49 vs. 44 years), more frequently men (44 vs. 52 vs. 58%) and had higher mean arterial blood pressure (MAP) (99 vs. 105 vs. 105 mmHg), but similar body mass index (BMI) (27.4 vs. 28.5 vs. 28.9 kg/m^2), plasma renin activity (PRA), aldosterone-levels (53 vs. 58 vs. 59 pg/ml), ERPF (441 vs. 485 vs. 406 ml/min) and 24h urinary sodium excretion (102 vs. 86 vs. 110 mmol/24 hours). The prevalence of GHF, defined as GFR >140 ml/min, was 17.2%, 29.2% and 52.8% in participants with NFG, IGT and DM, respectively (P trend <0.001); adjusted to age, sex, and MAP and compared to NFG, the odds ratio for GHF was 1.99 (95% CI 0.73–5.44) for IGT and 5.88 (2.39–14.45) for DM. Participants with IGT or DM had significantly lower clearance and fractional excretion of lithium than NFG participants, which indicates increased proximal sodium reabsorption, and their filtration fraction was higher (0.24 vs. 0.27 vs. 0.30; P trend <0.001).

Conclusion: African patients with type 2 diabetes are characterized by a high prevalence of GHF and an enhanced proximal sodium reabsorption, independently of BMI, MAP, sodium intake, PRA and aldosterone. These two observations might contribute to the higher incidence of hypertension and diabetic nephropathy in black patients with diabetes.

3A.5 DIFFERENTIAL SYMPATHETIC ACTIVATION IN MUSCLE AND SKIN NEURAL DISTRICTS IN THE METABOLIC SYNDROME

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Objectives: The present study was designed at determining whether and to what extent the activation of the sympathetic activation reported in the metabolic syndrome has a generalized or rather a regional distribution over the cardiovascular system.

Design and Methods: In 16 untreated patients with metabolic syndrome, 12 essential hypertensives, 12 obeses and 14 lean healthy normotensive controls, we measured blood pressure (Finapres), heart rate (EKG), venous plasma norepinephrine (HPLC) and postganglionic sympathetic nerve traffic in the skeletal muscle and in the skin areas (microneurography at peroneal nerve). The muscle and skin nerve traffic measurements were made in randomized sequence and included evaluation of skin sympathetic responses to arousal (acoustic stimulus).

Results: The four groups of subjects had superimposable ages (49.4±2.8, mean±SEM). Compared with controls, muscle sympathetic nerve traffic values were significantly higher in subjects with hypertension and in those with obesity (37.2±3.3 vs 51.2±2.8 and 52.0±3.0 bursts/100 heart beats, respectively, $P<0.01$), a further significant increase being detected in subjects with the metabolic syndrome (61.0±3.2 bursts/100 heart beats, $P<0.05$). In contrast, skin sympathetic nerve traffic was not significantly different in the four groups (15.4±1.0 vs 13.0±0.7, 14.3±1.3 and 12.5±0.8 bursts/minute, respectively, P=NS), this being the case also for the skin sympathetic responses to the acoustic stimulus. In the population as a whole, resting MSNA, but not SSNA, significantly and directly correlated with mean arterial pressure (MSNA: $r=0.30$, $p<0.05$ and SSNA: $r=0.04$, P=NS) and body mass index values (MSNA: $r=0.53$, $P<0.001$; SSNA: $r=0.05$, P=NS). Plasma norepinephrine was also directly and significantly related to MSNA ($r=0.37$, $P<0.02$), but not to SSNA ($r=0.07$, P=NS).

Conclusions: The present data provide the first direct evidence that in the metabolic syndrome the sympathetic activation is not uniformly distributed over the cardiovascular system. The different behaviour of skin and muscle sympathetic neural drive is likely to depend on the different impact of the metabolic syndrome components on the reflex and non-reflex mechanisms modulating muscle and skin adrenergic neural drive.

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ABSTRACT BOOK