Renal Nutrition News

The 6th Karolinska Institutet Advanced Renal Nutrition Conference was held April 3-5, 2014, in Stockholm, Sweden, and was organized by Peter Stenvinkel, MD, PhD, and Juan Jesús Carrero, PhD Pharm, PhD Med, of the Division of Renal Medicine at the Karolinska Institutet. One hundred delegates from 14 countries attended the conference.

The overall learning objectives this year were:

- To understand the causes and metabolic changes contributing to protein-energy wasting (PEW) in kidney disease.
- To evaluate screening measures of nutritional status and to develop the practical skills to implement and critically evaluate nutritional assessment methods and research published in professional journals.
- To review current renal-specific nutritional practices, nutritional guidelines and treatment options for the range of chronic kidney disease (CKD) patients in a variety of health care settings worldwide.

The following main topics were included in the conference:

- Causes and Consequences of PEW
- Nutritional Strategies for CKD
- Nutritional Screening and Assessment
- Nutritional Therapy in Action
- Debate on the use of PEW-preventive nutrition support in all CKD patients
- Case Studies
- Vitamin D Supplementation in CKD
- Global Research Updates
- Acidosis and Nutritional Status in CKD

Karolinska Institutet

Highlights from the 6th Karolinska Institutet Advanced Renal Nutrition Conference

Protein-energy wasting (PEW) affects 12%-18% of the patients with stage 3 and 4 CKD; approximately 2/3 of dialysis patients; and 20%-30% of kidney transplant patients. (p 2)

PEW has been shown to have a negative effect on quality of life, mortality, risk for cardiovascular disease, and infections. (p 2)

Inflammation, catabolism, and anorexia are key factors in the development of protein-energy wasting (PEW). (p 2)

Nutrition screening and assessment: important tools for developing nutrition-focused intervention for the prevention and treatment of PEW. (p 4)

Nutrition therapy, including renal oral nutritional supplements (ONS), plays a role in CKD management in dialyzed patients by helping to improve quality of life, nutrition status, and body composition. (p 7)

A large study of dialysis patients found the prevalence of frailty to be 73%, which was shown to have a negative impact on survival. (p 7)

Global research updates: New study results show that renal ONS increase serum albumin levels, muscle mass, and lean tissue mass, and improve nutritional status. (p 8)

Global research updates: Educating renal nurses and patients on the importance of ONS compliance resulted in an improvement from 25%-50% to > 80%. (p 9)

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Causes and Consequences of PEW

Epidemiology, growth, causes and consequences of PEW

Protein-energy wasting in acute and chronic kidney disease is defined as the loss of fat and lean body mass due to catabolism in the presence of nutritional and metabolic disorders. The International Society of Renal Nutrition and Metabolism (ISRNM) has identified four criteria used for the clinical diagnosis of PEW.[1] At least one measure from three out of the four criteria must be met to establish a diagnosis of PEW.

Juan Jesús Carrero, PhD Pharm, PhD Med, (Stockholm, Sweden), discussed how the prevalence of PEW varies depending on the criteria used to define it, and that it varies by country, with little data available from developing countries. Few studies have been done in early-stage CKD patients. Results of 2 studies show that PEW affects about 12%-18% of patients with stage 3 and 4 CKD; both studies used the subjective global assessment (SGA) to assess nutritional status and PEW.[2,3] Depending on the method used, PEW was identified in patients on hemodialysis (HD) and peritoneal dialysis (PD) in 17%-65% using SGA[4,5] and in 46%-60% using malnutrition inflammation score (MIS).[6,7] PEW appears to be prevalent in similar fashion in both HD (28%-54%)[8] and PD patients (28%-81%). The prevalence of PEW in renal transplant patients is around 20%-30%. PEW has been shown to have a negative effect on quality of life,[6] mortality,[6] risk for cardiovascular disease,[9,10] and infections.[9,10]

Inflammation, catabolism, and anorexia lead to PEW

Inflammation can act directly or indirectly as a cause of increased catabolism leading to PEW. **Peter Stenvinkel, MD, PhD (Stockholm, Sweden)** described six physiological pathways of inflammation that lead to increased catabolism, malnutrition, and the development of PEW. They are: (1) anorexia, (2) depression, (3) increased energy expenditure, (4) direct catabolic effects (e.g., IL-6), (5) indirect catabolic effects (e.g., insulin, myostatin), and (6) suppression and/or resistance to anabolic hormones (e.g., insulin-like growth factor, growth hormone).

ISRNM criteria for diagnosing PEW[1]

Category	Test
Serum chemistry	• Serum albumin < 3.8 g/100mL (Bromcresol Green) • Serum prealbumin (transthyretin) < 30 mg/100 mL for maintenance dialysis patients only; levels may vary according to GFR level for non-dialyzed CKD patients Stages 2-5 • Serum cholesterol < 100 mg/100 mL
Body mass	• BMI < 23 kg/m^2 • Unintentional weight loss over time; 5% loss over 3 months or 10% loss over 6 months • Total body fat percentage < 10%
Muscle mass	• Muscle wasting with mass reduced by 5% over 3 months or 10% over 6 months • Reduced mid-arm muscle circumference area > 10% reduced in relation to 50th percentile of reference population • Creatinine appearance
Dietary intake	• Unintentional low dietary protein intake (DPI) (< 0.80 g/kg/day for at least 2 months for dialysis patients, or <0.6 g/kg/day for non-dialyzed CKD patients Stages 2–5) • Unintentional low dietary energy intake (DEI) <25 kcal/kg/day for at least 2 months

Undernutrition in PEW

Anorexia, occurring in 40%-50% of dialysis patients,[12,13] has been show to decrease survival,[13-15] increase hospitalization rates,[12,13] and contribute to poor quality of life.[15] Anorexia may be a sign of uremic toxicity. Reduced food intake was noted in rats injected with uremic plasma versus control.[16] What is in uremic plasma that causes anorexia? Des-acyl ghrelin, a hormone that decreases food intake and delays gastric emptying, has been found to be elevated in end-stage renal disease (ESRD) patients.[17] Appetite improved in malnourished dialysis patients who were given acyl-ghrelin.[18]

Anorexia may also be due to low serum branched chain amino acid (BCAA) levels.[19] Oral food intake increased over 6-months in 28 patients on peritoneal dialsysis with anorexia and low albumin who were given oral BCAA.[20]

Other causes of anorexia include:

- inflammation[14]
- oral problems, such as taste abnormalities or swallowing problems
- dental problems
- impaired olfactory function
- gastrointestinal (GI) problems, including anatomic GI disorders such as gastritis, and functional GI disorders such as malabsorption and delayed gastric emptying[21] and dyspepsia[22]

Dr. Carrero provided his thoughts on how to treat anorexia. He stated we should assess for appetite, identify and treat the underlying cause of anorexia, support and promote food intake and physical activity, optimize dialysis dose, and consider appetite stimulants such as megestrol acetate.

Pathophysiology of PEW: Reduced anabolic drive

Alp Ikizler, MD (Nashville, Tennesse, USA) discussed protein balance and the catabolic effect of hemodialysis. Protein balance is achieved when protein synthesis and breakdown are balanced. Growth occurs when synthesis is greater than breakdown, whereas, wasting occurs when breakdown is greater than synthesis.

Higher negative nitrogen balance has been noted on dialysis days compared to non-dialysis days.[23] This observation suggests that hemodialysis induces catabolism. A significant amount of amino acids (about 7 g per dialysis session) have been shown to be lost into the dialysate.[24]

Hemodialysis causes net whole-body and net skeletal protein loss.[25] To compensate for this catabolism, providing intradialytic oral nutrition with amino acids, dextrose, and lipids can help promote net protein anabolism.[26]

Common hormonal and metabolic abnormalities can cause PEW. These include resistance to growth hormone and insulin-like growth factor-1, metabolic acidosis, and insulin resistance or deficiency, which worsens as kidney function declines.[27] Providing bicarbonate to correct acid-base balance has been shown to improve nutritional status.[28]

Patients with diabetes and CKD seem to lose more muscle mass than patients without diabetes.[29]

Nutritional Strategies for CKD

Nutritional requirements for ESRD dialyzed CKD patients

Dietary intake, especially protein and energy, is one of the four criteria used to diagnose risk of PEW. Protein intake should be assessed every month and energy intake every 3-6 months.[30] **Denis Fouque, MD, PhD (Lyon, France)**, conducted an observational cohort study in 3,000 maintenance hemodialysis patients and found the optimal protein intake to be 1.1 g/kg/day.[31]

Interventions to improve nutritional status include oral nutritional supplements (ONS) and intradialytic parenteral nutrition. As proposed by the ISRNM algorithm for nutrition support in patients with CKD, CKD-specific oral nutritional supplements at home or during dialysis can help patients achieve target dietary protein intake (DPI) of > 0.8 g/kg/day for patients with CKD stage 3-4 and DPI of 1.2 g/kg/day for patients with CKD stage 5.[32]

Renal specific ONS are preferred because they provide an optimized nutrient profile.

Lacson and colleagues reported up to a 34% risk reduction in 1-year mortality in hypoalbuminemic (albumin < 3.5 mg/dL) patients on hemodialysis who received intradialytic ONS compared to patients in the control group.[33] In a meta-analysis that included 429 patients on maintenance hemodialysis, enteral multinutrient support (oral or tube) was found to increase serum albumin and improve dietary intake versus routine care.[34]

Dietary modifications to modulate metabolic response in CKD patients

Risk of death and rate of cardiovascular events have been shown to increase as GFR declines.[35]

Glycemic load of the diet can also contribute to oxidative stress in maintenance hemodialysis patients.[36] Anti-inflammatory therapy, such as anti-cytokine (IL-1 beta inhibition) and omega-3 fatty acids and antioxidants, can help modulate inflammation that drives oxidative stress and insulin resistance that contribute to cardiovascular disease risk.

Phosphorus and nutrition in CKD

Dietary protein and phosphorus are tightly linked so that a change in one will result in a parallel change in the other. Non-dialyzed uremic patients who consumed a low protein (0.3 g/kg/day), low phosphorus diet for 3 months were noted to have a reduction in serum phosphorus.[37] However, restricting protein to control phosphorus may outweigh the benefit as seen with an increase in mortality.[38] The source of dietary protein also plays a role in its absorption. Phosphorus is present in plant foods in the form of phytic acid, which is less absorbed in humans due to deficient phytase. Plasma phosphorus was found to be higher from a meat diet versus a vegetarian diet.[39] Inorganic phosphorus is a food additive in processed foods and drinks and can provide a significant amount of phosphorus in the diet.[40]

Optimizing dietary fat for heart health

Data from the United States Renal Data System show that the percent of overweight and obese dialysis patients (based on BMI) is greater than those who are underweight or malnourished. Body mass index (BMI) has been shown to be a deteminant of insulin resistance[41] and oxidative stress[42] in patients with stage 3-4 CKD. Patients with CKD appear to consume too much fat[43] and the type of fat consumed (high intake of saturated fat) does not meet the American Heart Association recommendations for saturated fat < 10% of dietary energy.[44,45] Diets high in saturated fats have been associated with inflammation, hypertriglyceridemia, and increased mortality risk.[46] In contrast, fish intake has been associated with increased survival in dialysis patients.[47] Evidence from randomized, controlled trials support the intake of omega-3 fatty acids to improve blood lipids, reduce inflammation,[48] and improve graft patency[49] in people with CKD. Omega-3 fatty acids have also shown promise in delaying the progression of CKD.[48]

Nutritional Screening and Assessment

Tools and techniques

Nutrition screening helps identify patients who may be at nutritional risk who can benefit from a more indepth nutritional assessment and intervention. Screening should be quick, easy to use, reliable, valid, specific to the patient population and setting, and easily performed by non-nutrition professionals.

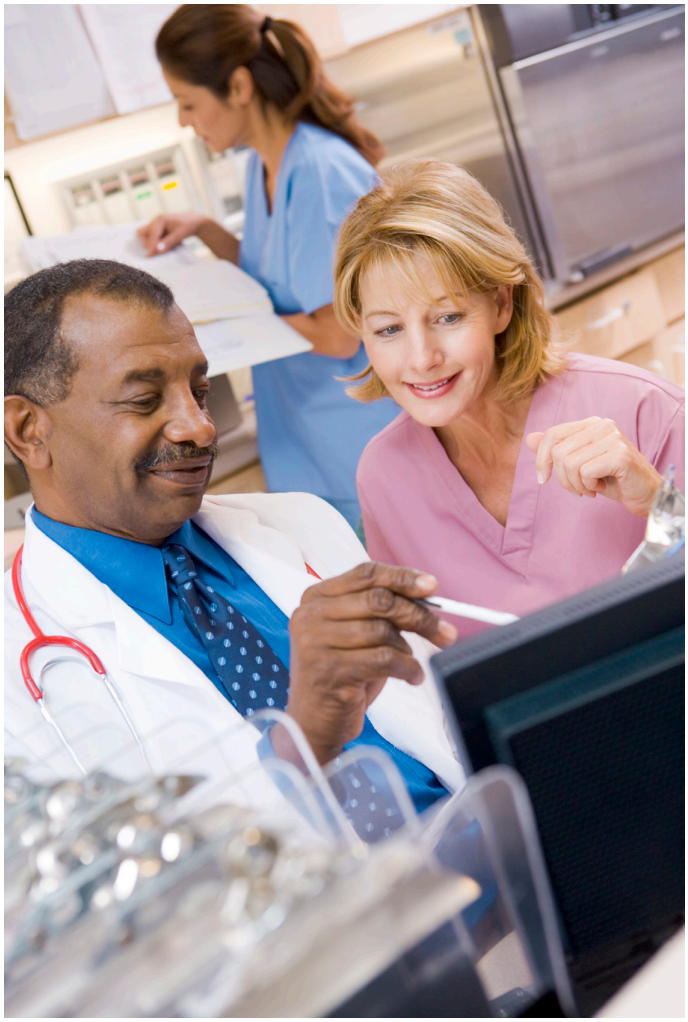
A nutrition assessment follows nutrition screening and provides a more detailed evaluation that leads to an intervention. **Maria de los Angeles Espinosa Cuevas, PhD (Mexico City, Mexico)** discussed the following assessment tools that are specific for use with patients with renal disease:

- Malnutrition inflammation score (MIS)
- Subjective global assessment (SGA) modified
- Dialysis malnutrition score (DMS)
- Composite nutritional indexes
- Objective score of nutrition on dialysis (OSND)

Of the 3 scoring systems: SGA, DMS, and MIS, MIS has a stronger association with relative risk for death and first hospitalization.[52]

The K/DOQI guidelines recommend using SGA.[50] The DMS was developed in 1999 by Dr. Kalantar and adds co-morbidities to the SGA.[51] In 2001, Dr. Kalantar developed the MIS, which includes the 7 components of the SGA (DMS) plus 3 new components: BMI, serum albumin, and serum transferrin.[52] Of the 3 scoring systems (SGA, DMS, and MIS), MIS has a stronger association with relative risk for death and first hospitalization.[52]

The OSND was created to include objective assessments including dry weight over the last 3-6 months, anthropometric measures, and biochemical measurements.[53] The seven components of the OSND are stratified as normal, moderate, or low.



Integrating screening and assessment into your practice: Institutional challenges and strategies

When a renal dietitian is not available, nurses and physicians can include simple screening tools and questions related to appetite when they conduct a medical history. Findings of the nutritional screening determine the need to conduct a nutritional assessment using either SGA or MIS. Nutritional screening and assessment are also part of the algorithm published by ISRNM for the prevention and treatment of PEW.[32]

Nutritional Therapy in Action

Diet and progression of CKD

Dr. Carrero discussed how dietary recommendations to delay CKD progression have focused on the following restrictions.

Protein Protein quantity as well as quality (meat vs vegetable sources) can have an impact on eGFR[54,55] and phosphorus levels.[39] Vegetarian diets have been shown to decrease production of uremic toxins.[56]

Heart-healthy fats As discussed earlier, the type of fat has been shown to affect renal function. In the Nurses Health Study involving 3350 women, after 10 years, saturated fat intake was associated with a higher risk of microalbuminuria and rapid eGFR decline.[57]

Carbohydrates Recent studies have reported conflicting results regarding the effect of sugar-sweetened or artificially- sweetened beverages on CKD progression and kidney function.[58,59] As such, a recommendation cannot be made regarding sugar restriction; however, increased sugar consumption can lead to obesity and an increased risk for diabetes, both risk factors for the development of CKD.

Fiber Fiber provides many benefits in the diet. However, many fruits and vegetables that are sources of fiber are restricted due to potassium content. In a large (14,543 participants) National Health and Nutrition Examination Survey (NHANES) III trial, a high fiber diet was associated with a decrease in inflammation and all-cause mortality in patients with CKD.[60]

Sodium Dietary sodium intake has been associated with albuminuria[61] and faster eGFR decline.[57] Although evidence currently does not suggest that sodium restriction delays CKD progression, strong evidence exists regarding the effect of sodium restriction on controlling blood pressure,[62] a known risk factor for CVD and CKD.

Role of the low protein diet in CKD management

A reduction in protein intake has been shown to reduce proteinuria.[63] The optimal protein intake for non-dialysis patients with CKD is 0.6-0.75 g/kg/day.[64] A low protein diet has been shown to improve dyslipidemia possibly as a result of less saturated fat intake associated with a reduction in meat intake.[65] Dr. Fouque noted that 1 out of 3 patients do not accept a low protein diet. Compliance can be assessed by measuring urinary urea.[66] A reduction in protein intake can also cause a reduction in energy intake if the diet is not carefully planned. The optimal protein intake for non-dialysis patients with CKD is 0.6-0.75 g/kg/day.

A reduction in protein intake can also cause a reduction in energy intake if the diet is not carefully planned.

Muscle dysfunction, frailty and the role of exercise therapy in CKD

The prevalence of frailty was observed in 73% of 1576 patients enrolled in the Comprehensive Dialysis Study.[67] In this study, frailty was shown to have a negative impact on survival. Physical activity (sedentary vs non-sedentary) was also shown to affect survival in patients on dialysis.[68] Low serum creatinine, a marker of muscle mass, can be used as a predictor of mortality in chronic hemodialysis patients.[69]

In healthy individuals, resistance exercise increases muscle protein synthesis. Patients on maintenance hemodialysis who exercised experienced an increase in fat-free mass at the end of exercise training. Dr Ikizler described a study that combined exercise with intradialytic parenteral nutrition. The additive effect of exercise and nutrition was demonstrated by an increase in forearm net muscle protein accretion.[70]

Diabetes, diabetic nephropathy and CKD: Nutritional challenges

Patrizio Tatti, MD (Rome, Italy) discussed nutritional challenges for people with diabetes and CKD. About 40% of people with type 2 diabetes show signs of CKD.[71] Chronic kidney disease is a major predictor of mortality in people with type 2 diabetes.[72] Dr. Tatti described the following potential risk factors for the development of diabetic nephropathy (DN):

- Sex (more common in males)
- Genetic factors
- Hypertension
- Blood glucose control
- Smoking

Diabetic nephropathy is diagnosed based on the presence of microalbuminuria, ultrasound, presence of retinopathy, and histology; however, the prevalence of DN is higher in patients with type 1 diabetes.

The kidney plays a role in removing exogenous insulin from the circulation. In patients with CKD, exogenous insulin dosage may need to be reduced to prevent hypoglycemia due to reduced exogenous insulin catabolism. As GFR declines, insulin clearance decreases.[73]

What about protein in the diet? A meta-analysis evaluated the effect of a low-protein diet on kidney function in diabetic nephropathy. The low-protein diet was significantly associated with improvements in diabetic nephropathy in terms of GFR and proteinuria.[74] Dr. Tatti discussed the potential causes of protein malnutrition in diabetes to be an increase in catabolism, microalbuminuria, reduced protein intake, and reduced anabolism.

Dr. Ikizler's 10 reasons to use renal-specific ONS in non-dialyzed CKD patients

1. **Prevent** development of PEW
2. **Prevent** frailty
3. **Prevent** loss of kidney function
4. **Improve** quality of life
5. **Prevent** obesity
6. **Improve** insulin sensitivity
7. **Prevent** systemic inflammation
8. **Prevent** oxidative stress burden
9. **Prevent** excessive health care costs
10. Lack of serious objective reasons for not recommending

Debate: Should all CKD patients have PEW-preventative nutritional support?

Drs. Ikizler and Fouque engaged in a debate about the use of nutritional support to prevent PEW for all patients with CKD. Dr. Ikizler presented the pro side of the debate stating 10 compelling reasons why one should use renal-specific ONS in non-dialyzed CKD patients. Seven of the 10 reasons he cites relate to prevention, while two relate to improvement. A number of studies have demonstrated positive nutritional and clinical benefits of using ONS for managing PEW in dialysis patients.[26,33,75-77] Additionally, ONS has been shown to reduce hospital length of stay, which leads to reduced hospital costs.[78]

Studies have shown nutritional and clinical benefits of ONS for dialysis patients[32]

- Increased calorie and protein intake
- Improved serum albumin, serum prealbumin
- Improved nutritional status (SGA)
- Increased body weight or BMI
- Increased bone mass and bone density
- Improved hand grip strength
- Improved quality of life and mental health scores

Professor Fouque presented the con side of the debate, suggesting protein intake should be reduced and energy intake increased because the problem is lack of energy (calories) not protein intake. He stated that CKD is associated with anorexia and it has been found that energy and protein intake declines as CKD progresses.[79] In a study of predialysis patients, serum albumin increased after 6 months of dietary counseling by an RD.[80] Professor Fouque concluded by stating that oral nutritional supplements are not needed as long as energy intake is increased and protein intake is optimal (0.6-0.7 g/kg/day).

Nutritional challenges with peritoneal dialysis

Nikolina Bašić-Jukić, MD, PhD (Zagreb, Croatia) gave an overview of the nutritional and clinical challenges with peritoneal dialysis (PD). She cited advantages to PD as greater flexibility, more liberal diet, no need for vascular access, and better maintenance of residual renal function. However, the disadvantages include daily use (no free days), cosmetic effect of the PD catheter and potential for abdominal distention, possible limted physical activity, and the need to store dialysate fluid. Based on data from 2008, of the 1.77 million people world-wide on dialysis, only 10.7% are on PD.[81] She stated that in Croatia, only 4% of the dialysis population is on PD.

Weight gain, diabetes, malnutrition, and PEW are nutritional challenges that can develop in patients on PD. In an observation study of 100 patients on PD, weight increased over 17 months possibly due to glucose absorption from the dialysate and improved dietary intake. However, after 17 months, patients began to lose weight.[82] Possible reasons for this weight loss include a loss of appetite, a decline in residual renal function, or healthier patients moving on to transplantation, leaving weaker patients on continuous ambulatory peritioneal dialysis (CAPD).

Patients on PD also lose protein (approximately 10 g/day and 3-4 g amino acids/day), which is higher than seen in HD.[83] Patients with moderate peritonitis can lose as much as 15 g of protein/day and up to 100 g of protein/day with severe peritonitis.[84] Evidence-based guidelines recommend 1.0-1.2 g/kg/day of protein for adults undergoing maintenance PD, in addition to adequate energy intake.[85] People who are not able to meet their protein and energy needs orally or enterally can be given intraperitoneal parenteral nutrition (IPPN) to provide amino acids. An improvement in nitrogen balance and nutritional status was noted in 11 studies of intraperitoneal AA infusions.[86]

People on PD are also prone to developing PEW due to nutrient losses in dialysis, loss of appetite due to glucose absorption from dialysis, peritonitis, and abdominal discomfort from dialysate.[87]

Vitamin D Supplementation in CKD

Vitamin D deficiency is common in patients with CKD. Vitamin D therapy has been shown to reduce cardiovascular morbidity and mortality in patients with CKD.[88,89] **Muhammad Magdi Yaqoob, MD, PhD, FRCP (London, UK)** reviewed two randomized controlled audit studies of two different clinical practices regarding vitamin D supplementation in haemodialysis patients. In the 1st study, 50,000 units of oral ergocalciferol was given once daily as a loading dose for 4 weeks followed by once monthly for 5 months as maintenance therapy. The primary outcome was change in weekly equivalent erythropoeitin stimulating agent dose requirement in relation to haemoglobin. After 6 months, 25-OH vitamin D levels increased significantly and EPO resistance index decreased in the patients who received supplementation compared to the controls who had a slight increase in 25-OH vitamin D levels and no change in EPO resistance index.

In a 6-month exploratory, double blind, placebo-controlled study, vitamin D supplementation was found to improve microcirculatory function.

Global Research Updates

Pratim Sengupta, MD, DM (Kolkata, India) discussed results from a study of intradialytic oral nutrition supplementation vs control in 110 patients. Interim results show a positive impact on albumin as well as significant changes in BMI, muscle mass and MIS.

A study of ONS in hospitalized patients with stage 3 and 4 kidney disease was reviewed by Dr. Espinosa from Mexico. Few studies of nutritional supplementation have been done in the predialysis patient population. Dr Espinosa conducted a 2-week randomized, controlled trial in non-dialyzed patients with an albumin < 3.1 g/dL. Patients were randomized to one can of low protein renal ONS per day or to standard diet (30 kcal/kg/day and 0.8 g protein/kg/day). Results showed an increase in albumin in both groups; however, the increase was significant in the supplement group ($P < 0.027$).

Josipa Radić, MD, PhD (Split, Croatia) attended the Karolinska conference in 2013 and afterwards was inspired to conduct a study on the effect of a high protein renal ONS on oxidative stress in hemodialysis patients. She hopes to present her results at the conference next year. She is also conducting a study to compare the effect of ONS and a standard nutrition regimen on biochemical and nutrition markers and cognitive and psychomotor function in malnourished patients on mainentance hemodialysis.

Božidar Vujičić, MD (Rijeka, Croatia) conducted a 6-month pilot study on the impact of ONS on body composition (measured by multifrequency bioimpedence analysis) in hemodialysis patients. After 6 months, he found a non-significant increase in albumin and a significant increase in lean tissue mass.

Helena Jackson, BSc, PgDip, MSc, RD (London, UK), discussed a renal-specific nutritional screening tool for use with patients in the hospital. Currently, there is no universal screening tool for hospitalized renal patients. The Malnutrition Universal Screening Tool (MUST) is not a good tool to use in renal patients because fluid shifts can affect weight and it is not sensitive enough to identify malnutrition in renal patients.[90] An in-patient nutritional assessment tool, iNUT, was created based on the MUST traffic light classification. This new tool was tested against MUST and SGA in 41 patients. The iNUT performed slightly better than MUST in identifying patients at nutritional risk and it was found to be easier to complete. Helena stated they would now like to do a larger, multi-center study.

Dragan Klaric, MD (Zadar, Croatia) presented an interesting case report about a 67-year-old female patient with ESRD on CAPD with depression and anorexia. On admission her body weight was 35 kg (ideal body weight 64 kg) and a BMI of 13.7. She was seen by a psychiatrist and described eating mostly fruits and vegetables and feeling well when hungry, but feeling heavy, slow, and tired when full. She was diagnosed with depression and an eating disorder. She was prescribed one tetra of a high protein renal ONS per day along with vitamins, physiotherapy, and sertraline. When she was released from the hospital, her weight had increased by 7 kg and her albumin was 3.7 g/L. In this patient, treating her depression helped improve her appetite and nutritional status.

Owen Kelly, PhD, RNutr (Columbus, Ohio, USA) presented a draft protocol for a PEW prevention study. The protocol includes two phases- a treatment phase and a prevention (reoccurence) phase. Patients will be screened based on 4 ISRNM criteria: serum albumin, weight loss, muscle mass (assessed by BIA or DEXA), and dietary intake. In the 1st phase, all patients will receive 1-2 servings of a renal-specific ONS every day for 3-6 months. Phase II will include a control group that will receive standard of care. Phase II will last 3-6 months and will include 50 patients per group. An optional 6-month follow-up can be conducted to assess for mortality.

Dr. Vujičić discussed patient education as an important driver of dietary compliance. At the Clinical Hospital Centre in Rijeka, Croatia, ONS are regularly prescribed according to the guidelines. A survey of 163 dialysis patients who were prescribed ONS found compliance to be 25% in the 143 hemodialysis patients and 50% in the 20 PD patients. The major reason for non-compliance in the hemodialysis patients was poor appetite followed by diarrhea and taste. Taste and diarrhea were also the major reasons for non-compliance cited by the PD patients. Based on these findings, two nurses were trained and patients were instructed to bring their supplements to the dialysis session and consume them during the last 2 hours of treatment. Compliance increased to 82% in the HD patients and 96% in the PD patients. Educating both the patients and renal nurses appears to be a key strategy in improving compliance and nutritional status.

Dr. Bašić-Jukić discussed the nutritional challenges for patients with encapsulating peritoneal sclerosis (EPS), a life-threatening complication of PD. These nutritional challenges include impaired peroral feeding and bowel motility, extreme PEW, and the need for PN. Strategies to overcome these challenges include initiating nutrition support early (at diagnosis of EPS) that may include peroral nutritional supplements and/or PN, immunosuppressive therapy, and/or surgery.

Acidosis and Nutritional Status in CKD

Professor Yaqoob discussed how acidosis contributes to malnutrition by promoting anorexia, inhibiting albumin synthesis, and increasing protein degradation. Correcting acidosis can improve nutritional status in patients with ESRD. He reviewed a study in patients with stage 4 and 5 CKD that evaluated the effect of correcting acidosis on the decline in eGFR and nutritional status. The study was a two-year, open-label, prospective randomized, controlled trial in 134 patients. Results revealed that 33% of the patients in the control group progressed to needing dialysis versus only 6% in the treatment group. Correcting acidosis (treatment group) had a positive affect on protein intake at 24 months, and decreased protein catabolic rate at 12 and 24 months, and there were increases in albumin levels and mid-arm muscle circumference at 12 and 24 months compared to the control group. Improvement in acidosis was also shown to improve quality of life scores for physical and mental health, kidney disease issues, and patient satisfaction as measured by KDQOL-SF36.

References

1. Fouque D, Kalantar-Zadeh K, Kopple J, et al. A proposed nomenclature and diagnostic criteria for protein-energy wasting in acute and chronic kidney disease. *Kidney Int.* Feb 2008;73(4):391-398.
2. Sanches FM, Avesani CM, Kamimura MA, et al. Waist circumference and visceral fat in CKD: a cross-sectional study. *Am J Kidney Dis.* Jul 2008;52(1):66-73.
3. Campbell KL, Ash S, Bauer JD. The impact of nutrition intervention on quality of life in pre-dialysis chronic kidney disease patients. *Clin Nutr.* Aug 2008;27(4):537-544.
4. Mazairac AH, de Wit GA, Penne EL, et al. Protein-energy nutritional status and kidney disease-specific quality of life in hemodialysis patients. *J Ren Nutr.* Sep 2011;21(5):376-386 e371.
5. Leinig CE, Moraes T, Ribeiro S, et al. Predictive value of malnutrition markers for mortality in peritoneal dialysis patients. *J Ren Nutr.* Mar 2011;21(2):176-183.
6. Rambod M, Bross R, Zitterkoph J, et al. Association of Malnutrition-Inflammation Score with quality of life and mortality in hemodialysis patients: a 5-year prospective cohort study. Am *J Kidney Dis.* Feb 2009;53(2):298-309.
7. Szeto CC, Kwan BC, Chow KM, Law MC, Li PK. Geriatric nutritional risk index as a screening tool for malnutrition in patients on chronic peritoneal dialysis. *J Ren Nutr.* Jan 2010;20(1):29-37.
8. de Mutsert R, Grootendorst DC, Boeschoten EW, et al. Subjective global assessment of nutritional status is strongly associated with mortality in chronic dialysis patients. *Am J Clin Nutr.* Mar 2009;89(3):787-793.
9. Lopes AA, Bragg-Gresham JL, Elder SJ, et al. Independent and joint associations of nutritional status indicators with mortality risk among chronic hemodialysis patients in the Dialysis Outcomes and Practice Patterns Study (DOPPS). *J Ren Nutr.* Jul 2010;20(4):224-234.
10. Dong J, Li Y, Xu Y, Xu R. Daily protein intake and survival in patients on peritoneal dialysis. *Nephrol Dial Transplant*. Nov 2011;26(11):3715-3721.
11. Stenvinkel P. Can treating persistent inflammation limit protein energy wasting? *Semin Dial.* Jan-Feb 2013;26(1):16-19.
12. Burrowes JD, Larive B, Chertow GM, et al. Self-reported appetite, hospitalization and death in haemodialysis patients: findings from the Hemodialysis (HEMO) Study. *Nephrol Dial Transplant.* Dec 2005;20(12):2765-2774.
13. Lopes AA, Elder SJ, Ginsberg N, et al. Lack of appetite in haemodialysis patients--associations with patient characteristics, indicators of nutritional status and outcomes in the international DOPPS. *Nephrol Dial Transplant.* Dec 2007;22(12):3538-3546.
14. Carrero JJ, Qureshi AR, Axelsson J, et al. Comparison of nutritional and inflammatory markers in dialysis patients with reduced appetite. *Am J Clin Nutr.* Mar 2007;85(3):695-701.
15. Kalantar-Zadeh K, Block G, McAllister CJ, Humphreys MH, Kopple JD. Appetite and inflammation, nutrition, anemia, and clinical outcome in hemodialysis patients. *Am J Clin Nutr.* Aug 2004;80(2):299-307.
16. Anderstam B, Mamoun AH, Sodersten P, Bergstrom J. Middle-sized molecule fractions isolated from uremic ultrafiltrate and normal urine inhibit ingestive behavior in the rat. *J Am Soc Nephrol.* Nov 1996;7(11):2453-2460.
17. Muscaritoli M, Molfino A, Chiappini MG, et al. Anorexia in hemodialysis patients: the possible role of des-acyl ghrelin. *Am J Nephrol.* 2007;27(4):360-365.
18. Ashby DR, Ford HE, Wynne KJ, et al. Sustained appetite improvement in malnourished dialysis patients by daily ghrelin treatment. *Kidney Int.* Jul 2009;76(2):199-206.
19. Bossola M, Tazza L, Luciani G. Mechanisms and treatment of anorexia in end-stage renal disease patients on hemodialysis. *J Ren Nutr.* Jan 2009;19(1):2-9.
20. Hiroshige K, Sonta T, Suda T, Kanegae K, Ohtani A. Oral supplementation of branched-chain amino acid improves nutritional status in elderly patients on chronic haemodialysis. *Nephrol Dial Transplant.* Sep 2001;16(9):1856-1862.
21. Aguilera A, Bajo MA, Espinoza M, et al. Gastrointestinal and pancreatic function in peritoneal dialysis patients: their relationship with malnutrition and peritoneal membrane abnormalities. *Am J Kidney Dis.* Oct 2003;42(4):787-796.
22. Van Vlem B, Schoonjans R, Vanholder R, et al. Delayed gastric emptying in dyspeptic chronic hemodialysis patients. *Am J Kidney Dis.* Nov 2000;36(5):962-968.
23. Borah MF, Schoenfeld PY, Gotch FA, Sargent JA, Wolfsen M, Humphreys MH. Nitrogen balance during intermittent dialysis therapy of uremia. *Kidney Int.* Nov 1978;14(5):491-500.
24. Ikizler TA, Flakoll PJ, Parker RA, Hakim RM. Amino acid and albumin losses during hemodialysis. *Kidney Int.* Sep 1994;46(3):830-837.
25. Ikizler TA, Pupim LB, Brouillette JR, et al. Hemodialysis stimulates muscle and whole body protein loss and alters substrate oxidation. *Am J Physiol Endocrinol Metab.* Jan 2002;282(1):E107-116.
26. Pupim LB, Majchrzak KM, Flakoll PJ, Ikizler TA. Intradialytic oral nutrition improves protein homeostasis in chronic hemodialysis patients with deranged nutritional status. *J Am Soc Nephrol.* Nov 2006;17(11):3149-3157.
27. Pham H, Robinson-Cohen C, Biggs ML, et al. Chronic kidney disease, insulin resistance, and incident diabetes in older adults. *Clin J Am Soc Nephrol.* Apr 2012;7(4):588-594.
28. de Brito-Ashurst I, Varagunam M, Raftery MJ, Yaqoob MM. Bicarbonate supplementation slows progression of CKD and improves nutritional status. *J Am Soc Nephrol.* Sep 2009;20(9):2075-2084.
29. Pupim LB, Heimburger O, Qureshi AR, Ikizler TA, Stenvinkel P. Accelerated lean body mass loss in incident chronic dialysis patients with diabetes mellitus. *Kidney Int.* Nov 2005;68(5):2368-2374.
30. Fouque D, Vennegoor M, ter Wee P, et al. EBPG guideline on nutrition. *Nephrol Dial Transplant.* May 2007;22 Suppl 2:ii45-87.
31. Fouque D, Pelletier S, Mafra D, Chauveau P. Nutrition and chronic kidney disease. *Kidney Int.* Aug 2011;80(4):348-357.
32. Ikizler TA, Cano NJ, Franch H, et al. Prevention and treatment of protein energy wasting in chronic kidney disease patients: a consensus statement by the International Society of Renal Nutrition and Metabolism. *Kidney Int.* Dec 2013;84(6):1096-1107.
33. Lacson E, Jr., Wang W, Zebrowski B, Wingard R, Hakim RM. Outcomes associated with intradialytic oral nutritional supplements in patients undergoing maintenance hemodialysis: a quality improvement report. *Am J Kidney Dis.* Oct 2012;60(4):591-600.
34. Stratton RJ, Bircher G, Fouque D, et al. Multinutrient oral supplements and tube feeding in maintenance dialysis: a systematic review and meta-analysis. *Am J Kidney Dis.* Sep 2005;46(3):387-405.
35. Go AS, Chertow GM, Fan D, McCulloch CE, Hsu CY. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. *N Engl J Med.* Sep 23 2004;351(13):1296-1305.
36. Limkunakul C, Sundell MB, Pouliot B, Graves AJ, Shintani A, Ikizler TA. Glycemic load is associated with oxidative stress among prevalent maintenance hemodialysis patients. *Nephrol Dial Transplant.* 2014 May;29(5):1047-53.
37. Rigalleau V, Blanchetier V, Combe C, et al. A low-protein diet improves insulin sensitivity of endogenous glucose production in predialytic uremic patients. *Am J Clin Nutr.* May 1997;65(5):1512-1516.
38. Shinaberger CS, Greenland S, Kopple JD, et al. Is controlling phosphorus by decreasing dietary protein intake beneficial or harmful in persons with chronic kidney disease? *Am J Clin Nutr.* Dec 2008;88(6):1511-1518.
39. Moe SM, Zidehsarai MP, Chambers MA, et al. Vegetarian compared with meat dietary protein source and phosphorus homeostasis in chronic kidney disease. *Clin J Am Soc Nephrol.* Feb 2011;6(2):257-264.

40. Carrigan A, Klinger A, Choquette SS, et al. Contribution of food additives to sodium and phosphorus content of diets rich in processed foods. *J Ren Nutr.* Jan 2014;24(1):13-19, 19e11.
41. Trirogoff ML, Shintani A, Himmelfarb J, Ikizler TA. Body mass index and fat mass are the primary correlates of insulin resistance in nondiabetic stage 3-4 chronic kidney disease patients. *Am J Clin Nutr.* Dec 2007;86(6):1642-1648.
42. Ramos LF, Shintani A, Ikizler TA, Himmelfarb J. Oxidative stress and inflammation are associated with adiposity in moderate to severe CKD. *J Am Soc Nephrol.* Mar 2008;19(3):593-599.
43. Therrien M, Byham-Gray L, Denmark R, Beto J. Comparison of dietary intake among women on maintenance dialysis to a Women's Health Initiative cohort: results from the NKF-CRN Second National Research Question Collaborative Study. *J Ren Nutr.* Mar 2014;24(2):72-80.
44. Khoueiry G, Waked A, Goldman M, et al. Dietary intake in hemodialysis patients does not reflect a heart healthy diet. *J Ren Nutr.* Nov 2011;21(6):438-447.
45. Bossola M, Leo A, Viola A, et al. Dietary intake of macronutrients and fiber in Mediterranean patients on chronic hemodialysis. *J Nephrol.* Sep-Oct 2013;26(5):912-918.
46. Huang X, Stenvinkel P, Qureshi AR, et al. Clinical determinants and mortality predictability of stearoyl-CoA desaturase-1 activity indices in dialysis patients. *J Intern Med.* Mar 2013;273(3):263-272.
47. Kutner NG, Clow PW, Zhang R, Aviles X. Association of fish intake and survival in a cohort of incident dialysis patients. *Am J Kidney Dis.* May 2002;39(5):1018-1024.
48. Huang X, Lindholm B, Stenvinkel P, Carrero JJ. Dietary fat modification in patients with chronic kidney disease: n-3 fatty acids and beyond. *J Nephrol.* Nov-Dec 2013;26(6):960-974.
49. Lok CE, Moist L, Hemmelgarn BR, et al. Effect of fish oil supplementation on graft patency and cardiovascular events among patients with new synthetic arteriovenous hemodialysis grafts: a randomized controlled trial. *JAMA.* May 2 2012;307(17):1809-1816.
50. Clinical practice guidelines for nutrition in chronic renal failure. K/DOQI, National Kidney Foundation. *Am J Kidney Dis.* 2000;35(Suppl 2):S1-S140.
51. Kalantar-Zadeh K, Kleiner M, Dunne E, Lee GH, Luft FC. A modified quantitative subjective global assessment of nutrition for dialysis patients. *Nephrol Dial Transplant.* Jul 1999;14(7):1732-1738.
52. Kalantar-Zadeh K, Kopple JD, Block G, Humphreys MH. A malnutrition-inflammation score is correlated with morbidity and mortality in maintenance hemodialysis patients. *Am J Kidney Dis.* Dec 2001;38(6):1251-1263.
53. Beberashvili I, Azar A, Sinuani I, et al. Objective Score of Nutrition on Dialysis (OSND) as an alternative for the malnutrition-inflammation score in assessment of nutritional risk of haemodialysis patients. *Nephrol Dial Transplant.* Aug 2010;25(8):2662-2671.
54. Woods LL. Mechanisms of renal hemodynamic regulation in response to protein feeding. *Kidney Int.* Oct 1993;44(4):659-675.
55. Kontessis P, Jones S, Dodds R, et al. Renal, metabolic and hormonal responses to ingestion of animal and vegetable proteins. *Kidney Int.* Jul 1990;38(1):136-144.
56. Patel KP, Luo FJ, Plummer NS, Hostetter TH, Meyer TW. The production of p-cresol sulfate and indoxyl sulfate in vegetarians versus omnivores. *Clin J Am Soc Nephrol.* Jun 2012;7(6):982-988.
57. Lin J, Hu FB, Curhan GC. Associations of diet with albuminuria and kidney function decline. *Clin J Am Soc Nephrol.* May 2010;5(5):836-843.
58. Bomback AS, Katz R, He K, Shoham DA, Burke GL, Klemmer PJ. Sugar-sweetened beverage consumption and the progression of chronic kidney disease in the Multi-Ethnic Study of Atherosclerosis (MESA). *Am J Clin Nutr.* Nov 2009;90(5):1172-1178.
59. Lin J, Curhan GC. Associations of sugar and artificially sweetened soda with albuminuria and kidney function decline in women. *Clin J Am Soc Nephrol.* Jan 2011;6(1):160-166.
60. Krishnamurthy VM, Wei G, Baird BC, et al. High dietary fiber intake is associated with decreased inflammation and all-cause mortality in patients with chronic kidney disease. *Kidney Int.* Feb 2012;81(3):300-306.
61. Aaron KJ, Campbell RC, Judd SE, Sanders PW, Muntner P. Association of dietary sodium and potassium intakes with albuminuria in normal-weight, overweight, and obese participants in the Reasons for Geographic and Racial Differences in Stroke (REGARDS) Study. *Am J Clin Nutr.* Oct 2011;94(4):1071-1078.
62. Sacks FM, Svetkey LP, Vollmer WM, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. *N Engl J Med.* Jan 4 2001;344(1):3-10.
63. Gansevoort RT, Heeg JE, Vriesendorp R, de Zeeuw D, de Jong PE. Antiproteinuric drugs in patients with idiopathic membranous glomerulopathy. *Nephrol Dial Transplant.* 1992;7 Suppl 1:91-96.
64. National Kidney Foundation. KDOQI Clinical Practice Guidelines and Clinical Practice Recommendations for Diabetes and Chronic Kidney Disease. *Am J Kidney Dis.* 2007;49(Suppl 2):S1-S180.
65. Bernard S, Fouque D, Laville M, Zech P. Effects of low-protein diet supplemented with ketoacids on plasma lipids in adult chronic renal failure. *Miner Electrolyte Metab.* 1996;22(1-3):143-146.
66. Fouque D, Le Bouc Y, Laville M, et al. Insulin-like growth factor-1 and its binding proteins during a low-protein diet in chronic renal failure. *J Am Soc Nephrol.* Nov 1995;6(5):1427-1433.
67. Bao Y, Dalrymple L, Chertow GM, Kaysen GA, Johansen KL. Frailty, dialysis initiation, and mortality in end-stage renal disease. *Arch Intern Med.* Jul 23 2012;172(14):1071-1077.
68. O'Hare AM, Tawney K, Bacchetti P, Johansen KL. Decreased survival among sedentary patients undergoing dialysis: results from the dialysis morbidity and mortality study wave 2. *Am J Kidney Dis.* Feb 2003;41(2):447-454.
69. Pupim LB, Caglar K, Hakim RM, Shyr Y, Ikizler TA. Uremic malnutrition is a predictor of death independent of inflammatory status. *Kidney Int.* Nov 2004;66(5):2054-2060.
70. Pupim LB, Flakoll PJ, Levenhagen DK, Ikizler TA. Exercise augments the acute anabolic effects of intradialytic parenteral nutrition in chronic hemodialysis patients. *Am J Physiol Endocrinol Metab.* Apr 2004;286(4):E589-597.
71. Koro CE, Lee BH, Bowlin SJ. Antidiabetic medication use and prevalence of chronic kidney disease among patients with type 2 diabetes mellitus in the United States. *Clin Ther.* Nov 2009;31(11):2608-2617.
72. Bruno G, Merletti F, Bargero G, et al. Estimated glomerular filtration rate, albuminuria and mortality in type 2 diabetes: the Casale Monferrato study. *Diabetologia.* May 2007;50(5):941-948.
73. Kobayashi S, Maesato K, Moriya H, Ohtake T, Ikeda T. Insulin resistance in patients with chronic kidney disease. *Am J Kidney Dis.* Feb 2005;45(2):275-280.
74. Nezu U, Kamiyama H, Kondo Y, Sakuma M, Morimoto T, Ueda S. Effect of low-protein diet on kidney function in diabetic nephropathy: meta-analysis of randomised controlled trials. *BMJ Open.* 2013;3(5).
75. Boudville N, Rangan A, Moody H. Oral nutritional supplementation increases caloric and protein intake in peritoneal dialysis patients. *Am J Kidney Dis.* Mar 2003;41(3):658-663.
76. Caglar K, Fedje L, Dimmitt R, Hakim RM, Shyr Y, Ikizler TA. Therapeutic effects of oral nutritional supplementation during hemodialysis. *Kidney Int.* Sep 2002;62(3):1054-1059.
77. Scott MK, Shah NA, Vilay AM, Thomas J, 3rd, Kraus MA, Mueller BA. Effects of peridialytic oral supplements on nutritional status and quality of life in chronic hemodialysis patients. *J Ren Nutr.* Mar 2009;19(2):145-152.
78. Cheu C, Pearson J, Dahlerus C, et al. Association between oral nutritional supplementation and clinical outcomes among patients with ESRD. *Clin J Am Soc Nephrol.* Jan 2013;8(1):100-107.
79. Kopple JD, Berg R, Houser H, Steinman TI, Teschan P. Nutritional status of patients with different levels of chronic renal insufficiency. Modification of Diet in Renal Disease (MDRD) Study Group. *Kidney Int Suppl.* Nov 1989;27:S184-194.
80. Leon JB, Majerle AD, Soinski JA, Kushner I, Ohri-Vachaspati P, Sehgal AR. Can a nutrition intervention improve albumin levels among hemodialysis patients? A pilot study. *J Ren Nutr.* Jan 2001;11(1):9-15.
81. Lameire N, Van Biesen W. Epidemiology of peritoneal dialysis: a story of believers and nonbelievers. *Nat Rev Nephrol.* Feb 2010;6(2):75-82.
82. Diaz-Buxo JA, Burgess WP. Is weight gain inevitable in most chronic peritoneal dialysis patients? *Adv Perit Dial.* 1992;8:334-339.
83. Lindholm B. Peritoneal dialysis. In: Nolph K, ed. Boston: Kluwer Academic Publishers; 1989:230-260.
84. Bergstrom J, Furst P, Alvestrand A, Lindholm B. Protein and energy intake, nitrogen balance and nitrogen losses in patients treated with continuous ambulatory peritoneal dialysis. *Kidney Int.* Nov 1993;44(5):1048-1057.
85. Naylor HL, Jackson H, Walker GH, et al. British Dietetic Association evidence-based guidelines for the protein requirements of adults undergoing maintenance haemodialysis or peritoneal dialysis. *J Hum Nutr Diet.* Aug 2013;26(4):315-328.
86. Mehrotra R, Kopple JD. Protein and energy nutrition among adult patients treated with chronic peritoneal dialysis. *Adv Ren Replace Ther.* Jul 2003;10(3):194-212.
87. Han SH, Han DS. Nutrition in patients on peritoneal dialysis. *Nat Rev Nephrol.* Mar 2012;8(3):163-175.
88. Shoben AB, Rudser KD, de Boer IH, Young B, Kestenbaum B. Association of oral calcitriol with improved survival in nondialyzed CKD. *J Am Soc Nephrol.* Aug 2008;19(8):1613-1619.
89. Ravani P, Malberti F, Tripepi G, et al. Vitamin D levels and patient outcome in chronic kidney disease. *Kidney Int.* Jan 2009;75(1):88-95.
90. Lawson CS, Campbell KL, Dimakopoulos I, Dockrell ME. Assessing the validity and reliability of the MUST and MST nutrition screening tools in renal inpatients. *J Ren Nutr.* Sep 2012;22(5):499-506.

ISRNM Nutrition Algorithm for Prevention and Treatment of PEW in CKD Patients

INTERNATIONAL SOCIETY OF RENAL NUTRITION AND METABOLISM

Periodic nutrition screening
- SAlb, weight, BMI, MIS, DPI, DEI

→ **Nutrition assessment (as indicated)**
- SPrealb; SGA; anthropometrics

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Continuous preventive measures
- Continuous nutritional counseling
- Optimize RRT-Rx and dietary nutrient intake
- Manage co-morbidities (acidosis, DM, inflammation, CHF, depression)

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Indications for nutritional interventions despite preventive measures
- Poor appetite and/or poor oral intake
- DPI <1.2 (CKD Stage 5) or <0.7 (CKD Stages 3-4); DEI <30 kcal/kg/day
- SAlb <3.8 g/dL or SPrealb <28 mg/dL
- Unintentional weight loss >5% of IBW or EDW over 3 months
- Worsening nutritional markers over time
- SGA in PEW range

↓

Start CKD-specific oral nutritional supplementation
- CKD Stages 3-4: DPI target of >0.8 g/kg/day (± AA/KA or ONS)
- CKD Stage 5D: DPI target >1.2 g/kg/day (ONS at home or during dialysis treatment; in-center meals)

↓

SAlb >3.8; SPrealb >28; Weight or LBM gain

↓

Maintenance nutrition therapy goals
- SAlb >4.0 g/dL
- SPrealb >30 mg/dL
- DPI >1.2 (CKD Stage 5D); >0.7 g/kg/day (CKD Stages 3-4)
- DEI 30-35 kcal/kg/day

No improvement or deterioration

↓

Intensified therapy
- Dialysis prescription alterations
- Increase quantity of oral therapy
- Tube feeding or PEG, if indicated
- Parenteral interventions
  - IDPN (esp. if SAlb <3.0 g/dL)
  - TPN

→ **Adjunct therapies**
- Anabolic hormones
  - Androgens, GH
- Appetite stimulants
- Anti-inflammatory interventions
  - Omega-3; IL-1ra
- Exercise (as tolerated)

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AA/KA, amino acid/keto acid; BMI, body mass index; CHF, congestive heart failure; CKD, chronic kidney disease; DEI, Dietary energy intake; DM, diabetes mellitus; DPI, Dietary protein intake; EDW, estimated dry weight; GH, growth hormone; IBW, ideal body weight; IDPN, intradialytic parenteral nutrition; IL-1ra, interleukin-1 receptor antagonist; LBM, lean body mass; MIS, Malnutrition Inflammation Score; ONS, oral nutrition supplement; PEW, protein-energy wasting; RRT-Rx, renal replacement therapy prescription; SAlb, serum albumin; SGA, Subjective Global Assessment; SPrealb, serum prealbumin; TPN, total parenteral nutrition

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