Median (10th-90th percentile) of age and estimated glomerular filtration rate (GFR) were 46 (24-69) and 64 (24-104). The analyzed data were: age, gender, body mass index (BMI) serum albumin, creatinine, calcium and phosphate; 24-h urine creatinine, phosphate (P),proteinuria (DP). Estimated parameters includes: eGFR, fractional P excretion (FEP), 24-h P excretion (24-h UP), and P clearance (CP). Dietary protein intake (DPI) was based on 24-h urinary urea excretion. No significant differences in serum phosphate were found in groups with various DPI. FEP, 24-h UP and CP were significantly higher in higher DPI range. DPI was positively associated with 24-h UP ($\beta$=0,287, $p < 0.000001$) in multivariate model adjusted for age, gender, DP, eGFR, serum P, FEP, BMI, and Ca. Thus, DPI is considered to be the independent factor influencing urinary P excretion and hence contributing to progression of mineral and bone disease in renal dysfunction.

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**PATIENT SATISFACTION AND DIETARY OUTCOMES FROM ATTENDING A MULTIDISCIPLINARY CYSTINURIA CLINIC**

Angela Doherty, Kathie Wong, Kay Thomas, Gosia Kamoga
*Guy's and St.Thomas' Foundation Trust, London, England*

Cystinuria is a genetic disease that leads to frequent cystine stone formation. A reduced methionine (precursor to cystine) diet has been recommended for cystinuria patients, involving maintenance of healthy weight, limiting intake of animal protein and salt whilst increasing vegetable protein, fruit, vegetables and fluid intake. The ability of patients to adhere to these dietary regimes has been questioned. We evaluated the dietetic service and dietary changes within our multidisciplinary cystinuria clinic using food frequency questionnaires (ffqs), dietary change questionnaires (dcqs) and diet histories. Most patients were seen by the dietitian 6–12 monthly.

100% of patients felt they benefited from dietetic input. There was a move away from ffqs due to them being difficult and cumbersome to complete. Dcqs and diet histories revealed 23/28 increased fruit and vegetable intake, 20/28 decreased salt intake, 20/28 decreased animal protein intake, 14/28 increased vegetable protein intake and 9/28 increased fluid intake. There were a few discrepancies in reported change between dcqs and diet histories, supporting the use of multi-source feedback for diet analysis.

Patients had varying degrees of success with making changes to each dietary parameter. Multi-source diet analysis allowed us to develop tailored consultations. All patients made some positive dietary changes which may help prevent stone formation. The areas of least change were fluid (due to changes made prior to clinic attendance) and vegetable protein. Our results support the need for continued dietetic input. These results should be re-audited to check that patients are maintaining the changes made.

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**52**

**EVALUATION OF NUTRITIONAL STATUS IN PERITONEAL DIALYSIS (PD) PATIENTS**

Max Dratwa [1], Christian Verger [2]
[a]*CHU Brugmann, Brussels, Belgium*
[b]*RDPLF, Pontoise, France*

In 2009, analysis of the French Language Peritoneal Dialysis Registry (RDPLF) data revealed that 6 patients out of 355 (1,7%) were transferred to HD because of malnutrition after a median period of 31 months. Furthermore, malnutrition was responsible for the death of 5.1 % patients after a median period of 14 months. So, as nutritional status has been shown to impact morbidity and mortality of dialysis patients, we set out to measure this status in incident PD patients of 2 European centers using various validated methods. The techniques used were:

anthropometric,BMI and biochemical measurements, SGA, Brandes score, total (renal and peritoneal) creatinine excretion, dietician records, handgrip strength, DEXA, multifrequency electrical bioimpedance (TBIA). Daily dietary protein intake was less than 1g/kg ideal BW in 35 % of pts while 38% ate more than 1.2 g/kg; daily total (oral and peritoneal) caloric intake was 28 $\pm$ 8 kcal/kg. Fifteen % of patients (median age 71) had a very low serum albumin level ( $<$ 30 g/l) while 50 % maintained their albumins $>$ 50 g/l (median age 64 years). Very significant correlations were found between: 1) estimation of fat mass by skinfold thickness and that modelised from the water volume determined by TBIA ($r^2$: 0.56, $p < 0.0001$); 2) brachial circumference and lean body mass determined by TBIA (but no correlation between middle arm circumference and handgrip strength) ; 3) muscle mass determined by creatinine kinetics and TBIA ($r^2$: 0.68, $p < 0.001$); 4) muscle mass estimated by brachial circumference and lean body mass estimated through creatinine kinetics; 5) handgrip strength and lean body mass determined both by TBIA and by creatinine kinetics ($r^2$: 0.59, $p < 0.0001$); 6) lean body mass and age (but different between males and females); 7) protein losses in the dialysate and lean body mass derived from creatinine kinetics. In conclusion, malnutrition is more threatening as patients age. Patients with a low initial lean body mass and low protein intakes should be closely monitored such as protein losses do not counterbalance the theoretical advantages of a higher dialysis dose.

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**53**

**ALDOSTERONE AND CORTISOL AFFECT THE RISK OF SUDDEN CARDIAC DEATH IN HEMODIALYSIS PATIENTS**

*Background:* Sudden cardiac death is common and accounts to a large extent for the excess mortality of patients on maintenance dialysis. It is unknown whether aldosterone and cortisol increase the incidence of sudden cardiac death in dialysis patients.
*Methods:* We analyzed data from 1255 hemodialysis patients with type 2 diabetes mellitus, participating in the German Diabetes and Dialysis Study (4D Study). Aldosterone ( $<$ 15 pg/mL; 15–100 pg/mL, 100–200 pg/mL, $>$ 200 pg/mL), and cortisol (quartiles) were determined at baseline and patients were followed up for a median of 4 years. By Cox regression analyses hazard ratios (HR) were determined for the effect of aldosterone, cortisol and its combination on sudden death and other cardiovascular outcomes.
*Results:* The mean age of the patients was 66 $\pm$ 8 years (54% male). The median levels of aldosterone was 15 pg/mL and of cortisol 16.8 µg/dL. Patients with aldosterone levels $>$ 200 pg/mL had a significantly higher risk of sudden death (HR 1.69; 95% CI 1.06-2.69) compared to those with an aldosterone $<$ 15 pg/mL. The combined presence of high aldosterone ( $>$ 200 pg/mL) and high cortisol ( $>$ 21.1 µg/dL) levels increased the risk of sudden cardiac death strikingly compared to patients with low aldosterone ( $<$ 15 pg/mL) and low cortisol ( $<$ 13.2 µg/dl) levels (HR 2.86, 95% CI 1.32-6.21). Furthermore, all-cause mortality was significantly increased in the patients with high levels of both hormones (HR 1.62, 95% CI 1.01-2.62).
*Conclusions:* The joint presence of high aldosterone and high cortisol levels strongly increased the incidence of sudden cardiac death as well as all-cause mortality in hemodialyzed type 2 diabetic patients. Whether blockade of the mineralocorticoid receptor decreases the risk of sudden death without causing side effects must be examined in future trials.

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**54**

**CHRONIC KIDNEY DISEASE IN ADULTS WITH THE METABOLIC SYNDROME IN BENIN CITY: PREVALENCE AND CORRELATES.**

Oghenekaro Egbi [1], Evelyn Unuigbe.I [2], Efosa Oviasu [2], Louis Ojogwu [1,2]
[1] *Department of Medicine, Federal Medical Centre,Yenagoa,Bayelsa State, Nigeria*
[2] *Department of Medicine, University of Benin Teaching Hospital, Benin City, Nigeria*

The Metabolic Syndrome (MetS) is a constellation of interrelated risk factors that appear to directly promote the development of atherosclerotic cardiovascular disease. Recently, an association with chronic kidney disease (CKD) has been documented severally in literature. Only a few prevalence studies on MetS exist in this part of the world. The aims of the study were to determine the prevalence of CKD in patients with MetS and to determine the association between MetS- related variables and CKD. The updated NCEP ATP III guideline was used for definition of MetS. Patients had their fasting plasma glucose, serum lipids, urea and creatinine as well as spot urine albumin: creatinine ratio done. Two hundred and twenty two patients completed the study. The prevalence of CKD was 20.3% while 20.0% had abnormal ACR value. Body mass index (BMI), waist/hip ratio, diastolic blood pressure, total cholesterol and LDL-cholesterol were associated with CKD in univariate analysis but only BMI and diastolic blood

pressure were independent predictors of CKD in multivariate analysis. There was a graded relationship between the number of MetS traits and the presence of CKD and also between MetS traits and ACR.

In conclusion, CKD is prevalent in patients with the metabolic syndrome and may be due to a synergistic effect of the various components of the syndrome. Diastolic blood pressure and obesity may predict CKD in MetS patients. Albuminuria may also be prevalent in MetS patients; increasing with increasing number of MetS traits.

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**55**

**PREVALENCE OF HOSPITAL MALNUTRITION IN RENAL PATIENTS**

Atilano-Carsi X., Campos-Collado AX., Miranda-Alatriste P, Leyva-Rendon F., Rosas-Jimenez M., Correa-Rotter R, Espinosa-Cuevas MA.*
*Depto. Nefrologa y Metabolismo Mineral. Instituto Nacional de Ciencias Medicas y Nutricion "Salvador Zubiran", Mexico, D.F*

The prevalence of protein energy wasting varies enormously depending on definition and parameters used. Most evidence include clinical stable patients only. There is few evidence in pre dialysis patients. The purpose of this study was to assess nutritional status at hospital admittance of renal patients NKF-DOQI 2–5 predialysis and dialysis patients and its association with morbidity and mortality. Nutritional assessment was done during the first three days of admission to the hospital with a composite nutritional index in 907 renal patients (589 pre dialysis and 318 dialysis). Length of stay and frequency of hospitalizations, mortality and comorbidities were registered. ANOVA, Cox and Kaplan-Meier statistics were done where needed. A prevalence of 76% of moderate to severe malnutrition was found to the general population being more prevalente in dialysis patients (81% dialysis vs 73% pre dialysis) ($p \leq 0.05$). Hazard ration of malnutrition was 2.07 and 2.57 in DOQI 4 and 5 non dialysis patients respectively ($p < 0.05$). Hazard ratio of mortality was 2.91 and 8.67 in moderate and severe malnutrition respectively predialysis patients ($p < 0.05$) and 2.72 and 3.22 in moderate and severe malnutrition respectively in dialysis patients ($p < 0.05$). Survival during the follow up was inferior in severe malnutrition in both groups ($p < 0.05$). In conclusion a prevalence of 76% of malnutrition was found in this cohort of hospitalized renal patients, greater than reported elsewhere being in dialysis patients more prevalent. Malnutrition was negatively associated with survival.

*Corresponding author.

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**56**

**CALCINEURIN A (CA) INHIBITION SUPRESSES PROTEIN SYNTHESIS VIA AMP DEPENDENT KINASE (AMPK).**

H. Franch, C. Ding, S. Zoromsky, S. Williams, J. Gooch
*Atlanta VAMC & Emory University, Decatur & Atlanta, GA, USA.*

Diabetes stimulates CA activity in the kidney and CA inhibitor cyclosporine A (CYA) or CA$\beta$ isoform knock-out (CA$\beta$ -/-) block diabetic renal hypertrophy.CA regulates the mammalian target of rapamycin (mTOR) to reduce protein synthesis (PS) during cardiac hypertrophy.To study this pathway, NRK-52E and SV40 transformed CA$\beta$ +/+ and -/- proximal tubule cell lines were treated with 10 nM epidermal growth factor (EGF) and/or 8 μM CYA. After 48 hrs of CYA in NRK-52E cells, EGF-induced protein/well was $30 \pm 4$% lower with decreased PS ($49 \pm 2$%) rather than increased protein breakdown. In CA$\beta$ -/- cells, protein/well (20+2%) and PS (16 $\pm$1%) were lower and CYA did not decrease PS further. In CA$\beta$+/+ cells, CYA reduced PS 20+2%. However, CYA did not block mTOR signaling as measured by phosphorylation (P) at S2448 or by p70S6 kinase P at T389. CYA also did not alter the mTOR dependent pathway of macroautophagy. However, CYA in NRK-52E or CA$\beta$ -/- cells showed increased activation of the metformin-sensitive energy sensor, AMPK, as measured by P of T172 by ~5- or > 10-fold, respectively. AMPK reduces PS via P of eukaryotic elongation factor 2 (eEF2) at T356 and CYA increased eEF2 P by~3 fold.We conclude CA inhibition reduces PS in renal hypertrophy via a novel pathway involving AMPK and eEF2 rather than by mTOR and p70S6 kinase. Activation of AMPK or altered energy metabolism via CA$\beta$ may be important in CA inhibitor nephrotoxicity.

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**HIGH PROTEIN DIETS STIMULATE RENAL PROTEIN OXIDATION IN EXPERIMENTAL DIABETES.**

H. Franch, S. Zoromsky, S. Williams, C. Din
*Atlanta VAMC & Emory University, Decatur & Atlanta, GA, USA.*

Calorie restriction increases macro- and chaperone-mediated autophagy (MA & CMA) protecting against organ damage by destroying oxidized proteins (OX) and p62/sequestisome 1 labeled aggregates (p62). Altering dietary protein changes kidney damage in DM, but the effect on renal cortical MA, CMA, OX and p62 is unknown. 60 mg/kg streptozotocin Diabetic (D) Sprague-Dawley rats (200 g) or control (C) were fed isocaloric 12% (L), 23% (M) or 40% (H) protein diets for 21 days (6 groups, each n= 7-11). Body weight at 21 days was 16-22% lower than C in all D rats ($p < 0.01$, NS between diets). Blood glucose was lower in DH vs. DL diets (408+25vs. 482+23 mg/dl, $p < 0.05$). Kidney wt/body wt increased in all DM groups, but increase was greater in DH than DL (104+13 vs. 66+11%, $p < 0.02$). MA, CMA, and p62 were estimated by western blotting for light chain 3b II/I ratio, lysosome associated membrane protein 2a, and p62 respectively, while OX was measured by Oxyblot. D reduced CMA by 65-76% ($p < 0.05$ vs. C) while H diet reduced it by ~45% in C and D rats (NS). MA increased in all D, but was ~10 fold higher in HD vs. HC or LD ($p < 0.02$). There was a trend toward increased OX with D, but only HD reached significance vs. all C (95+32%, $p < 0.05$). The trend for p62 to increase with D or H was NS. We conclude that despite lower glucose, H diets worsen diabetic renal hypertrophy and cortical OX possibly by suppressing CMA. Increases in MA by D and H prevent p62 protein aggregates from accumulating.

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**58**

**SERUM OMEGA-3 FATTY ACID LEVELS IN A LARGE INCIDENT U.S. HEMODIALYSIS POPULATION**

Allon Friedman, MD[1], Zhangsheng Yu [1], Rebecca Tabbey [1], Cheryl Denski [1], Ravi Thadhani [2], Yong Li [3], Bruce Watkins [3]
[1] *Indiana University, Indianapolis, IN, USA*
[2] *Massachusetts General Hospital, Boston, MA, USA*
[3] *University of Connecticut, Storrs, CT, USA*

Long chain omega-3 (n-3 PUFA) fatty acids may influence clinical outcomes in hemodialysis patients. We therefore performed the largest characterization to date of n-3 PUFA levels in an incident hemodialysis population.

Serum n-3 PUFA were measured in samples obtained at baseline in a 400 person subgroup of the ArMORR study, a large representative cohort of U.S. incident hemodialysis patients. n-3 PUFA were measured by gas chromatography, with phospholipid (PL) and triglyceride (TG) fatty acids being separated by solid phase extraction. Signed rank test was used to compare PL and TG fractions.

Mean ($\pm$SD) age was $66 \pm 14$ years and body mass index $26 \pm 7$ kg/m$^2$. 58% were men and 68% white. Cause of ESRD was diabetes and hypertension in 47% and 40% of subjects, respectively. Total serum 20:5n-3 (EPA) (by mean weight %) was $0.09 \pm 0.36$ (PL vs. TG: $0.09 \pm 0.43$ vs. $0.07 \pm 0.30$, $p = 0.14$), and total serum 22:6n-3 (DHA) was $1.46 \pm 0.72$ (PL vs. TG: $3.03 \pm 1.24$ vs. $0.21 \pm 0.40$, $p < 0.001$). Minimal 22:5n-3 (DPA) was detected. Total long chain n-3 PUFA levels were $1.55 \pm 0.95$ (PL vs TG: $3.13 \pm 1.47$ vs. $0.28 \pm 0.64$, $p < 0.001$).

In summary, n-3 PUFA levels in this large U.S. cohort are low relative to published reports of levels in the general population or even in hemodialysis patients outside the U.S., making this a potential target for intervention.1 In addition, future enquiries into n-3 PUFA in hemodialysis cohorts should focus on the phospholipid fraction since we have demonstrated that, similar to the general population, this is where they primarily accumulate.

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