Free Communications  
Annual Meeting Danish Society of Nephrology 2016

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Predicting albuminuria response to spironolactone treatment with urinary proteomics in patients with type 2 diabetes and hypertension  
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Association of apolipoprotein M with cardiovascular risk factors in patients with chronic kidney disease  
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No evidence for long term effect on mortality, diabetes or infections of mannose-binding lectin genotypes (MBL2) among uremic patients  
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Treatment of hypertension using telemedical home blood pressure measurements
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Ultrasound Vector Flow Imaging for Surveillance of Arteriovenous Fistulas
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Can identification and standard of care of patients with AKI in emergency medical units be improved through an interactive smart phone app?
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**Chronic kidney disease – Clinical Studies**
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Oral Magnesium Supplementation Improves Serum Calcification Propensity in Chronic Kidney Disease Stage 3-4
Iain Bressendorff, Ditte Hansen, Morten Schou, Andreas Pasch, Lisbet Brandi

Sexual dysfunction and hyperprolactinemia in uremic men.
Rikke Juul-Sandberg, Ann Madsen, Ellen Grodum, Gudrun Kjær Steffensen, Karoline Schousboe
Inhibition of the FGF receptor decreases FGF23 levels in uremia

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**Background:** High levels of FGF23 and PTH are in uremia associated with increased morbidity and mortality. In uremia resistance to the inhibitory effect of FGF23 on PTH has been postulated. Our aim was to study the effect of FGF receptor inhibition (FGFRI) on FGF23 and PTH regulation.

**Methods:** Uremia was induced in rats by 5/6 nephrectomy and a high phosphate diet. After 8 weeks, 20 µg of the pharmacological FGFRI, PD173074, or vehicle was administered to uremic (U) and age-matched control (C) rats. Plasma intact FGF23, PTH and FGF23 mRNA in bone and kidney together with renal expression of target genes were measured after 5hr.

**Results:** U rats compared to C rats had significant higher levels of creatinine (72±11 vs. 32±1 µM), phosphate (2.57±0.18 vs. 2.05±0.08 mM), PTH (932 (355-2929) vs. 224 (169-290) pg/ml) and iFGF23 (1925±544 vs. 367±21 pg/ml) (all p<0.05). FGFRI resulted in a significant decrease in plasma iFGF23 to a third of baseline in both C and U rats after 5 hours (54±18 and 738±174 pg/ml, respectively (p<0.01)). iFGF23 was stable in vehicle groups. Plasma Ca²⁺ and phosphate remained unchanged in all groups, whereas PTH rose significantly in FGFRI C and U rats to 392 (281-1079) and 2719 (1579-16912) pg/ml (p<0.05). Expression of FGF23 in bone was down-regulated by FGFRI in C and U rats (veh C 1.54±0.23 vs. FGFRI C 0.15±0.02 and veh U 2.79±0.61 vs. FGFRI U 0.14±0.03), whereas it had no effect on the injured kidney’s expression of FGF23 (veh U 1.56±0.49 vs. FGFRI U 1.06±0.23). FGFRI had no effect on renal NaPi2a and NaPi2c mRNA in U rats in contrast to a significant upregulation in C rats (p<0.05).

**Conclusion:** Inhibition of the FGF receptor, FGFRI, by PD173074 suppressed FGF23 plasma levels and the bone’s expression of FGF23 in both normal and uremic rats, but had no effect on the expression of FGF23 in the injured kidney. PTH was similarly and significantly increased in both groups, which is contrasting the hypothesis of parathyroid resistance to FGF23 in uremia.
Rapid down-regulation of Klotho in the obstructed kidney  
No compensatory up-regulation in the contralateral kidney

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Background: The anti-ageing hormone, Klotho, is a renoprotective protein alleviating acute kidney injury and promoting kidney regeneration. The kidney is a major source of Klotho. In the present study the time course of Klotho changes in experimental unilateral ureter obstruction (UUO) and in the contralateral kidney was followed.

Methods: UUO rats (n=42) were examined at 0, 1, 2, 3, 4, 7, and 10 days and the obstructed kidney was compared to the contralateral kidney and to kidneys from unilateral nephrectomized (UNX) control rats (n=36) as well as normal kidneys. Kidney Klotho mRNA, the renoprotective BMP7 mRNA, the stem cell marker LGR5+ together with markers of fibrosis were examined.

Results: Unilateral ureter obstruction resulted in severely decreased Klotho expression already at day 1, which decreased further progressively to day 10. Day 0: 1.9 ± 0.3; day 1: 0.9 ± 0.2 (p<0.05), day 10: 0.1 ± 0.02 (p<0.01). Similarly LGR5+ was significantly reduced (p<0.05) in the obstructed kidney, suggesting stem cell depletion. The decrease in Klotho expression in UUO kidney was associated with a decreased expression of BMP7 and an increased expression of TGFb and induction of periostin. The contralateral kidney had similar expression of Klotho compared to normal and UNX kidneys.

Conclusion: In a unilateral ureter obstruction model a very rapid decrease in Klotho expression took place in the obstructed kidney. The contralateral kidney had however no compensatory increase in Klotho.
Deletion of T-type calcium channels Ca\textsubscript{v}3.1 attenuates endothelial dysfunction in aging mice
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**Introduction:** Endothelial dysfunction is associated with aging and is accelerated in chronic kidney disease. T-type calcium channels are present in human renal blood vessels and T-type blockers are suggested to protect against endothelial dysfunction and lower proteinuria in patients with hypertension. It was hypothesized that T-type channels contribute to the endothelial dysfunction of aging.

**Methods:** Endothelial function was determined in mesenteric arteries (perfusion) and aortae (myography) of young and old wild type and Ca\textsubscript{v}3.1 knock-out mice. Furthermore, NO production was measured by fluorescence imaging.

**Results:** Endothelium-dependent dilatation in mesenteric arteries and aortae was diminished in arteries of aging wild type mice, but increased in Ca\textsubscript{v}3.1\textsuperscript{−/−} mice. A nitric oxide synthase inhibitor (L-NAME) abolished the dilatations in both types of blood vessels. NO levels were significantly attenuated in mesenteric arteries of old compared to young Wt mice. In contrast, NO levels were significantly increased with age in Ca\textsubscript{v}3.1 knock-out mice. However, the level of activated (phosphorylated) eNOS was significantly increased in aorta from aged Ca\textsubscript{v}3.1\textsuperscript{−/−} mice.

**Conclusion:** T-type calcium channel deficient mice are protected against age-dependent endothelial dysfunction. Changes in NO levels are responsible for this phenomenon. In perspective, T-type calcium channel blockers could therefore protect against endothelial dysfunction in patients.
Predicting albuminuria response to spironolactone treatment with urinary proteomics in patients with type 2 diabetes and hypertension

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**Background** The mineralocorticoid receptor antagonist spironolactone significantly reduces albuminuria in patients with diabetes. Prior studies have shown a large between patient variability in albuminuria treatment response. We previously developed and validated a urinary proteomic classifier that predicts onset and progression of chronic kidney disease. Here we tested whether the proteomic classifier predicts albuminuria response to spironolactone treatment.

**Methods** We performed a post hoc analysis in a double-blind randomized clinical trial with allocation to either spironolactone 25-50 mg/ day (n=57) or placebo (n=54) for 16 weeks. Patients were diagnosed with type 2 diabetes and resistant hypertension. Treatment was an adjunct to renin-angiotensin-system inhibition. Primary endpoint was the percentage changes in urine albumin to creatinine ratio (UACR) during treatment. Capillary electrophoresis mass spectrometry was used to quantify urinary peptides at baseline. The previously validated combination of 273 known urinary peptides was used as proteomic classifier.

**Results** Spironolactone reduced UACR relative to placebo by 50%, although with a large between patient variability in UACR response (5th to 95th percentile, 7 to 312%). An interaction was detected between CKD273 and treatment assignment (Beta -1.09, p=0.026). Higher values of CKD273 at baseline were associated with a larger reduction in UACR in spironolactone group (beta=-0.70, p=0.05) but not in placebo group (beta=0.39, p=0.25). Stratified in tertiles of baseline
CKD273, reduction in UACR was greater in the highest tertile 63 % (95 % CI, 35 to 79%) as compared to the two other tertiles combined 16 % (-17 to 40%) (p=0.011).

**Conclusion** A urinary proteomics classifier can be used to identify individuals more likely to show an albuminuria lowering response to spironolactone. These results suggest that urinary proteomics may be a valuable tool to tailor therapy, but confirmation in clinical trials is required.

**Association of apolipoprotein M with cardiovascular risk factors in patients with chronic kidney disease**

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**Background:** Apolipoprotein M (apoM) is expressed in the liver and the kidney proximal tubular cells. Plasma apoM is a carrier of sphingosine-1-phosphate (S1P), and is mainly bound to HDL-particles. Both apoM and S1P show anti-atherogenic and endothelium protective actions. In mouse models of atherosclerosis, moderate chronic kidney disease (CKD) is associated with increased plasma levels of apoM and S1P. The present study explored whether plasma levels of apoM and S1P were elevated in patients with reduced GFR and were associated with risk factors of cardiovascular disease (CVD) in patients with CKD.

**Materials and methods:** Plasma samples were collected from a cohort of patients with CKD stage 1 to 5, aged 30-75 years (N=409), recruited from the nephrology outpatient clinic and healthy controls from the Danish Voluntary Blood Donor Corps, Rigshospitalet (N=71). Plasma apoM was measured by ELISA and S1P by HPLC.

**Results:** Plasma apoM was reduced in patients with CKD as compared to healthy controls (0.87+/-0.02 vs. 0.97+/-0.03 mol/l, p<0.01). Likewise, S1P was reduced in patients with CKD compared to controls (0.89+/-0.03 vs. 1.02+/-0.04 umol/l, p<0.05). Plasma apoM concentrations correlated to eGFR (r=0.152, p<0.01) in CKD patients. However, apoM also correlated to HDL-C (r=0.577, p<0.001) and LDL-C (r=0.505, p<0.001). CKD patients with DMT2 displayed even further reduction in plasma apoM levels than CKD patients without DMT2 (0.69+/-0.03 vs. 0.93+/-0.02 umol/l, p<0.001). Finally, CKD patients with CVD had lower plasma apoM concentration than CKD patients without CVD (0.83+/-0.03 vs. 0.91+/-0.02 umol/l, p<0.01).
**Conclusion:** Patients with CKD have reduced plasma apoM and S1P concentrations. Both CVD and DMT2 add further to reduction of plasma apoM in patients with CKD. Whether the apoM/S1P axis plays a role in human uremic atherogenesis or will be useful as a marker of accelerated CVD in CKD warrants further studies.

**No evidence for long term effect on mortality, diabetes or infections of mannose-binding lectin genotypes (MBL2) among uremic patients**

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**Background:** Patients with end stage renal disease (ESRD) have a high morbidity and mortality rate with cardiovascular diseases and infections being the major causes of death. Mannose-binding lectin (MBL) has been suggested to play a beneficial role. The aim of this study was to investigate a possible clinical association of MBL genotypes (MBL2) with outcome among patients in dialysis or with a functioning graft.

**Methods:** 98 patients with ESRD accepted for living donor renal transplantation or on the waiting list for transplantation were included and prospectively followed for nine years in average (range: 7.5-9.9). Medical records were evaluated regarding transplantation status, diabetes, vascular parameters and infections for all the patients. Cox regression models were used for statistical analyses.

**Results:** The cohort was divided into two groups according to the MBL2 genotype (normal: A/A versus variant A/O and O/O). The MBL2-genotype was not associated with all-cause mortality, cardiovascular events or frequency of bacterial infections (pneumonia, urinary tract infection, fistula infection or other infection).

**Conclusions:** In this cohort the MBL2 genotype did not seem to be associated with any long term clinical effects in patients in dialysis or with a functioning graft.
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**Diagnostics and measurements**

**Treatment of hyperptension using telemedical home blood pressure measurements**

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**Background and aim:** Telemonitoring of home blood pressure measurements (TBPM) is a new and promising supplement to diagnosis, control and treatment of hypertension. We wanted to compare the outcome of antihypertensive treatment based on TBPM and conventional monitoring of blood pressure.

**Methods:** Participants (n=356) were recruited from a prevalence study among citizens aged 55-64 years in the municipality of Holstebro, Denmark. The study was a randomised, controlled, unblinded 3 months trial. In the intervention group, antihypertensive treatment was based on TBPM with transmission of the measurements and subsequent communication by telephone or e-mail. In the control group, patients received usual care. Primary outcome was reduction in daytime ambulatory blood pressure measurements (ABPM) from baseline to 3 months’ follow-up.

**Results:** In both groups, daytime ABPM decreased significantly. The decrease in daytime ABPM in the intervention group was systolic/diastolic, -8 ±12/-4±7 mmHg. This did not differ significantly from the control group’s -8±13/-4±8 mmHg. An equal number of participants obtained normal daytime ABPM, in the intervention group 17% (31/175) versus control 21% (37/181), p=0.34.

**Conclusion:** No further reduction in ABPM or number of patients reaching blood pressure targets was observed when electronic transmission of TBPM was applied in the treatment of hypertension by GPs. Thus, as an isolated tool TBPM did not improve BP control during a 3-month period.
**Ultrasound Vector Flow Imaging for Surveillance of Arteriovenous Fistulas**

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**Background:** The arteriovenous fistula (AVF) is the preferred vascular access for hemodialysis in end-stage renal disease. However, approximately 60 percent will develop stenosis on average 18 months after surgical creation. Stenosis may lead to thrombosis and access failure. The National Kidney Foundation Kidney Disease Outcomes Quality Initiative (NKF KDOQI) recommends that patients with AVF should be monitored with regular volume-flow measurements for evaluation of stenosis and treated with percutaneous transluminal angioplasty or surgery if hemodynamically relevant. Ultrasound dilution technique (UDT) is considered the reference method for volume-flow estimation of the AVF. The ultrasound vector-flow imaging technique (VFI) can measure volume flow with a superior reproducibility than UDT. The purpose of this study was to investigate if ultrasound vector-flow imaging (VFI) is equal to the reference method ultrasound dilution technique (UDT) in estimating volume flow and changes over time in arteriovenous fistulas (AVF) for hemodialysis.

**Methods:** From January 2014 to January 2015, patients with end-stage renal disease and matured functional AVF were consecutively solicited to participate in this prospective study. VFI and UDT measurements were performed monthly over a six-month period. Nineteen patients were included in the study. VFI measurements were performed before dialysis, and UDT measurements after. Statistical analyses were performed with Bland Altman plot, student t-test, four-quadrant plot, and regression analysis. Repeated measurements and precision analysis were used for reproducibility determination.

**Results:** Precision measurements for UDT and VFI were 32 % and 20 %, respectively (p = 0.33). Average volume flow measured with UDT and VFI were 1161 ml/min (±778 ml/min) and 1213 ml/min (±980 ml/min), respectively (p = 0.3). The mean difference was -51 ml/min (CI: -150 ml/min to 46 ml/min) with limits of agreement from -35 % to 54 %, with a strong correlation (r² = 0.87). A large change in volume flow between dialysis sessions detected by UDT was confirmed by VFI (p = 0.0001), but the concordance rate was poor (0.72).

**Conclusion:** VFI is compared to UDT an acceptable method for evaluation of volume flow and volume flow changes over time in AVFs.
Can identification and standard of care of patients with AKI in emergency medical units be improved through an interactive smart phone app?

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Studies of unselected emergency admissions have found the incidence of AKI (Acute Kidney Injury) to be between 10-25% and these patients are often identified at late stages. The care they receive is in many cases sub-standard despite the existence of internationally approved guidelines for the identification and treatment of AKI. Danish national guidelines are underway.

Traditionally, guidelines have been distributed physically to relevant clinicians, and more recently also digitally on the web pages of medical societies. A few examples of attempts to heighten the awareness and use of national guidelines through the development of applications for smart phones have already been seen in some Danish medical societies and are generally considered to be successful.

This project attempts to improve the outcome and general focus on patients with - or at risk of - AKI by developing a smart phone application containing not only national guidelines on identification and treatment of AKI but also featuring an innovative algorithm. The user will be intuitively led through the guidelines and be notified on findings of importance to the specific patient case. This will bring the specialized knowledge of nephrologists closer to junior doctors in emergency medical units, thus insuring that decisions such as the use of contrast in the elderly or monitoring of the septic patient are made with the risk of AKI in mind.
Figure: *Left:* Intended flowchart of application. Based on the specified $S$-creatinine and urine output, according to KDIGO guidelines, the user will intuitively be led either to the steps for treating and impairing the succession of AKI or to an evaluation of the patients’ risk of developing AKI. *Right:* Screenshots of visual presentation.

A visual presentation of the functions of the application will be available for display during the presentation at the conference.
Oral Magnesium Supplementation Improves Serum Calcification Propensity in Chronic Kidney Disease Stage 3-4

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Background: In previous experimental studies of chronic kidney disease (CKD) magnesium has been shown to improve vascular calcification. Serum calcification propensity measured using the T50 analysis has been shown to predict all-cause mortality among patients with CKD stage 3-4.

Methods: In a randomised placebo-controlled double-blinded trial of placebo versus slow-release magnesium supplementation at two different doses (360 mg once daily or 360 mg twice daily) for eight weeks in 34 subjects with CKD stage 3-4 and plasma magnesium <0.82 mmol/L, T50 was measured to examine whether magnesium supplementation affects serum calcification propensity.

Results: In subjects randomized to slow-release magnesium hydroxide 360 mg twice daily (n = 11) plasma magnesium increased by 0.11 mmol/L (95% confidence interval; 0.03 to 0.19, p = 0.003) and T50 increased by 40 minutes (95% confidence interval; 11 to 70, p = 0.003) after eight weeks of treatment, while there were no changes in plasma phosphate, ionised calcium, albumin, bicarbonate or fetuin-A. There were no significant changes in T50 in the placebo (n = 12) or magnesium hydroxide 360 mg once daily (n = 11) groups.

Conclusions: Oral supplementation with slow-release magnesium hydroxide 360 mg twice daily improves serum calcification propensity in CKD stage 3-4 after eight weeks of treatment. Larger, long-term trials are needed to assess whether this translates into reductions in vascular calcification and cardiovascular events.
Sexual dysfunction and hyperprolactinemia in uremic men.
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Background: Both Sexual Dysfunction (SD) and hyperprolactinemia is highly prevalent in uremic men. The aim of this study was to quantify the extent and the degree of severity of both SD and hyperprolactinemia, investigate whether there is a difference between non-dialysis CKD patients (ND-CKD P) and dialysis patients (DP) in those manners, and investigate whether there is an association between SD and hyperprolactinemia.

Methods: We enrolled men between 20-70 years of age; with a creatinine clearance <= 40 ml/min or time of dialysis > 3 months. Blood samples were withdrawn and the patients answered a questionnaire including a Danish version of The International Index of erectile function (IIEF). SD was assessed as erectile dysfunction (ED), nocturnal erections, libido and self-reported problems.

Results: In total 77 patients, 47 ND-CKD P and 30 DP were enrolled in the study. Eighty-seven percent had ED, 80% of ND-CKD P and 100% of DP (p=0,04). Thirty-two percent had nocturnal erection, 36% of ND-CKD P and 27% of DP (p=0,41). None had normal libido. Sixty-one percent had severely decreased libido, 56% ND-CKD P and 70% DP (p=0,2). Seventy-one percent of all patients reported that they had a sexual problem; 62% of ND-CKD P and 87% of DP (p=0,02). The prevalence of hyperprolactinemia was 21%, 13% of ND-CKD P and 33% of DP (p=0,03). We found a significant association between libido and S-Prolactin, as well as ED and S-Prolactin in the total study population.

Conclusion: The prevalence of SD and hyperprolactinemia is high in uremic men, especially amongst patients receiving dialysis therapy. We showed a significant association between a low degree of libido and S-Prolactin, as well as ED and S-Prolactin.