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Nutritional Management of the Chronic Kidney Disease Patient

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Introduction

An estimated nineteen million (11%) adults in the United States have chronic kidney disease (CKD) (1). Both CKD and acute renal failure (ARF) are common illnesses treated in the hospital setting. This article will summarize the nutritional goals and interventions throughout the spectrum of kidney disease including CKD Stages 1 - 5, renal transplantation, and ARF. The role of nutrition in nephrology should not be underestimated. The nutrition prescription, which must be continually altered during the progression of CKD, is an essential component in the overall treatment plan. In addition, nutritional status plays a significant role in the well-being and survival of these patients. Kidney disease, from etiology to treatment, is a very complicated illness which requires a multi discipline team approach to manage. The following provides practical and scientific reasoning for the nutritional care of these patients.

Chronic Kidney Disease: Stages 1 to 4

Nutrition goals in chronic kidney disease include decreasing the accumulation of nitrogenous wastes, prevention of malnutrition and delaying the progression of kidney disease (2). One criterion defining CKD is kidney

damage for three months or greater, characterized by structural or functional renal abnormalities, presenting with either pathological abnormalities or irregular blood, urine or imaging study results, which may or may not be associated with a decreased Glomerular Filtration Rate (GFR) (3). A second criterion of CKD is a GFR less than 60 mL/min/1.73m² that is present for three months or greater, irrespective of the presence of renal damage (3). As GFR declines, the severity of associated complications increases. The use of both low protein diets (LPD) and very low protein diets (VLPD) supplemented with essential amino acids (EAA) and/or ketoacids (KA) has been evaluated in the treatment to delay the progression of CKD.

Patients with CKD, not undergoing dialysis, may be managed with a low protein, low phosphorus diet to prevent or treat uremic symptoms and to delay the progression of the disease (4, 5, 6). Elevations in serum phosphorus and potassium noted in early stages CKD suggest that dietary modification of these nutrients may be necessary (7). Protein restrictions may be used in patients with CKD that have a) symptoms of uremia, b) edema or poorly controlled hypertension c) continued decline in kidney function despite blood pressure control and use of angiotensin converting enzyme inhibitors (2). There is evidence that a protein- sparing mechanism occurs when dietary protein intake is reduced allowing for a sustained favorable metabolic response to decreased protein intakes in patients with CKD (8).

Nutritional Adequacy of Protein Restriction

Various studies have evaluated the nutritional adequacy of LPD and VLPD in patients with mild to severe CKD, predialysis. Patients receiving a LPD

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FROM THE EDITOR'S DESK

Time and again, I hear dietitians ask, "Why are the same people always on the committees, running for office, presenting at key meetings, and receiving awards?" My answer to this question is that at some point in their careers, these dietitians chose to make a difference in our profession — so they volunteered. Probably they started small — but one thing leads to another and more people get to know you — and it spirals into more.

In most written publications, organizations beg for people to get involved through feedback forms and ads asking for volunteers. So how do you begin the process? These days it's so easy, in many cases it can begin with an e-mail expressing interest in getting involved. The key is actually taking that first step and taking accountability for the future of our profession.

You may actually be surprised at all that you get back from volunteering. You may be able to enhance your writing skills or spread your creative wings. For example, the Renal Practice Group is always looking for new people to write for the Forum or even participate in editing articles. There is definitely something for everyone — when you make the first step. So I encourage all of you to continually give us feedback, send ideas, and most of all let us know that you are willing to make a commitment get involved and what area you would like to focus on.

I would like to thank all of you who have made a significant time commitment to the forum time and again. Please join me in thanking Philipa Norton Feiertag, MEd, RD, LD, who continually provides us with clinically relevant articles to help us provide the

most up to date nutritional care for our CKD patients. In this Forum, please take a look at her article on dietary supplements, and take the time to answer the CPE questions on pages 1-2 of the insert and submit the CPE answer sheet on page 3 of the insert. Please retain the Certificate of Completion on page 21 for your records.

Another contributor, Lee Ann Smith, MPH, RD took the initial step not too long ago to let us know that she wanted to start writing for the forum. Lee Ann has now completed her second article, on nutritional care throughout CKD, which I hope you will all find helpful as we begin to focus more on early stages of CKD in order to try to delay the progression of kidney disease.

You will probably notice something different about this edition of the Forum. Kidney Friendly Food Facts that has been written by Sharon Schatz, MS, RD, CSR, CDE, for numerous years is not in this publication. We would like to thank Sharon for all of the time and effort that she continues to put into her very informative column. You will still be able to read Kidney Food Facts on the RPG website www.renalnutrition.org, and now you will be able to print out these very important educational handouts for your patients.

Please take the first step — feel free to send me an e-mail at mfeditor@yahoo.com. I look forward to hearing from you.

Sharon Griff

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(0.6 g/kg/day of protein) or a VLPD (0.3 g/kg/day of protein) supplemented with EAA and/or KA experienced no signs of severe malnutrition (9, 10). Furthermore, a reduction in serum urea nitrogen is attained with the VLPD and KA (10). A study of diet in mild CKD providing >31 kcal/kg/day and 0.7 g/kg/day of protein was considered "metabolically and nutritionally safe" (8). K/DOQI guidelines indicate a LPD allowing 0.60 g/kg/day of protein and 35 kcal/kg/day (<60 years of age) and 30-35 kcal/kg/day (≥ 60 years of age) in CKD with a GFR <25 mL/min without dialysis (3). However, modification of dietary protein in these patients is appropriate only when energy consumption is adequate (3).

Delay of Chronic Kidney Disease Progression

To evaluate the potential delay of initiation of dialysis on a VLPD, 0.3 g/kg of ideal body weight (IBW)/day and 35 kcal/kg IBW/day plus supplemented EAA and/or KA was administered (5). Renal survival on nutritional therapy was calculated as the time from when the GFR became 10 mL/min (15 mL/min in patients with diabetes) to the date of dialysis initiation. Median renal survival was 353 days and there was no significant change in albumin levels or experience of malnutrition. Mortality during the first two years of dialysis was well below the national average (5). This suggests that a very low protein diet, supplemented with EAA and/or KA is nutritionally safe and may delay the necessity of dialysis initiation. However, further study of this diet with larger numbers of patients is necessary.

Current studies indicate a favorable role for LPD in mild to severe CKD, stages 3 and 4, in delaying the progression of renal disease while maintaining nutritional status. Further research is needed regarding the amount and type of protein required and safety of length of time on the diet. Also, it is important to note that very low protein diets are supplemented with EAA or KA to maintain nutritional adequacy and prevent protein malnutrition. A metabolic response to decreased protein intakes in patients with CKD may be present, resulting in a nitrogen-sparing effect. Individualized nutrition counseling with a renal dietitian, who possesses expertise in renal nutrition principles, is imperative to achieving compliance with the predialysis nutrition prescription and insuring nutritional adequacy.

Nephrotic Syndrome

Nephrotic syndrome, characterized by high levels of urinary protein loss (≥ 3 g proteinuria/day) and urinary albumin loss (microalbuminuria), affects large numbers of patients (11). Microalbuminuria in both diabetes and hypertension predicts progression of kidney disease and cardiovascular risk (11). Both the type and amount of dietary protein consumed has an effect on urinary albumin excretion in patients with diabetes.

Although high biological value protein is usually recommended, vegetarian diets containing soy and plant-based protein may reduce urinary protein loss, improve serum protein levels, ameliorate the progression of diabetic nephropathy, and prevent obesity-related renal diseases (11). A vegetarian diet consisting of 0.7 g/kg/day of protein and 33 to 35 kcal/kg/day in the early stages of CKD and 0.3 g/kg/day of protein and 30 to 35 kcal/kg/day once creatinine clearance is <20 mL/min has been shown to delay the progression of CKD in patients with diabetic nephropathy (12). As mentioned previously, EAA and KA supplements are necessary in the very low protein diet to avoid protein malnutrition. Patients experienced decreases in urinary protein losses and fasting blood glucose levels (12).

A LPD of 0.45 to 0.80 g/kg/day of protein and 35 kcal/kg/day is considered safe for patients with nephrotic syndrome without catabolic illnesses, not receiving catabolic medications and without severe proteinuria (>15 g/day) (11). Furthermore, 0.8 g/kg/day of protein plus 1 g protein per gram urinary protein loss and 35 kcal/kg/day maintains nitrogen balance in nephrotic patients (13). It should be emphasized that adequate calories must be given to prevent the loss of lean muscle mass. A low sodium (<2 g sodium), reduced fat diet containing 0.8 to 1.0 g protein/kg/day and 35 kcal/kg/day is currently recommended for patients with nephrotic syndrome (11).

Chronic Kidney Disease: Stage 5

CKD Stage 5 is indicated by either a GFR <15 mL/min/1.73 m², which is often associated with uremia, or the necessity to initiate renal replacement therapy (dialysis or transplantation) (3). This section discusses the nutrition

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issues of patients with kidney failure undergoing dialysis. Multiple factors contribute to compromised nutritional status and protein depletion in patients with kidney failure. Protein energy malnutrition (PEM) is common among patients with kidney failure and is associated with increased morbidity and mortality, including cardiovascular-related fatality (14, 15). The renal dietitian, along with the nephrology team, takes steps to prevent malnutrition, identify potential causes of malnourishment and devise nutritional interventions to improve patient outcomes.

There are two distinct forms of malnutrition in this patient population. The first type is compared with "classic" malnutrition associated with inadequate dietary consumption, a decrease in lean body mass and usually a normal serum albumin level. The second form is related to inflammation and atherosclerosis, resulting in decreased serum albumin despite adequate dietary intake (16). Low body mass index (BMI) is associated with decreased survival in kidney failure (17). Two significant factors predisposing patients undergoing dialysis to loss of lean body mass include dialysate amino acid, protein and glucose losses and anorexia (16).

Patients undergoing hemodialysis (HD) and peritoneal dialysis (PD) may lose 2-8 g and 5-12 g of free amino acids per treatment day, respectively (16). These losses are more profound when coupled with poor appetite and dietary intake. Among the 300,000 individuals with CKD Stage 5 on dialysis, it is suggested that approximately 70% may consume less than recommended amounts of calories and protein (17,18). Causes of poor appetite among individuals with kidney failure include underdialysis, comorbid conditions, use of multiple medications, and psychosocial issues, should be considered when intervening to improve the nutritional status of patients (16).

Malnutrition-Inflammation Complex Syndrome

There is a mounting body of literature and discussion surrounding the phenomenon known as malnutrition-inflammation complex syndrome (MICS). This term is used to describe the relationship between PEM and cardiovascular disease in patients on dialysis, both common and coexisting conditions in this population, which are related through inflammation. MICS is associated with hypoalbuminemia, suboptimal appetite, hypercatabolism,

poor dialysis outcomes, and decreased quality of life (19, 20, 21, 22).

Markers of MICS include proinflammatory cytokines such as interleukin-1, interleukin-6 and tumor necrosis factor- α ; serum negative acute phase proteins albumin and prealbumin; and C-reactive protein, total iron binding capacity, creatinine, total cholesterol and normalized protein nitrogen appearance (22, 23). These are common correlates of mortality in patients undergoing hemodialysis (19).

Both calorie and protein recommendations are greater for patients with CKD Stage 5 undergoing dialysis compared to the recommendations for patients with CKD Stages 2 - 4. Patients on dialysis have significantly higher resting energy expenditure (REE) than predialysis patients (24). Potential factors contributing to increased REE in kidney failure include severe secondary hyperparathyroidism and

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Table I. Causes of Protein Catabolism in Patient with ARF (22, 32)

Uremic Toxins (ureagenesis)

↑ hepatic glucose production in animal studies

Altered Carbohydrate and Protein Metabolism

↓ protein synthesis, ↑ protein breakdown and
↑ amino acid uptake ↓ activity of adenosine triphosphate and ubiquitin-dependent proteolytic pathway

Hormone and Immune Responses

Insulin resistance

Increased secretion of catabolic hormones (catecholamine, glucagon, growth hormone, and glucocorticoids)

Secretion of proinflammatory cytokines, acute phase reaction (tumor necrosis factor, interleukin-6 and interleukin-1)

Hyperparathyroidism

Metabolic Acidosis

Inadequate Nutritional Intake

Renal Replacement Therapy

Loss of nutritional substrates

Activation of protein catabolism

the hemodialysis treatment (25, 26). While it is indicated that inflammation is correlated with an increase in REE in CKD, additional studies are needed to investigate the role of inflammation on resting energy expenditure in patients on renal replacement therapy (27).

Nutrition Recommendations for CKD Stage 5

Protein recommendations are 1.2 to 1.4 g/kg/day for hemodialysis patients and up to 1.5 g/kg/day for PD patients. It is suggested that greater than 50% of protein should be of high biological value (16). Energy requirements should be generous to allow for utilization of dietary protein in healing and tissue repair and must be individualized to account for activity level and nutritional goals. Approximately 30 to 35 kcal/kg/day is suggested on hemodialysis and 25 to 30 kcal/kg/day on PD (16). To account for substantial energy absorption in the form of glucose through dialysate (approximately 680 kcal/day) on PD, dietary energy requirements are slightly less than what is recommended on HD (16).

Renal Transplantation

Patients with functioning kidney transplants typically have fewer dietary restrictions that are common with patients with CKD Stages 2-5, however, nutrition continues to play a vital role in the health and survival of these patients. When advising renal transplantation patients and candidates, nutrition objectives include mitigating the potential side effects of immunosuppressive therapy, addressing previously existing nutrition-related conditions, and maintaining optimal function of the kidney.

The adverse effects of glucocorticoid immunosuppressive therapy on the nutritional and metabolic status of patients living with renal transplants are numerous and include protein catabolism, obesity, hyperlipidemia, and glucose intolerance (28). Insulin resistance caused by steroid therapy further complicates the care of patient with diabetes (28). In addition, a portion of patients receiving kidney transplants without diabetes will develop the illness within the first three weeks after surgery (29). These metabolic alterations and nutritional consequences must be considered when providing diet counseling to kidney transplant recipients.

Both very low and very high BMI is adversely correlated with patient and kidney transplant survival (30). This suggests

that maintaining a healthy weight is imperative for patients with a kidney transplant.

Use of high protein, low carbohydrate diets have proved successful in preventing cushinoid features and improving nitrogen balance in renal transplant patients receiving steroid therapy (31). However there is insufficient data evaluating the safety and efficacy of this type of diet in recipients of kidney transplants. When considering protein needs of these patients, equilibrium must be achieved between providing sufficient protein for wound healing, treating pre-existing protein deficiency, and improving nitrogen balance while minimizing the accumulation of waste products.

Nutrition Recommendations in Renal Transplantation

The first month after renal transplant 1.3 to 1.5 g/kg/day of protein and 30 to 35 kcal/kg/day is recommended (28). After one month post-transplant 1.0 g/kg/day of protein and adequate calories to achieve and maintain ideal weight is suggested. It is important that patients receiving kidney transplants receive comprehensive nutrition therapy not only to prepare for surgery, but to transition into a new stage of kidney disease.

Acute Renal Failure

Risk of mortality in patients with acute renal failure is correlated with nutritional and metabolic factors (32). Goals of nutrition therapy in ARF include the prevention of malnutrition and the preservation of lean body mass. An appropriate nutrition plan, which considers the presence and severity of catabolism and the type and intensity of renal replacement therapy, while addressing both the underlying illness and resulting complications, must be developed to meet the unique needs of the patient.

Often ARF is associated with sepsis, trauma and multi-organ failure resulting in a hypercatabolic state with complex hormonal and metabolic alterations such as insulin resistance, hypertriglyceridemia and hepatic gluconeogenesis (32, 33, 34, 35). A major metabolic alteration in ARF, protein catabolism, characterized by the release of amino acids and a negative nitrogen balance is a result of numerous factors present in these critically ill patients (see Table I). Therefore, nutritional requirements in ARF

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are influenced by a multitude of potential complications in addition to the underlying disease. When delivering nutrition support to these patients, meeting the minimum nutritional requirements for chronic kidney disease is inadequate.

Nutrition Recommendations and Severity of ARF

Acute renal failure may be categorized into three groups related to nutrition needs and the severity of ARF. Group I usually consists of patients that are often well nourished and able to feed orally. Often the cause of ARF is due to nephrotoxins such as therapeutic drugs, radiocontrast agents, carcinogens, metals, abused drugs, and industrial chemicals (36). These patients may be given 0.6 g/kg/day of protein initially and gradually increased to 0.8 g/kg/day with a caloric provision of 25 kcal/kg/day (32). When undergoing hemodialysis or peritoneal dialysis, this amount of protein should be increased to 1.0 to 1.2 g/kg/day and 1.4 d/kg/day, respectively to compensate for amino acid and protein losses (32).

Patients in group II may be moderately hypercatabolic and suffering from infections or injury. Enteral feedings, parenteral nutrition or both may be necessary forms of nutrition support. These patients will require approximately 0.8 to 1.2 g/kg/day of protein and 20 to 30 kcal/kg/day (32).

Group III includes patients with severe infection, major trauma and burns presenting in a severe hypercatabolic state. Patients with ARF and sepsis have a 68% rate of mortality (37). Patients in Group III should receive 1.2 to 1.5 g/kg/day of protein (or amino acids) and when undergoing renal replacement therapy, protein intake should reach 1.5 g/kg/day (32). Energy requirements are 25 to 35 kcal/kg/day.

Medical Nutrition Support in ARF

Enteral nutrition is considered the standard method of nutrition support in critically ill patients, including those with ARF. Though enteral feeding is considered safe and effective in patients with ARF, it may be impossible to utilize this route exclusively to meet nutrition needs (38, 39). It may be necessary to supplement enteral nutrition with parenteral nutrition or total parenteral nutrition (TPN) may be necessary.

One study of critically ill patients on continuous renal replacement therapy for ARF concluded that increased nitrogen balance, achieved with high doses of protein (>2 g/kg/day), improved probability of survival (40). High protein (1.5-1.8 g/kg/d) and relatively low calorie (25-35 kcal/kg/d) dietary regimens may improve nitrogen balance (41). One study of patients receiving both TPN and renal replacement therapy found that on a nitrogen intake of 0.25 g/kg/day, higher caloric provision (40 kcal/kg/day) did not improve nitrogen balance compared with a lower caloric intake (30 kcal/kg/day) (42). This suggests that nutrition support in severe cases of ARF should provide high doses of protein and sufficient calories to improve nitrogen balance, which leads to increased likelihood of survival.

Additional research on the nutritional management of CKD is needed to ensure the best care and quality of life for patients. As focus shifts to identifying patients with CKD early, it will be imperative to understand the role that nutrition plays in delaying the progression of CKD in much greater detail.

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Hawai'i

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■ ADVANCES IN PRACTICE:

■ Dietary supplement use in patients with chronic kidney disease

By Philippa Norton Feiertag, MEd, RD, LD. *Philippa is a clinical analyst/renal nutrition specialist with Clinical Computing, Inc. in Cincinnati, Ohio. She can be reached at feier@fusenet.com.*

Dietary supplements are defined as products containing one or more of the following ingredients: a vitamin or mineral; an herb or other botanical; an amino acid; a dietary substance to supplement the diet by increasing total nutrition intake; or a concentrate, metabolite, constituent extract or combination of these ingredients (1). A dietary supplement is further defined as a product intended for ingestion in the form of a tablet, capsule, powder, softgel, gelcap or liquid form.

During the past three decades, dietary supplement use has increased by up to 29% among adults, and dietary supplements now rank third in over-the-counter product sales (2,3). Findings from an ancillary study to the 1995-1996 Washington State Cancer Risk Behavior Survey indicated that participants took dietary supplements to feel better and prevent chronic diseases (4).

Other studies reveal that patients with chronic diseases are the most frequent users of dietary supplements. A cross-sectional study of 45,748 U.S. adults aged 50 to 75 years showed that use of multivitamin supplements plus 16 individual vitamins and minerals was higher in participants whose medical history included cancer, cardiovascular disease and other self-reported medical conditions (5). Information on supplement use, health status and hospitalizations in 11,775 adults who participated in the 1986 National Health Interview Survey suggests that supplement use is more likely and more intense among individuals with one or more chronic health problems (6).

The issue of vitamin and mineral supplementation in patients with chronic kidney disease (CKD) is important for renal nutrition professionals. Absorption, retention and activity of vitamins and minerals in patients with CKD are impacted by nutrition restrictions, uremic toxins and drug-nutrient interactions (7). Patients with CKD Stage 5 undergoing maintenance dialysis therapy experience loss of water-soluble vitamins during dialysis at rates exceeding loss with normal urinary excretion (7). Vitamin and mineral bioavailability in elderly patients may be affected not only by declining renal function but also by low acidity in the

stomach, which impairs absorption of folic acid, vitamin B-12, calcium, iron and beta-carotene (8). In maintenance dialysis patients, deficiencies have been reported most commonly for vitamin C, folic acid, vitamin B-6, 1,25-dihydroxycholecalciferol, iron, zinc and selenium (9).

According to the Renal Dietitian's Standards for Clinical Practice, renal dietitians are responsible for prescribing nutrition therapy based on analysis of biochemical parameters (10). More recently, in its proposal for revision of conditions of coverage for renal providers, Centers for Medicare and Medicaid Services (CMS) specified that the renal dietitian's responsibilities include monitoring vitamin and mineral supplementation in their patients (11). Of concern to all health care providers is the fact that despite widespread availability of dietary supplements in supermarkets, specialty stores and via the Internet, their distribution is not subject to standards applied to prescription and over-the-counter medications. Dietary supplements are regulated as foods, and there is no requirement for demonstration of effectiveness unless health claims are made in advertising or labeling (12). In this column, patterns of vitamin and mineral intake in patients with CKD will be reviewed and recommendations regarding vitamin and mineral supplementation for these patients will be summarized.

Patterns of dietary supplement use in patients with CKD

Information about a patient's current vitamin and mineral intake, as well as dietary supplement use, is needed to make recommendations and interventions regarding vitamin and mineral supplementation. Several recent studies have evaluated vitamin and mineral intake from food, and use of vitamin and mineral supplements in patients with CKD.

Block's food frequency questionnaire (version 98) was administered to 30 adult maintenance hemodialysis (MHD) patients (mean age 55.8±14.6 years) who were randomly selected from a pool of patients in a community dialysis unit (13). Vitamin and mineral intake was lower in MHD patients than in a control group without any known kidney

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disease. Statistically significant differences were observed between the MHD and control groups for vitamin C, and for the carotenoids cryptoxanthin and lycopene. Eleven MHD patients reported taking the renal multivitamin Nephrovite® (R&D Laboratories, Marina del Ray, CA) at least four times per week. Use of this multivitamin resulted in significantly higher vitamin B6 intake than in nonusers and control individuals.

In another study of vitamin and mineral intake in MHD patients, 25 patients (mean age 49.3 ± 12.9 years) received standardized instruction on keeping food records and estimating portion size before completing 9-day food records (14). Findings from this investigation indicated a wide range of vitamin and mineral intake for the small study group. Mean daily calorie intake correlated strongly with intake of vitamin E, vitamin B2, calcium, phosphorus and potassium. Mean daily protein intake correlated most strongly with vitamin B6, niacin, iron and phosphorus intake. While most patients consumed 100% of the recommended dietary allowances (RDAs) for niacin and vitamin B12, mean daily intake of vitamins A and B6 were least likely to exceed two-thirds of the RDA.

Vitamin and mineral intake has also been compared with RDAs in patients undergoing continuous ambulatory peritoneal dialysis (CAPD) (15). Seven-day food frequency questionnaires completed by 242 CAPD patients (mean age 55 ± 12 years) showed that intakes of iron, zinc, calcium, phosphorus and most water-soluble vitamins, except vitamin B12, were low in most patients. Vitamin C intake was below the RDA in almost 50% of patients and was particularly deficient in those with low residual renal function and total urea clearance. Also noted was a low intake of calories and B vitamins in patients with poor residual function and low urea clearances.

A cross-sectional survey of 100 adults (age >18 years) with CKD stages 2 through 5 used a detailed questionnaire to collect data on dietary supplements consumed, sources of information regarding dietary supplements and participants' perceptions of health improvement resulting from supplement use (16). Prevalence of dietary supplement use in this study group was 45% and number of dietary supplements used ranged from one to five or more daily. Most commonly used dietary supplements were vitamin E (50% of supplement users), multivitamin/mineral products

(47%) and vitamin C (29%). Supplement use occurred most frequently in the early stages of CKD and decreased as renal failure progressed. Supplement users obtained information about dietary supplements from a variety of sources including physicians (27%), printed materials (27%), naturopaths/herbalists (20%), the Internet (13%), friends and family (6%) and allied health professionals (5%). Seventy-six percent of supplement users reported health improvements as a result of dietary supplement use. When compared with non-users, dietary supplement users were significantly more concerned with their health and preferred involvement in health-related decisions.

Collectively, these studies indicate wide variability in vitamin and mineral intake among patients with CKD. Patients undergoing maintenance dialysis therapy may be particularly at risk for low intake of vitamin B6 and vitamin C (13-15). However, variability in micronutrient intake among patients underscores the need for renal nutrition professionals to identify current vitamin and mineral intake to determine appropriate and individualized levels of supplementation for their patients. High incidence of vitamin E supplementation in patients with CKD may be of concern due to its ability to increase bleeding risk in patients who are also taking the anticoagulant warfarin (16,17).

Recommendations for vitamin and mineral supplementation in patients with CKD

Table 1 summarizes general guidelines for vitamin supplementation in patients with CKD. As more information about vitamin status and metabolism in CKD becomes available, these recommendations are likely to change (19). The pharmacological use of some vitamins is currently under investigation in sub-groups of the CKD population, especially in those with known risk factors for cardiovascular disease.

Currently, 60 mg of vitamin C daily is recommended for patients with CKD to offset increased loss in the urine or into dialysate (19, 20). However, vitamin C is an important serum and intracellular antioxidant, and its beneficial effects include inhibition of lipid peroxidation, which has been associated with damage to cellular macromolecules, inactivation of antioxidant enzymes and increased risk of atherosclerosis. Maintenance dialysis patients show both vitamin C deficiency and oxidative stress, and there is interest in the effects of higher doses of vitamin C on

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Table 1. Recommendations for daily vitamin supplementation in patients with Chronic Kidney Disease (CKD) (19)

Vitamin	Pre-dialysis CKD	Chronic HD/PD
Vitamin C	60 mg/day	60 mg/day
Vitamin B1	1-5 mg/day	1-5 mg/day
Vitamin B2	1.2-1.7 mg/day	1.2-1.7 mg/day
Niacin	13-19 mg/day	13-19 mg/day
Vitamin B6	5 mg/day	10 mg/day
Vitamin B12	2 mcg/day	2 mcg/day
Folic acid	1 mg/day	1 mg/day
Vitamin B5	4-7 mg/day	4-7 mg/day
Biotin	30-100 mcg/day	30-100 mcg/day
Vitamin A	none	none
Vitamin E	unknown	unknown
Vitamin K	none	none

oxidant levels in these patients (21). While short-term oral vitamin C supplementation (250 mg three times per week for 2 months) does not modify markers of oxidative stress in maintenance HD patients, the effects of higher vitamin C doses over a longer time period remain to be investigated.

Vitamin E, a fat-soluble antioxidant and scavenger for oxygen-free radicals, has also been investigated for its potential protective effects against lipid peroxidation in patients with CKD. Studies of vitamin E status in both pre-dialysis and maintenance dialysis patients show contradictory results and have reported decreased, normal and elevated serum vitamin E levels (22). When combined with the lipid-lowering agent atorvastatin calcium (Pfizer Inc. New York, NY), 800 IU alpha-tocopherol given once daily for 12 weeks decreased oxidisability of low-density lipoprotein (LDL) in patients undergoing maintenance dialysis therapy (23). However, alpha-tocopherol administered daily for 12 months to hemodialysis patients increased levels of anticardiolipin antibodies, which have been linked with higher incidence of thrombosis and progression of atherosclerosis (24).

Serum levels of lipoprotein (a), a risk factor for atherosclerotic heart disease, increase as renal failure progresses, but

can be lowered by the administration of niacin derivatives (25). The National Kidney Foundation Kidney Disease Outcomes Quality Initiative (NKF K/DOQI) Clinical Practice Guidelines for Managing Dyslipidemias in Chronic Kidney Disease also recommend niacin as a therapeutic agent (26). Niacin therapy may be considered in this population when lifestyle changes including nutritional intake, weight reduction, physical activity and abstinence from alcohol fail to reduce triglycerides to < 500 mg/dL (< 5.65 mmol/L) or when lifestyle changes in combination with statin therapy are insufficient to reduce LDL to < 100 mg/dL (< 2.59 mmol/L). Recommended dose ranges for niacin are shown in Table 2. Although no data is available in the CKD population, studies in the general population show that niacin reduces triglycerides by 20-50%, reduces LDL by 5-25% and increases high-density lipoprotein (HDL) by 15-35%. Adverse effects of niacin therapy may include flushing, hyperglycemia and hepatotoxicity.

Table 2. Niacin dose for treatment of dyslipidemias in Chronic Kidney Disease (CKD) (26)

Agent	Dose (g/day)
Immediate release	1.5 - 3.0
Extended release	1.0 - 2.0
Sustained release	1.0 - 2.0

Hyperhomocysteinemia (total homocysteine > 1.89 mg/L [14.0 µmol/L]) is present in over 85% of patients with moderate to severe CKD (Stages 3 to 5), promoting endothelial changes that accelerate atherosclerosis and contribute to cardiovascular disease (27-30). Folic acid, which is essential for homocysteine metabolism, reduces homocysteine levels in the non-CKD population when given as an oral supplement. Ten MHD patients (mean age 37±2 years) who received 10-20 mg folic acid daily for 6 months experienced a significant drop in homocysteine levels, suggesting that relatively large doses of folic acid may decrease cardiovascular disease risk factors (31).

A meta-analysis of 12 randomized trials of vitamin supplements in the non-renal population was directed to determine the optimal folic acid dose to lower homocysteine levels and to evaluate the additional effects of vitamins B6

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and B12 (32). According to this investigation, a daily dose of 0.5 to 5 mg folic acid reduced homocysteine levels by 25%, and 0.5 mg vitamin B12 daily produced an additional 7% reduction in homocysteine levels. Vitamin B6 (mean dose 16.5 mg) did not have any significant effect on homocysteine level.

Similar studies have recently been conducted in maintenance dialysis patients, and high doses of folic acid, vitamin B6 and vitamin B12 for 6 months normalized homocysteine levels in 72% of patients undergoing CAPD (33). In a prospective, randomized, double-blind trial of 60 stable MHD patients (mean age 68 ± 13 years), participants received either a daily renal multivitamin containing 5 mg folic acid, 10 mg vitamin B6 and 0.4 mg vitamin B12 or supraphysiological doses of these vitamins (15 mg, 100 mg and 1 mg respectively) (34). The renal multivitamin elicited a 23.6% reduction in homocysteine levels after one month and a 28.3% reduction after 6 months; homocysteine levels remained stable thereafter and only 12% of participants had normal homocysteine levels after 12 months of therapy. Supraphysiological doses of folic acid, vitamin B6 and vitamin B12 did not improve outcomes.

Although the trace elements iron, selenium and zinc are commonly deficient in patients with CKD, there is no data to support routine use of oral supplements (22). While oral iron is the cheapest and easiest means of supplementing iron intake, poor absorption from the gut combined with gastrointestinal side effects and interaction with calcium-based phosphate binders limits effectiveness of these supplements.

Selenium is essential for the activity of the enzyme glutathione peroxidase, which helps to protect cells against oxidative damage (22). Low selenium levels have been linked with lipid peroxidation abnormalities in patients undergoing MHD. While low serum selenium levels in MHD patients have been attributed to poor dietary protein intake and losses through the dialyzer membrane, a recent investigation of selenium status in this population reported plasma selenium concentration within the normal range ($1.51\text{--}1.70 \mu\text{mol/L}$ [$119\text{--}134 \mu\text{g/L}$]) (22,35). A liquid formula supplemented with selenate maintained serum concentrations in the normal range in stable, well-nourished MHD patients who were adequately dialyzed; plasma selenium concentrations were higher in this group of patients than in those who did not

receive the supplement (35). MHD patients who received erythropoietin (EPO) and an oral selenium supplement showed increased glutathione peroxidase activity in their red blood cells as well as significantly higher serum selenium concentrations when compared with MHD patients who received EPO therapy alone (36). Nevertheless, selenium supplementation in patients with CKD requires caution because of its potential toxic effects.

Low levels of zinc have been reported in pre-dialysis patients with CKD and in patients undergoing maintenance dialysis therapy (22). Zinc deficiency has been linked to poor appetite and altered sense of taste and smell, which occur commonly in renal failure. While hemodialysis has minimal effects on serum zinc levels, red blood cell zinc levels are lower in CAPD patients than in those receiving MHD. In a study designed to determine the effects of zinc supplementation on serum zinc and cholesterol levels, 20 MHD patients received either 50 mg elemental zinc or placebo for 90 days and completed 2-day food records (one dialysis day and one nondialysis day) at the beginning and end of the study (37). Patients who received the zinc supplement showed significant increases in serum zinc and total cholesterol levels, both of which had been low at the onset of the study. In addition, mean reported energy intake in the zinc supplemented group increased from 1385 to 1682 kcal/day. On the contrary, CAPD patients who received 100 mg element zinc daily showed no improvement in nutritional status after 90 days, despite increased serum zinc levels (38).

Applications for the renal nutrition professional

Patients with CKD report that they obtain information about dietary supplements mainly from physicians and printed materials (16). However, patients are not routinely asked about dietary supplement use by either physicians or nurses, and there is limited availability of educational materials on vitamin and mineral supplements for patients with CKD (1,39,40). Nutrition professionals may be best suited to identify use of dietary supplements when they collect information on a patient's nutritional intake during the nutritional assessment phase of medical nutrition therapy (1).

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Wide variability in vitamin and mineral intake is evident in patients with CKD (13-16). For this reason, the renal nutrition professional must gather detailed information about vitamin and mineral intake from food, and use of vitamin and mineral supplements. Since intake of vitamins B2, niacin, B6 and vitamin E correlates with mean daily calorie and protein intake, nutrition counseling to increase both calories and protein could significantly increase vitamin and mineral intake (14).

Table 3 shows the vitamin and mineral contents of supplements formulated to meet the requirements of patients with

CKD. The need for vitamin and mineral supplementation depends on the extent of renal insufficiency, type and adequacy of renal replacement therapy, the patient's nutritional status and prescribed medications (14,15,22). Recommendations regarding vitamin supplementation should therefore be used as guidelines that may be individualized according to the patient's nutritional intake and medical status. Antibiotics can decrease absorption of vitamins A, D, K, B12, B6, folic acid and zinc while fecal loss of most vitamins increases with laxative use (41). Patients taking anticonvulsants may have impaired absorption or

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Table 3. Comparison of vitamin supplements formulated for patients with Chronic Kidney Disease (CKD)

Product	Vit C mg	B1 mg	B2 mg	Niacin mg	B6 mg	B12	Folic Acid	B5 mg	Biotin mcg	Vit A IU	Vit E IU	Zn mg	Se mcg
Dialyvite®	100	1.5	1.7	20	10	6 mcg	1 mg	10	300	-	-	-	-
Dialyvite® with Zinc	100	1.5	1.7	20	10	6 mcg	1 mg	10	300	-	-	50	-
Dialyvite® 3000	100	1.5	1.7	20	25	1 mg	3 mg	10	300	-	30	15	70
Diatx®	60	1.5	1.5	20	50	1 mg	5 mg	10	300	-	-	-	-
Diatx® Zn ^a	60	1.5	1.5	20	50	2 mg	5 mg	10	300	-	-	25	-
Nephrocaps®	100	1.5	1.7	20	10	6 mcg	1 mg	5	150	-	-	-	-
Nephron FA® ^b	40	1.5	1.7	20	10	6 mcg	1 mg	10	300	-	-	-	-
Nephrovite®	60	1.5	1.7	20	10	6 mcg	0.8 mg	10	300	-	-	-	-
Nephrovite® Rx	60	1.5	1.7	20	10	6 mcg	1 mg	10	300	-	-	-	-
NephPlex® Rx	60	1.5	1.7	20	10	6 mcg	1 mG	10	300	-	-	12.5	-
PS Nephro-Aid	60	1.5	1.5	20	20	1 mg	950 mcg	10	300	3000	100	-	-
RenaPlex®	60	1.5	1.7	20	10	6 mcg	0.8 mg	10	300	-	-	12.5	-
Renax	50	3.0	2.0	20	16	12 mcg	2.5 mg	10	300	-	35	20	70

^a Diatx® Zn also contains 1.5 mg copper gluconate.

^b Nephron FA® also contains 200 mg ferrous fumarate.

Dialyvite®, Dialyvite® with Zinc and Dialyvite® 3000 are registered trademarks of Hillestad Pharmaceuticals, Woodruff, WI.

Diatx® and Diatx® Zn are registered trademarks of PamLab, LLC, Covington, LA.

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utilization of vitamins B6, B12, folic acid, D and K, and anti-gout medications increase excretion of vitamin B2. Patients with CKD who are prescribed diuretics have increased urinary excretion of vitamin B1 and zinc, and lipid-lowering agents decrease vitamins A, D, K and B12 (41). Corticosteroids increase the need for vitamins C, D, B6 and folic acid, and for zinc while levels of folic acid, vitamin C and vitamin B12 are impacted by anti-inflammatory agents.

Higher doses of niacin, vitamin B12 and folic acid than are currently recommended may be needed to decrease risk factors for cardiovascular disease in patients with CKD (26,33, 34). While renal failure is also accompanied by oxidative stress, currently available data provides limited evidence for the benefits of antioxidant supplements in this population (42). As more investigations are performed on the pharmacologic use of vitamins in patients with CKD, recommendations regarding supplementation in these patients are likely to change (19). Therefore, it is important for the nutritional professional to balance each individual patient's needs with the current scientific evidence available when making dietary supplement recommendations and intervention in the CKD population.

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The results of the *Compensation & Benefits Survey of the Dietetics Profession 2005* are in and the news is good: dietetics salaries are on the rise. The survey, conducted May 11 to July 5, 2005 by Readex Research on behalf of the American Dietetic Association (ADA) and the Commission on Dietetic Registration (CDR), collected demographic, employment, and compensation data from over 12,000 dietetics professionals. Both registered dietitians (RDs) and dietetic technicians, registered (DTRs) reported wage gains equal to or greater than inflation, an 8.2% increase over the median income levels reported in the previous compensation survey, conducted in 2002. It was also found that dietetics professionals receive benefits packages considered among the best in any profession.

In order to understand the factors that contribute to the level of compensation dietetics professionals receive, the survey collected specific information about education levels, work experience, job responsibilities, ADA membership, and practice area. First, it was found that higher education isn't just its own reward, as advanced degree earned brought with it a substantial increase in median income, with a jump of \$5,000 per year a Master's degree, and a median increase of over \$20,000 a year for a Doctoral degree. Dietetics professionals also earn more than the national average for each degree they earn. When it comes to work experience and responsibility, the more you have, the more you earn. Not surprisingly, income continues to rise as experience is gained. Compensation also increases with greater responsibility, as management of both employees and budgets brings significant gains in pay. Earnings varied according to practice area, and the survey identified those areas experiencing the most growth as well as those with the highest salaries, with private practice being the most lucrative. Last but not least, the survey found that ADA members had a median income more than \$3,000 higher than non-ADA members.

The complete, 190-page *Compensation & Benefits Survey of the Dietetics Profession 2005* contains much more detailed, in-depth analysis of salaries, including compensation analysis for 48 different positions in dietetics and salary calculation worksheets to help dietetics professionals determine their own fair level of compensation. To order your own copy of the survey, call ADA at 800-877-1600, ext. 5000 or visit the online catalog at www.eatright.org/catalog.

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■ STIPEND REPORT: Demonstrated Best Practice:

■ Bone Mineral Metabolism

By Jean I. Olson, MS, RD, LD. *Jean works as a renal dietitian at DaVita Dialysis Clinics in St. Paul and Stillwater, Minn.*

The following is a summary of a presentation by Marianne Hutton, RD, CDE, which took place at the NKF — Clinical Meetings, - Chicago, Ill. on April 23, 2006

The presenter discussed how her dialysis clinic achieved top ranking for bone mineral outcomes. The achievement was attained through the following key factors: 1) developing and using a protocol based on medical evidence. 2) identifying target groups of patients and defining care based on laboratory values. 3) implementing a successful patient education/motivation program.

Key concepts

- The root problem is dietary phosphorus and phosphate binder non-adherence.
- Calcium and phosphorus will be higher if PTH (parathyroid hormone) is not aggressively managed.
- Bone resorption is a key process that must be controlled.
- Individual patient's labs must be carefully tracked for trends with adjusted Vitamin D doses based on previous individual patient response.
- Calcimimetics allow the Vitamin D dose to be increased as they cause the serum calcium, phosphorus, and product to decrease.

The protocol encourages

1. Vitamin D analogs for most patients.
2. Titrating rather than holding Paricalcitol for $[Ca^{++} \times P]$ product > 56 due to high phosphorus levels $[> 5.6 \text{ mg/dl}]$.
3. Holding Vitamin D in patients with high levels of calcium, phosphorus, or $Ca \times P$ product:
 - a) May not be an effective strategy, and b) May delay the initiation of appropriate corrective actions. These include dietary counseling, adjusting dose of phosphate binders, reducing dose of calcium supplements, and adjusting dialysate calcium.
4. Holding Paricalcitol in patients with high phosphorus and/or calcium is seldom required. If $[Ca^{++} \times P]$ product

> 56 due to high Calcium levels of $[> 10.2]$, oral calcium supplements should be reduced or discontinued first. If the calcium remains elevated Paricalcitol dose should be reduced to $< 10 \text{ mcg}$ 3 times per week. If serum calcium remains elevated, Paricalcitol should be held.

5. Calcium free phosphate binders. Especially if PTH levels are continuously low, calcium supplements/sources should be stopped since extra calcium suppresses PTH.
6. Aggressive dietary counseling to maintain serum phosphorus levels under 5.5.

The protocol discourages

1. Holding Vitamin D therapy which results in immediate increases in PTH levels and augmented skeletal resorption.
2. Use of Calcitriol which is associated with more sustained episodes of hypercalcemia and increased $[Ca^{++} \times P]$ product.

Ideas for patient education/motivation and other suggestions to improve outcomes

- Listen to patients and gain their trust. Find out barriers to their compliance and help them set realistic goals. Follow-up with them as soon as possible on lab rechecks and be positive on any improvement no matter how little. Assist patients with the development of their individual goals.
- Work toward successful patient education and behavior change by building on previously learned concepts and get the patient's buy-in, i.e. What's in it for me? Use a variety of educational methods and tools including handouts, bulletin boards and visuals such as examples of phosphate binders.
- Involve other members of the health care team, both in-center such as RNs and PCTs to reinforce your teaching and help motivate the patient. Develop a good working relationship with patient families, RDs and other care providers at nursing homes and residential facilities. Partner with vendors and clinical support specialists to hold staff inservices, patient education days and other learning and motivational activities that are fun.

Continued on page 23

- Collaborate with the LCSW to evaluate and define strategies for improved patient compliance and positive behavior change. Enlist the LCSW to aid in obtaining prescription coverage, thus improving patient potential for medication compliance.
- Utilize information technology reports to identify patient profiles and to track trends.

Suggested Readings:

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¹ Nissenson, et. al. AJKD 2003; 42:325-330 (data on file)

Nutrition Labeling and Food Fortification

Deborah Brommage

Food fortification and the use of food additives in food processing are growing concerns for patients with kidney disease. In most cases the proportion of additives is relatively high in contrast to the actual food content.⁽¹⁾ These practices in combination with limited information on nutrition labels makes patient adherence to the renal diet more challenging than ever and affects our ability as renal dietitians to help our patients with kidney disease make good food choices.

Fortified Foods and Food Additives

Vitamin and mineral compounds are added to foods in an effort to increase the nutrient content that occur naturally. Food fortification is helpful for the general population to achieve adequate intake and prevent deficiencies, but it poses a problem for patients with kidney disease, especially when package labels do not clearly specify these additions. A prime example is the addition of calcium to foods such as juices, cereals and breads which can result in calcium intake above the KDOQI recommendation of 2000 mg per day.

Another growing concern is the increased use of food additives that act as flavor enhancers, preservatives, leavening agents, etc. While additives positively impact the quality of convenience foods for most consumers, patients with kidney disease must beware. Additives such as phosphates may significantly increase the phosphorus intake beyond recommended levels for patients with kidney disease, even when dairy and other naturally high phosphorus content foods are restricted.

Product Update

Keeping updated on the overabundance of new food products and food processing trends can be overwhelming. Product Update, an online feature of the Journal of Renal Nutrition, is a valuable resource for learning about new or reformulated foods on the market and the implications for the renal diet with regard to sodium, potassium, calcium or phosphorus content. This free electronic journal feature is available at www.jrnjournal.org.

In 2005, a three part series on "Hidden Phosphorus" authored by CRN member Lisa Murphy-Gutekunst, MEd, RD, CSR, CDN, focused on phosphorus additives to beverages, breakfast foods and enhanced meats. (2-4) This series is a "must-have" reference for anyone working to control serum phosphorus levels in patients with chronic kidney diseases.

Nutrition Labeling Challenge

The Nutrition Labeling and Education Act of 1990, which requires that certain nutrient and food components be included in nutrition labeling, indicates that the Secretary of Health and Human Services can add or delete nutrients included in the food label if the modification is deemed necessary to help consumers in maintaining healthy dietary practices. An example of such a change took place in July of 2003 when the Food and Drug Administration (FDA) issued a final rule mandating that the amount of trans fatty acids present in foods be included in the nutrition label (5). This amendment was in response to an increasing body of evidence to suggest that trans fatty acids raise blood cholesterol levels, thereby increasing the risk of coronary heart disease.

The complications related to elevated serum potassium and phosphorus levels in patients with kidney disease are well known to the nephrology community. Less well known are the potassium and phosphorus content of packaged foods, especially when food additives are involved, as previously mentioned. The lack of potassium and phosphorus information on nutrition labels has been a growing concern for patients with chronic kidney disease and nephrology professionals. One CRN member, Jennifer Kurzawa, RD, was so passionate about this issue that she began a letter writing campaign to the FDA. Jennifer, along with Cathi Martin, RD, CSR, LDN, now serves as a technical advisor on a joint project with the National Kidney Foundation and American Dietetic Association. The panel has developed a citizen petition to the FDA to amend the nutrition fact panel on food packages so that people with kidney disease can improve their health management. The petition will request that potassium and phosphorus content of packaged foods be required on the nutrition facts panel. We know we can count on the support of CRN and RPG members for this monumental endeavor.

Continued on page 25

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3. Murphy-Gutekunst L, Barnes, K: Hidden phosphorus at breakfast: Part 2. *JREN* 15:e1-e6, July 2005
4. Murphy-Gutekunst L, Uribarri J: Hidden phosphorus-enhanced meats: Part 3. *JREN* 14:e1-e4, October 2005.
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RPG CHAIR MESSAGE

Cathi J. Martin, RD, CSR, LDN
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The time has certainly flown by and it is time for the Chair-Elect, Patricia Weber, MS, RD, CDE, CSR, LD to move into the position as Chair. Pat works with Fresenius Medical Care (formerly Renal Care Group) in Anniston and Talladega, Alabama. I am leaving you in very capable hands and wish her well in the upcoming year. It has been such a pleasure to lead the Renal Practice Group this year, and I am so impressed by the dedication of so many individuals involved on the Executive Committee and various committees. I would like to thank each person for their commitment and hard work over the past year.

The Renal Practice Group has accomplished a number of achievements over the past year. A leadership retreat was held last Spring for all incoming, incumbent and outgoing officers to assure a smooth transition for the new Board. The Renal Nutrition Forum has continued to evolve and provides you with the latest and cutting-edge knowledge available. We have successfully marketed the *Eating Simply with Renal Disease* pamphlet and also authored a Spanish version which is now available. RPG is active in Government Affairs including activity with a proposal to the FDA for including Phosphorus, Calcium and Potassium levels on food labels and piloting a brochure for the National Kidney Disease Education Project (NKDEP). We have been involved in supporting ADA efforts to increase reimbursement for Medical Nutrition Therapy. We are also working on some major improvements to the RPG website, including e-blast capability to keep you better informed. In addition, we part-

nered with Ross Products Division of Abbott Laboratories to review a videotape on the dialysis diet. Perhaps the most exciting project is creating the Scope of Practice for Nephrology Care. RPG and the National Kidney Foundation's Council on Renal Nutrition have partnered to establish a Scope of Practice and Standards of Professional Performance using the ADA's nutrition care model. The timeline for accomplishing this goal is the end of 2006.

Please join me in welcoming our incoming Executive Committee members: Patricia Weber, MS, RD, CDE, CSR, LD — Chair; Lois Hill, MS, RD, CSR — Chair-Elect; Jane Louis, MS, RD, CSR — Secretary, Pamela Kent, MS, RD, CSR returning as treasurer. In addition, our Editorial Board will be returning, along with our new assistant editor, Lesley Wujastyk, RD. The Nominating Committee will be chaired by Kathleen Madigan, MS, MBA, RD, CSR, LDN and include Paula Frost, RD, LD and Joanne Cooke, MS, RD. The Membership Chair, Connie Cranford, MS, RD will remain, along with all of the Area Coordinators.

As always, YOU are our most important and valuable asset. I want to encourage you to continue to contact us whenever you have a question. We are striving to improve communication channels.

Best regards,

Cathi

Answers to CPE questions: 1. A, 2. C, 3. D, 4. B, 5. C, 6. B, 7. B, 8. A, 9. A, 10. D

Renal Practice Group: Educational Materials

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Eating Simply with Renal Disease: (25 per pkg) Simple overview of the renal nutrition plan for patients with CKD Stage 5	\$15.50 per pkg
Eating Simply with Renal Disease: (25 per pkg) Spanish version of the renal nutrition plan overview for patients with CKD Stage 5	\$15.50 per pkg
Bone Store Kit: Patient education module focusing on calcium and phosphorus to improve mineral metabolism imbalances	\$10.00 each
Dietitian's Manual: Camera-ready, single-page patient education compiled from previous Renal Nutrition Forum Issues	\$10.00 each

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Vision: RPG members are a valued source of expertise in nephrology nutrition.*

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