

# Renal Nutrition Forum

A Peer Reviewed Publication of the Renal Dietitians Dietetic Practice Group

**Volume 31 • Number 1**

## In This Issue

1  
Feature Article

2  
Letter from the Editor

14  
Protein-Energy Wasting  
in CKD Patients: Are We  
Wasting an Opportunity  
to Improve Outcomes?

16  
2010 Guidelines and  
My Plate

20  
Member Spotlight

22  
Member Spotlight

23  
Member Spotlight

24  
Renal Dietitians Chair  
Message

25  
Calendar of Events

27  
RPG Executive  
Committee

## Interpreting and Applying the Kidney Disease: Improving Global Outcomes (KDIGO) Guidelines for Chronic Kidney Disease-Mineral and Bone Disorder (CKD-MBD): A Guide for Renal Dietitians

**Linda McCann, RD, CSR, LD**  
Vice President of Clinical Applications  
Satellite Healthcare, Inc  
San Jose, CA  
E-Mail: MccannL@SatelliteHealth.com

**This article has been approved for 2 CPE units. The online CPEU quiz and certificate of completion can be accessed in the Members Only section of the RPG web site via the My CPEU link. This CPE offering is available to current RPG members only and the expiration date is April 15, 2013.**

### Abstract

The Kidney Disease: Improving Global Outcomes (KDIGO) Guidelines for Chronic Kidney Disease Mineral and Bone Disorder (CKD-MBD), published in 2009, represent a paradigm shift from renal osteodystrophy to a broader description of the complex, underlying metabolic anomalies that develop as CKD progresses. CKD-MBD encompasses abnormal calcium, phosphorus, PTH and vitamin D metabolism, and changes in bone turnover and structure, as well as increased risk for vascular and soft tissue calcification.

The KDIGO guidelines were developed under more rigorous evidence review criteria than previous guidelines. They broaden acceptable ranges of surrogate bone and mineral biochemical markers and recognize the

importance of repleting nutritional vitamin D while continuing to guide interventions to minimize the risk for secondary hyperparathyroidism and soft tissue calcification. Dietitians have long been important members of the nephrology care team, especially in managing bone and mineral metabolism. Their expertise and experience will help promote the implementation of these KDIGO guidelines.

**Key words:** KDIGO; K/DOQI; CKD-MBD; phosphate; calcium; PTH; CKD

### Introduction

Outpatient dialysis clinics provide a unique opportunity for advanced dietetic practice as an extension of nephrologists' care. Nephrologists typically visit dialysis clinics periodically to see patients and review each patient's plan of care with the care team. Nephrology dietitians and nurses, in contrast, are consistently present in the dialysis clinic and can help the nephrologist manage bone and mineral abnormalities with careful monitoring and timely interventions as guided by the KDIGO guidelines.

Nephrology dietitians currently face an economic challenge to advanced practice in the area of CKD-MBD. The Center for Medicare/Medicaid Services (CMS) has modified dialysis reimbursement by implementing the Medicare Improvements for Patients and Providers Act (MIPPA), commonly called "bundling," to ensure quality care for dialysis recipients while reducing the costs of end stage renal disease (ESRD) (1). As of January 1, 2011 an updated prospective payment system began to shift the cost of many commonly used

– Continued on page 3.

Renal Nutrition Forum is published quarterly (summer, fall, winter, spring) as a peer-reviewed publication of the Renal Dietitians Dietetic Practice Group of the Academy of Nutrition and Dietetics.

*The views expressed in this publication are those of the author and are not necessarily those of The Academy of Nutrition and Dietetics. Publication of an advertisement in the Forum should not be construed as endorsement by the RPG of the product or the advertiser.*

Articles about successful programs, research interventions, evaluations and treatment strategies, educational materials, meeting announcements and information about educational programs are welcome and should be emailed to the editor by the next deadline.

#### Future Deadlines:

June 1, 2012  
September 1, 2012  
December 1, 2012  
March 1, 2013

Please forward information to:  
Sara Erickson, RD, CSR, LDN, CNSC  
saraericksonrd@gmail.com

Subscription cost is \$35.00 for individuals who are ineligible for Academy membership and \$50.00 for institutions. A check or money order should be made payable to AND/DPG #21 and sent to:

Stacey C. Phillips, MS, RD  
4360 4 Mile Road NE  
Grand Rapids, MI 49525

**Remember to update your profile electronically in the 'members only' section of the Academy's web site. You will need your registration number and web password. Keeping the Academy informed of your name and contact information will help avoid delayed issues of your Renal Nutrition Forum.**

## From the Editor's Desk

**Sara Erickson, RD, CSR, LDN, CNSC**  
Editor



Hello RPG Members!  
We are pleased to offer 3 CPEU hours in this issue of the Renal Nutrition Forum. For those members who are preparing for the bundling deadline, our featured article, authored by Linda

McCann, RD, CSR, provides an excellent guide in the nutritional assessment and management of metabolic bone disease. Our advanced article, co-authored by Abby C. Sauer, MPH, RD, LD and Anne Coble Voss, PhD, RD, LD, provides a literature review on the benefits of using a renal specific oral nutrition supplement for patients with chronic kidney disease.

Also included in this issue, we hear from three RPG Educational Stipend Award Winners! Adele Huls, PhD, RD, LMNT, Cheryl Montgomery, RD, LDN, and Marianne Wolfe-Hutton, RD, CSR, CDE attended the 2011 Food & Nutrition Conference & Expo in San Diego, Ca. These recipients provide summaries of their favorite FNCE sessions and/or experiences. If you are planning to attend a conference in the near future, don't miss out on this RPG member benefit! To learn more, visit the "awards/stipends" section on [www.renalnutrition.org](http://www.renalnutrition.org).

Sincere thanks to my fellow Editorial Board Members, Megan Sliwa, RD, LDN, MBA and Jackie Abels, MA, RD, LD for all their support. Finally, Thank you to the peer-reviewers who provide invaluable feedback, the authors for their contributions, and to Amy Hess-Fishl, MS, RD, LDN, BC-ADM, CDE, for providing the CPEU test questions.

Best Regards,

### Erratum

#### From Fall 2011:

Please accept our apologies, in the Featured Article of the Fall 2011 *Renal Nutrition Forum* (Vol. 30, No. 4), the author's email did not appear correctly. The correct email is [joanhogan@fhshealth.org](mailto:joanhogan@fhshealth.org). Please note that on the RPG web site, the pdf version of the Fall Forum and the pdf of the article have both been corrected.

**Would you like to attend an upcoming conference?**

**Are you pursuing a post-baccalaureate degree in a field related to nutrition?**

**Are you looking to do research in an area benefitting people with renal disease?**

Check out the awards, grants and scholarships area on the RPG website at

[http://www.renalnutrition.org/members\\_only/awards.php](http://www.renalnutrition.org/members_only/awards.php)

**Don't miss out on these opportunities!**  
**Applications are due by April 30, 2012**

# Feature Article...

dialysis medications and laboratory tests to dialysis providers. Providers were allowed to embrace the new payment system in full as of January 1, 2011 or phase it in over a three-year period. The final result is that payment for drugs and services such as erythropoietic agents, oral or intravenous vitamin D analogs, intravenous antibiotics, and common ESRD laboratory tests have become or will become the responsibility of dialysis providers. This shift in reimbursement challenges clinicians to maintain optimal care while controlling costs (2,3). CKD-MBD care will face an even greater challenge when phosphate binders and calcimimetics (e.g., cinacalcet) become bundled on January 1, 2014 (3). Guideline-based practice under MIPPA requires balancing systemic resource constraints with individual patients' needs.

Dietitians and nurses in the United States have helped to implement the 2009 KDIGO CKD-MBD guidelines, which were reinforced by the late 2010 publication of a US Kidney Disease Outcome Quality Initiative (K/DOQI) commentary on KDIGO (2,4). The K/DOQI commentary supports the majority of the KDIGO guidelines and agrees that greater flexibility in treatment goals is appropriate given the uncertainty and scarcity of evidence for many current treatment approaches (2).

KDIGO's move away from absolute numerical targets to wider ranges and time trends empowers clinicians to individualize care. Understanding the logic and evidence behind the KDIGO targets will help dietitians interpret and apply them appropriately. The KDIGO workgroup systematically reviewed and graded evidence according to study designs, evidence quality and strength, and risk-benefit assessment (methods summarized in Table 1) (4–6). KDIGO's recommendations for identifying and managing patients at risk for disordered calcium and phosphate metabolism and secondary hyperparathyroidism (SHPT) have the potential to help avoid adverse outcomes.

## Objective

This review will summarize the 2009 KDIGO guidelines for management of CKD-MBD, comment on their differences from the K/DOQI 2003 guidelines as emphasized in the 2010 K/DOQI commentary and help dietitians interpret and apply the KDIGO guidelines for improved mineral management in patients with CKD stages 3–5D.

## KDIGO guidelines for mineral monitoring and maintenance levels

KDIGO guidelines' divergence from the earlier K/DOQI mineral management guidelines reflect new literature review criteria, new research, evolving understanding of mineral metabolism in CKD, improvements in testing technology, and changes in mineral management methods.

KDIGO defines CKD-MBD as “a systemic disorder of bone and mineral metabolism in CKD manifested by either one or a combination of the following: abnormalities of calcium, phosphorus,

**Table 1: Quality of the Evidence - Grades of Recommendations, Assessment, Development, and Evaluation (GRADE)(4,6)**

Step 1: Grade quality of evidence	
High (Randomized Trial)	Low (Observational Study)
Moderate (Quasi-randomized Trial)	Very Low (Other Evidence)
Step 2: Reduce the grade if there are:	
Quality limitations	Lack of directness
Inconsistencies	Other – data is sparse or reporting bias is very probable
Step 3: Raise the grade if there is:	
Association strength	
Dose response shows evidence of gradient	
Minimal confounders	
Step 4: Final Evidence Quality Grade	
High (A)	“We are confident that the true effect lies close to that of the estimate of the effect.”
Moderate (B)	“The true effect is likely to be close to the estimate of the effect, but there is a possibility that it is substantially different.”
Low (C)	“The true effect may be substantially different that the estimate of the effect.”
Very Low (D)	“The estimate of the effect is very uncertain and often will be far from the truth.”
Using the levels and strength of the evidence to draft the guideline statements.	
“We recommend”:	
<ul style="list-style-type: none"><li>• Most patients should receive the recommended course of action.</li><li>• High quality evidence warrants a strong recommendation.</li><li>• A large difference between the desired and undesired effects of an intervention warrants a strong recommendation.</li></ul>	
“We suggest”:	
<ul style="list-style-type: none"><li>• Different treatment decisions will be made for different patients who should be helped to select the interventions based on their values and preferences.</li><li>• A narrow gradient between desired and undesired effects of an intervention warrants a weak recommendation.</li><li>• Variability or uncertainty in treatment values or preferences warrants a weak recommendation.</li><li>• High resource cost of an intervention warrants a weak recommendation.</li></ul>	

# Feature Article...

parathyroid hormone (PTH), or vitamin D metabolism; abnormalities of bone turnover, mineralization, volume, linear growth, or strength; vascular or other soft tissue calcification” (7). Mineral and endocrine derangements begin earlier in CKD than bone-level changes (7). In response to increasing phosphate load and decreasing calcitriol levels, fibroblast growth factor-23 (FGF-23) and PTH increase per-nephron phosphate excretion via sodium-dependent phosphate co-transporters NPT2a and NPT2c (8). FGF-23 and PTH levels increase as CKD progresses in compensatory adaptations striving to maintain normophosphatemia (8). Increased FGF-23 is an early biochemical marker of mineral derangement (9). In the prospective Chronic Renal Insufficiency Cohort (CRIC), elevated FGF-23 was independently associated with mortality risk at all stages of CKD and with risk of progression to ESRD in patients with baseline GFR  $\geq 30$  mL/min (10). Biochemical signs of CKD-MBD generally manifest in adults at GFR  $\leq 60$  mL/min (CKD stage 3 and beyond) and progressively worsen as GFR declines (11).

## Monitoring and Diagnosis Recommendations

For patients in CKD stages 3–5D, KDIGO recommends routine monitoring of serum phosphate, calcium, PTH, and alkaline phosphatase (ALP), and suggests monitoring of serum 25(OH)D, at frequencies summarized in Table 2 (4).

KDIGO suggests considering serum phosphate and calcium individually rather than in the calcium x phosphate product (2). The KDIGO evaluators recognized that the calcium x phosphate product is a mathematical construct dominated by serum phosphate and that the product may be within acceptable range with either individual parameter out of range (4).

KDIGO notes that serum calcium is a poor reflection of overall body calcium, with only 1% of total body calcium measurable in the extracellular compartment. Ionized calcium, approximately 40–50% of total serum calcium, is physiologically active. The remainder is bound to albumin or anions such as citrate, bicarbonate, and phosphate, which render it physiologically inactive. When serum albumin is low, the ratio of ionized calcium to total calcium is increased (4). While measuring ionized calcium is ideal, it is more expensive, time-consuming, and less reproducible than total calcium (2,12). KDIGO recognizes that most US ESRD-specific laboratories provide a corrected calcium measurement, however, recent data corroborates that corrected calcium is not superior to total calcium and is less specific than ionized calcium (4). Among 691 French patients with CKD stages 3–5, ionized calcium was only modestly correlated with total calcium (with or without albumin correction), and weakly predicted abnormal ionized calcium (12). More accurate correction of total calcium considers total CO<sub>2</sub> concentration along with hypoalbuminemia (12). The KDIGO workgroup did not find evidence that using a corrected calcium (compared with a total calcium or an ionized calcium) changed the treatment approach or clinical outcomes, thus they did not recommend abandoning corrected calcium (4).

KDIGO recommends measuring PTH by a second-generation assay. The Allegro assay, referenced in K/DOQI, 2003, was the original second-generation PTH assay (“intact” PTH assay) and is no longer available (2,13). There have been ongoing evaluations of both second and third generation PTH assays without conclusive agreement on which assay can most reliably diagnose SHPT (14,15). Intact PTH in second-generation assays typically includes circulating fragments with variable correlation to bone resorption. This adds to the variability in PTH measurements and their inconsistent relationships to bone status

**Table 2.** KDIGO laboratory monitoring for mineral metabolic parameters (4). The frequency of monitoring serum calcium, phosphorus, and PTH should be based on the presence and magnitude of abnormalities and the rate of progression of CKD. Reasonable monitoring intervals are tabulated below. In addition to the serum parameters shown, KDIGO suggests that serum 25(OH)D be tested in stages 3–5D and post-transplant at a frequency determined by baseline level and treatment.

CKD Stage	Serum Parameter	KDIGO - Recommended Frequency
3	Phosphate & Calcium	Every 6–12 months
	PTH	According to baseline level and disease progression
4	Phosphate & Calcium	Every 3–6 months
	PTH	Every 6–12 months
	ALP	Every 12 months (more often if $\uparrow$ PTH)
5-5D	Phosphate & Calcium	Every 1–3 months
	PTH	Every 3–6 months
	ALP	Every 12 months (more often if $\uparrow$ PTH)
Post-transplant	Phosphate & Calcium	Weekly until stable; then according to abnormalities and renal function
	PTH	According to abnormalities and renal function
	ALP	Every 12 months (more often if $\uparrow$ PTH)

ALP-alkaline phosphatase; CKD-chronic kidney disease; KDIGO-Kidney Disease: Improving Global Outcomes; PTH-parathyroid hormone. The frequencies above are meant to be minimum; more frequent testing may be appropriate for individual patients or situations.



(4). KDIGO does not recommend using third-generation assays because, although their full-length bioactive PTH specificity is greater, their relationship to bone metabolism is still being investigated (13). Because PTH resistance can occur in CKD-MBD, bone status cannot be unequivocally predicted from PTH concentrations (13).

PTH concentrations may differ by a factor of  $> 2$  in the same samples using different assays (13). KDIGO suggests that laboratories report assay methods, sample sources, and sample handling, so clinicians can interpret results in the context of the assay characteristics and limitations (4). PTH levels show endogenous diurnal/circadian rhythm, therefore efforts should be made to schedule laboratory tests at a consistent time of day and day of the week (2,16). PTH levels may also be influenced by blood collection methods, sample timing in relation to dosing of calcimimetics, and other medications.

Abnormal ALP levels can be considered along with PTH to characterize bone metabolism more accurately especially when values are markedly high or low (2,4). Bone-specific ALP levels correlate with some histomorphometric measurements of bone biopsy and may be measured if the source of serum ALP is uncertain or the clinical situation is unclear (2).

KDIGO recommends extending serum 25(OH)D testing to all CKD patients (stage 3 onward) irrespective of their PTH levels, using baseline levels and therapy to guide the frequency of repeat tests (4). KDIGO recognizes the importance of seasonal 25(OH)D variations for interpreting test results (2). Summer increases may occur, with magnitudes dependent on age, gender and ethnicity (17). The level of vitamin D that represents sufficiency is the subject of an ongoing debate with no data to show how CKD would alter those levels. KDIGO suggests deciding on an individual basis whether to measure, when to measure, how often to measure and to what target level (4).

For all laboratory parameters, KDIGO emphasizes following trends over time. Mineral management should be guided by all parameters, considered together, across multiple measurements, rather than in reaction to isolated values. KDIGO's emphasis on laboratory trending, physiologic variations and potential assay disparity is meant to caution clinicians about making changes based on a single measurement, which could be skewed. This caution may contrast with performance metrics used by CMS and dialysis providers, which commonly emphasize single time-point laboratory values and interventions (2). Additionally, bundling may discourage more frequent performance of expensive laboratory tests, such as PTH. Within the framework of KDIGO's stage-specific recommendations for test intervals (Table 2), laboratory ordering frequencies should reflect individual blood levels, changes in therapy, and rates of CKD progression, rather than a clinic's effort to "make the numbers" or reduce laboratory testing costs (4). Patients with known abnormalities and/or those receiving treatment for CKD-MBD often need more frequent monitoring (4).

## Maintenance Levels: Goals for Management

KDIGO's language concerning mineral maintenance targets is "We suggest" throughout, allowing for provider and patient values, preferences, and resource allocations (4). Table 3 compares the KDIGO and K/DOQI stage-specific maintenance levels (2).

For *phosphate*, KDIGO suggests maintaining normal serum levels in CKD stages 3–5 and striving to lower elevated levels "toward normal" in 5D (4). KDIGO's phosphate target range is lower than K/DOQI's for CKD stage 5 not requiring dialysis, reflecting evidence that associates higher serum phosphate with cardiovascular risk and mortality in patients with CKD stages 3–5 (18). For dialysis recipients, KDIGO suggests lowering phosphorus toward normal because elevated phosphorus has robust, concentration-dependent associations with increased mortality in patients on either hemodialysis (HD) or peritoneal dialysis (PD) in observational studies worldwide (4,19–24). KDIGO does not provide absolute phosphate target levels in CKD stage 5D because the threshold associated with increased mortality varies among studies, and the optimal target range has not been defined by high quality outcome studies (4). Additionally, it is recognized that reducing a high serum phosphorus level has the potential to improve outcomes even if it does not reach a specific target level. Individual cardiovascular risk profiles, benefits, and patient preferences should guide phosphorus control strategies and maintenance levels (2).

For *calcium*, KDIGO suggests maintaining normal serum levels in CKD stages 3–5D (4). Among dialysis recipients, elevated serum calcium has similar associations with adverse events as elevated phosphorus (4). Thus the interpretation of serum calcium should be evaluated based on trends considering that specific medications (calcium-based binders and vitamin D sterols) raise serum levels and others (cinacalcet) lower serum levels. KDIGO suggests using dialysate calcium concentrations between 2.5 and 3.0 mEq/L (1.25 and 1.50 mmol/L) and individualizing them where possible to achieve near-neutral calcium balance (4).

For *PTH*, KDIGO states that the optimal PTH level for CKD Stage 3–5 is unknown, but suggests intervening to maintain normal levels since PTH provides early warning of CKD-MBD dysregulation (2). KDIGO generally allows a wider PTH range than K/DOQI, recognizing PTH assay variability, the diversity of PTH levels relating to bone turnover status, and their minute-to-minute physiologic changes. If PTH exceeds the reference range in CKD stages 3–5, phosphate, calcium, and 25(OH)D levels should be evaluated to identify and treat modifiable causes of that PTH elevation. Continued PTH elevation may require treatment by active vitamin D agents, even in stages prior to dialysis; calcimimetics and phosphate binders can be used concomitantly or alternatively to control PTH levels (4).

The K/DOQI 2003 PTH range for ESRD (150–300 pg/mL) does not consistently predict normal bone turnover (25). In CKD stage 5D, KDIGO suggests maintaining serum PTH between 2 to 9 times

**Table 3.** KDIGO and K/DOQI target ranges for mineral metabolic parameters by CKD stage (2)

Parameter	CKD Stage	KDIGO	K/DOQI
Serum phosphate	3 4	Reference range (normal)	$\geq 2.7$ mg/dL $\leq 4.6$ mg/dL
	5–5D	Towards normal reference range based on risk-benefit and patient preference	3.5–5.5 mg/dL
Serum calcium	3–4	Serum calcium in reference range (normal; corrected total calcium not specified)	Corrected total calcium in reference range
	5–5D		Corrected total calcium at low end of reference range: 8.4–9.5 mg/dL
Serum PTH	3	If above upper reference limit, evaluate for hyperphosphatemia, hypocalcemia, and vitamin D deficiency	35–70 pg/mL
	4		70–110 pg/mL
	5		150–300 pg/mL
	5D	Within ~2–9 times reference range or ~130–600 pg/mL with the suggestion that marked changes in PTH levels in either direction within this range prompt an initiation or change in therapy to avoid progression to levels outside this range	150–300 pg/mL
Vitamin D 25(OH)D	3–5D	Suggest 25(OH)D (calcidiol) levels might be measured with repeat testing determined by baseline values and therapeutic interventions. Vitamin D deficiency and insufficiency be corrected using treatment strategies recommended for the general population (refs. 27, 28)	No consensus on optimal levels, deficiency often defined as serum 25(OH)D values <10 or 15 ng/ml (<25 or 37 nmol/l)

the upper limit of the reference range (corresponding to ~130–600 pg/mL); PTH deviations outside this range or significant trends up or down within the range warrant starting or changing therapy (4). The PTH level associated with increased mortality varies, but typically exceeds 400–600 pg/mL (13). Though it is ideal to follow a patient's PTH trajectory over time rather than make decisions on single concentration time points, this strategy becomes more difficult with bundling, where many dialysis providers have reduced PTH testing frequencies (13).

For 25(OH)D, KDIGO recommends treating deficiency and insufficiency in alignment with the general population and acknowledges continuing controversy regarding the definitions of deficiency and insufficiency (4). KDIGO states: “Most studies define deficiency as serum 25(OH)D (calcidiol) values < 10 ng/ml (25 nmol/l), and insufficiency as values > 10 but < 20–32 ng/ml (50–80 nmol/l)” (4).

## Bone Health Considerations

Dietitians deal more directly with the biochemical abnormalities than the skeletal aspects of CKD-MBD. However, they should be able to recognize metabolic issues warranting closer attention to bone. Among these are persistent, unexplained hypercalcemia or hyperphosphatemia or possible aluminum toxicity, conditions for which KDIGO considers bone biopsy a reasonable investigation (4).

Intercurrent, age-related osteoporosis may arise in senior patients with early CKD (stages 1–2, before CKD-MBD develops) and may be comorbid with CKD-MBD in stages 3–5D (4). The World Health Organization FRAX® fracture risk assessment tool provides 10-year fracture probabilities from non-nephrogenic osteoporosis; however, bone mineral density generally relates differently to fracture risk in patients with CKD-MBD than in senior adults without CKD (26). Collagen-derived bone turnover markers are helpful in age-related osteoporosis but lack utility in CKD-MBD (4). Calcium consumption for prevention or management of comorbid non-CKD-related osteoporosis should be kept within current general-population limits as defined by the Institute of Medicine (IOM) (27). The IOM general-population recommendations (recommended intakes 1000–1200 mg per day according to age; upper tolerable limits 2000 mg per day for adults over 50, not intended as an intake level) are more restrictive than the earlier K/DOQI 2003 guidelines that limit total daily intake to 2000 mg per day for CKD patients.

## Cardiovascular Calcification

Arterial and valvular calcification occurs even in the general population, but is much more prevalent in the CKD population, contributing to increased cardiovascular mortality (4). Comorbid factors such as diabetes, hypertension, uncontrolled hypercalcemia or hyperphosphatemia, low or high PTH, or aggressive treatment with active vitamin D may increase CKD patients' calcification risk (28).

# Feature Article...

KDIGO does not recommend indiscriminate calcification screening in all patients with CKD, but does suggest considering vascular or valvular calcification in managing CKD-MBD (4). In patients with CKD stages 3–5D at risk or with known cardiovascular disease, KDIGO suggests assessing presence/absence of calcification from available tests, e.g., lateral lumbar radiographs for abdominal aortic calcification and echocardiograms for cardiac valvular calcification (4). Patients with known calcification are in the highest cardiovascular risk category (4). The KDIGO workgroup extensively discussed the role of calcium versus non-calcium-based binders in the pathogenesis of vascular calcification and acknowledged that the evidence is not conclusive. Based on current knowledge of calcium balance in CKD, KDIGO suggests restricting the dose of calcium-based phosphate binders in the presence of persistent hypercalcemia, arterial calcification, adynamic bone disorder, and/or persistently low serum PTH.

## KDIGO guidelines for treatment of CKD-MBD

Hyperphosphatemia is also associated with vascular calcification, cardiovascular disease, and mortality in CKD-MBD (20,29). Thresholds for increased mortality vary among studies from 5.0 to 7.0 mg phosphate/dL (2). Phosphate control is important to avoid vascular and endocrine sequelae as the kidneys' ability to excrete phosphate declines. Phosphate control interacts with management of calcium, vitamin D, and PTH; treatment of CKD-MBD with phosphate binders and other agents should take all these parameters into account.

## Management of Mineral Metabolism

Disordered mineral metabolism entails hypocalcemia, hyperphosphatemia and hypovitaminosis D, and precedes the development of frank metabolic bone disease, which is associated with increased risk of fractures and mortality in dialysis patients (30). Screening for calcium, phosphate, PTH and vitamin D (calcidiol) is recommended, followed by treatment with nutritional and active

vitamin D, dietary modification, calcimimetics, and phosphate binders (30).

Based on moderate evidence, KDIGO suggests using phosphate binders to treat hyperphosphatemia in CKD stages 3–5D (4). Labeling by the US Food and Drug Administration (FDA) allows use of non-calcium phosphate binders only in dialysis patients, whereas the European Medicines Agency also approves binder use in earlier stages of CKD when phosphate levels are  $\geq 1.78$  mmol/L (5.5 mg/dL). Calcium-containing products are commonly used both as phosphate binders and for calcium supplementation in all stages of CKD.

Phosphate binders are a major strategy in managing CKD-MBD and are based on sevelamer, calcium, lanthanum, or aluminum. Currently available phosphate binders are listed in Table 4. KDIGO recognizes that all phosphate binders effectively decrease serum phosphorus, although warn that the side effects, pill burden, and patient response may differ between them. As with other therapies, KDIGO suggests that phosphate binder selection consider the individual patient's needs and biochemical markers of bone and mineral metabolism.

## Treatment of Secondary Hyperparathyroidism

PTH progressively rising above the reference range in CKD stages 3–5 (not requiring dialysis) should trigger evaluation and treatment of hyperphosphatemia, hypocalcemia, and/or vitamin D deficiency (4). If addressing these modifiable causes of SHPT is unsuccessful, parathyroid-specific interventions may be undertaken.

In CKD stage 5D, KDIGO suggests that interventions for PTH levels outside of ~2–9 times the reference range or significant changes within that range be guided by patients' serum calcium and phosphate levels (4). Therapy selection depends on individual clinical and biochemical status. In light of the risks of calcification, KDIGO recognizes that relying on elevated serum calcium to suppress PTH has potential for higher risk than benefit

**Table 4.** Potential advantages and disadvantages of phosphate-binding compounds (adapted from KDIGO 2009) (4)

Binder source	Potential advantages	Potential disadvantages
Aluminum hydroxide	Very effective phosphate binding capacity; variety of forms	Potential for aluminum toxicity; altered bone mineralization, dementia; GI side effects
Calcium-containing (calcium carbonate/acetate/gluconate)	Effective phosphate binding, inexpensive	Potential for hypercalcemia-associated risks including extraskeletal calcification and PTH suppression; GI side effects
Lanthanum carbonate	Effective; no calcium	Cost; potential for accumulation of lanthanum due to GI absorption; may require calcium supplementation in the presence of symptomatic hypocalcemia; GI side effects
Sevelamer (HCl or carbonate)	Effective; no calcium/metal; not absorbed; potential for reduced coronary/aortic calcification compared with calcium-based binders	Cost; may require calcium supplementation in presence of symptomatic hypocalcemia; GI side effects

(11). Calcimimetics, which act on the calcium-sensing receptor in parathyroid and other tissues, mimic or potentiate the effects of calcium without causing hypercalcemia. Calcimimetics are also useful adjunctively with vitamin D derivatives in patients at increased risk of hypercalcemia and hyperphosphatemia.

Vitamin D treatment involves non-selective vitamin D receptor (VDR) activators (calcitriol), vitamin D prohormones (doxercalciferol and alfacalcidol), or selective VDR activators (maxacalcitol and paricalcitol). VDR activators control SHPT through VDR-mediated suppression of PTH transcription in parathyroid cells (31). Selective VDR activators suppress PTH similarly with fewer hypercalcemic and hyperphosphatemic adverse events (32).

In hypercalcemia, calcium sources should be limited and calcitriol or active vitamin D sterols should be reduced or stopped. In hypophosphatemia, phosphate binders and calcimimetics may be stopped but active vitamin D agents may be continued. When serum calcium is below the lower limit of normal or the patient has symptoms of hypocalcemia, calcimimetics should be reduced or stopped, and other medications such as vitamin D sterols can be appropriately initiated, continued or increased (2). KDIGO advises cautious dosing of active vitamin D to avoid raising serum calcium and phosphate excessively (2,11). When using active vitamin D or calcimimetics, phosphate binder doses may need adjustment to maintain serum phosphate and calcium within normal limits (4). PTH-modulating treatments should be reduced or stopped if PTH decreases to < 2 times upper normal range (4). For severely elevated PTH that resists pharmacotherapy, KDIGO suggests parathyroidectomy (4).

## Management of CKD-MBD: the role of diet and phosphate binders

Dietary management of CKD-MBD includes modification of dietary phosphate intake within the confines of meeting protein needs. Dietary modification is typically used in conjunction with phosphate binders, especially in CKD Stage 5D. Hyperphosphatemia in dialysis patients can also be treated with intensified dialytic phosphate removal (2).

The IOM guidance for phosphorus intake, which has not been revised since 1997 and does not address the specific needs of CKD patients, provides an estimated average requirement of 580 mg/day and a recommended daily allowance of 700 mg/day for all adults (33). The upper tolerable limit, 4000 mg/day, should not be approached even by those without CKD given recent learnings on the association of high phosphate intake and cardiovascular disease in the general public. Since intakes exceeding metabolic requirements directly increase serum phosphate, it is important to keep CKD patients from overly exceeding the recommended daily phosphorus allowance.

Improving awareness of foods high in phosphorus and

understanding differences in phosphorus absorption between animal, plant, and additive sources is essential for the dietitian. Despite reported high phosphorus content of many plant protein sources in food composition databases, patients (mean GFR 32 ml/min) who consumed a metabolic-lab-prepared vegetarian diet (grain- and soy-based) for 1 week had significantly lower serum phosphate and FGF-23 levels than those consuming equivalent protein and calories in a meat- and dairy-based diet (34). Thus, plant sources of protein may have lower phosphorus bioavailability than their *in vitro*-measured phosphorus content. More research is required to fully understand the variability of phosphate bioavailability and patient-specific absorption (34,35).

Dietitians are key to patient education about the dangers of bone and mineral abnormalities in CKD. Phosphorus sources are an important educational topic along with making patients aware of inadequate food labeling and hidden phosphorus sources such as processed-food additives, which are underrepresented in nutrition labels and databases (2,35,36). Significant underestimations of phosphorus content by as much as 350 mg/day have been reported in Europe, Japan and the US (37–39). Analysis of chicken products in the US showed actual phosphorus contents exceeding those estimated from a nutrient database (36). The use of additives that increase phosphorus by two-fold or more, together with a lack of information on the nutritional label, has motivated nephrology dietitians to discourage manufacturers' indiscriminate use of phosphorus additives and to call for greater transparency regarding the phosphorus content of foods (35). While dietitians have taken an active role in treating hyperphosphatemia with phosphate binders, dietary counseling becomes an even more critical intervention considering the significant contribution of food additives to the daily phosphorus load.

## Controlling calcium and vitamin D intake

While KDIGO suggests modifying calcium intake as well as the use of calcitriol or vitamin D analogs in patients with hypercalcemia, known arterial calcification, suppressed bone turnover, or persistent PTH suppression, the KDIGO guidelines do not place an absolute limit on the daily calcium intake (4).

Many naturally low-calcium foods such as orange juice, breads and cereals are industrially fortified with calcium by adding calcium phosphate. Such foods can directly affect both phosphate and calcium intake. This is another area where the dietitian can help the patient understand his or her best food choices as they relate to mineral metabolism.

In the US, publicity about age-related osteoporosis has encouraged self-administered calcium and nutritional vitamin D supplementation. The IOM recently systematically reviewed the evidence for tolerable upper limits of calcium and vitamin D intake, considering effects on hypercalcemia, hypercalciuria, vascular/soft tissue calcification, and kidney stones (27). Considerations for nutritional vitamin D limits included evidence of U-shaped epidemiology relating intake to all-cause mortality, cardiovascular disease, vascular calcification, pancreatic cancer, and musculoskeletal outcomes (falls, weakness,



# Feature Article...

**Table 5.** US Institute of Medicine (IOM) calcium and vitamin D intake recommendations as of November, 2010 and phosphorus intake recommendations as of 1997 (27,33).

Patient group	Calcium		Vitamin D		Phosphorus	
	Recommended Daily Allowance*	Upper Limit**	Recommended Daily Allowance	Upper Limit**	Recommended Daily Allowance	Upper Limit**
Adults 19–50 yr	1000 mg/day	2500 mg/day	600 IU/day	4000 IU/day	700 mg	4000 mg/d
Men 51–70 yr	1000 mg/day	2000 mg/day	600 IU/day	4000 IU/day	700 mg	4000 mg/d
Women 51–70 yr	1200 mg/day	2000 mg/day	600 IU/day	4000 IU/day	700 mg	4000 mg/d
Adults >70 yr	1200 mg/day	2000 mg/day	800 IU/day	4000 IU/day	700 mg	3000 mg/d

\*The Recommended Daily Allowance is an intake sufficient for the needs of at least 97.5% of the population.

\*\*The Upper Limit is an intake above which adverse event risk occurs. IOM states that the upper limit is “not intended as a target intake”.

fractures) (27). Updated reference intakes and upper limits for the general public were released in November 2010 and are summarized by age group in Table 5 (27).

Dietitians should observe the current IOM 2010 upper limits when recommending calcium intake for CKD patients. The IOM’s 2010 recommended daily calcium allowance (1000–1200 mg/day) is designed for adults with normal kidney function (27). The IOM specifies an upper tolerable limit of 2500 mg calcium/day for adults 19–50 years and 2000 mg/day for those  $\geq 51$  years; this upper limit is not intended as a target intake (27). These recommendations are in agreement with K/DOQI’s opinion-based suggestion on calcium intake, which states that total elemental calcium intake (including both dietary calcium intake and calcium-based phosphate binders) should not exceed 2,000 mg/day and that calcium supplementation to symptom-free hypocalcemic CKD patients should be limited (2,40,41). It is important to be aware that dialysate calcium, calcium-based phosphate binders and vitamin D analogs may contribute to a positive calcium balance. In contrast to IOM and K/DOQI, the KDIGO guidelines do not specify calcium intake levels or limits for CKD patients, citing a lack of high-quality evidence to make any specific guidelines for calcium intake, binder type, or one active vitamin D agent over another (2).

## Nutritional vitamin D

Recent attention to nutritional vitamin D deficiency has added another dimension to the management of CKD-MBD. KDIGO recognizes that vitamin D deficiency or insufficiency is common, although there is no consensus as to what defines ‘adequate’ serum calcidiol levels for the general public, much less in CKD (42). It has been suggested that a “normal” calcidiol level is the level associated with a normal serum PTH level in the general population; others define normal as the level above which there is no further reciprocal reduction in serum PTH upon vitamin D supplementation (43). KDIGO recommends repletion of CKD patients as is recommended for the general public (2). The IOM recommendation to replete

patients whose serum 25(OH) D < 20 ng/mL seems reasonable. Controversy continues regarding the IOM daily intake recommendations, and critics of the IOM favor higher daily doses of nutritional vitamin D, up to 2000 IU/day (42,44). Risk for toxicity is thought to be minimal. Nutritional vitamin D supplementation is also generally agreed to be appropriate for those with CKD and low blood calcidiol levels. Once-monthly dosing of ergocalciferol at 50,000 IU or daily dosing of cholecalciferol up to 2000 IU has been used. Further research is needed to determine the optimal dosing and serum levels for nutritional vitamin D in CKD.

## Translating KDIGO guidelines into clinical practice for the dietitian

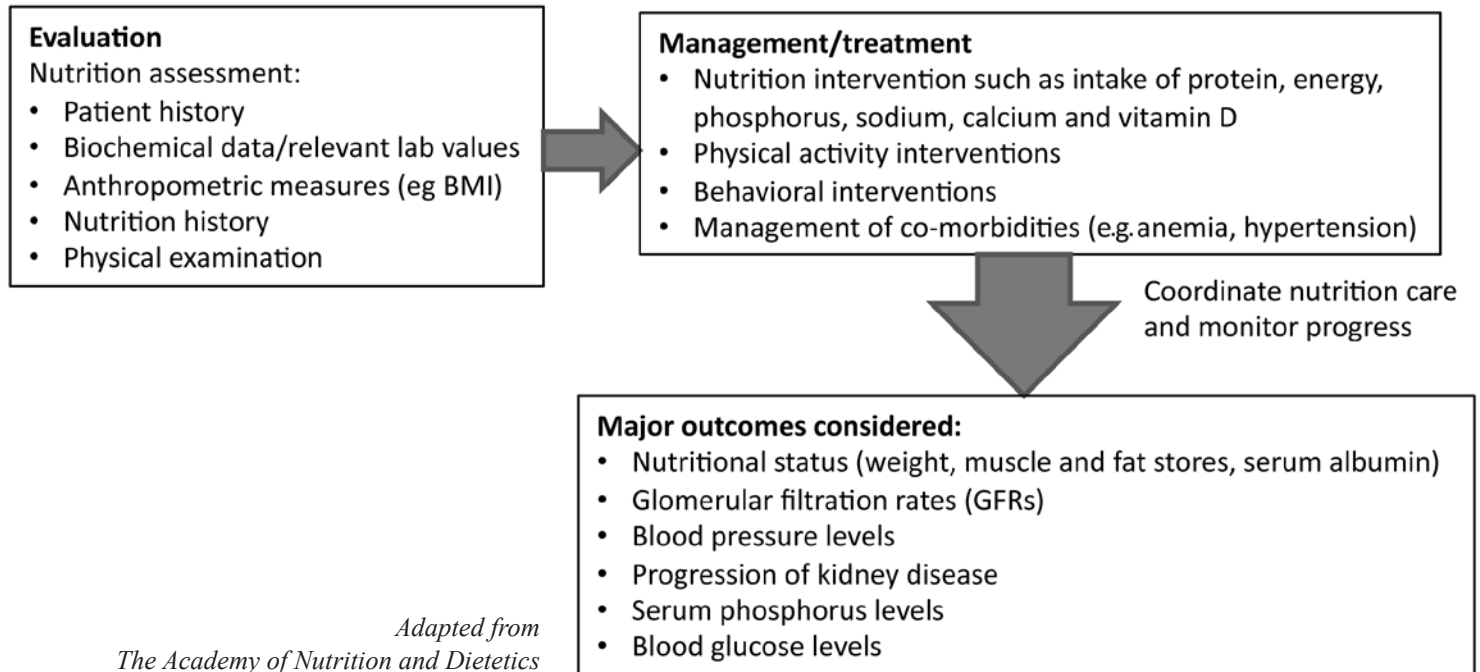
Nephrology dietitians are central to the management of CKD-MBD. They are well placed to educate patients about bone and mineral abnormalities and to help prevent and treat protein-energy malnutrition, mineral and electrolyte disorders (e.g., phosphorus, calcium, vitamin D), and minimize the impact of co-morbidities on kidney disease progression. Frequent dietetic consultations have been shown to improve near-term phosphate control (45).

The Academy of Nutrition and Dietetics recently published US guidelines on evidence-based nutrition practice in CKD. According to the Academy, dietary intervention should be initiated at least 12 months before the planned start of renal replacement therapy (46). Dietitians should monitor the nutritional status of CKD patients every one to three months, more frequently in the presence of inadequate nutrient intake, protein-energy malnutrition, or mineral and electrolyte disorders.

During the initial assessment, dietitians should evaluate CKD patients’ food- and nutrition-related history, along with various biochemical parameters related to glycemic control, protein-energy malnutrition, inflammation, kidney function, mineral and bone disorders, and electrolyte disorders (Figure 1).

# Feature Article...

**Figure 1:** CKD-MBD: Dietary Interventions and Practices Considered



In addition to macronutrient recommendations, the Academy provides suggestions for phosphate management. At each visit the dietitian should estimate dietary intake of phosphorus based on its content in natural food (those high in protein such as meat, eggs and milk) and also as an additive in processed food. The dietitian should also encourage patients to consume fresh rather than processed foods since the phosphorus in additives is more readily absorbed than naturally occurring phosphorus and contributes more to hyperphosphatemia. This is supported by a recent meta-analysis of seven randomized controlled trials in CKD patients with hyperphosphatemia, which demonstrated improved reductions in phosphorus levels for more than 4 months following intensive educational intervention aimed at avoidance of phosphate additives (47). Another strategy to avoid exacerbation of protein-energy wasting is to make use of low-phosphorus protein sources, such as egg whites, to avoid phosphorus loading while providing adequate protein (48). The dietitian should also encourage patients to consume a mixed diet instead of relying exclusively on animal proteins whose bioavailability of phosphorus is much higher. In addition the dietitian should be aware of low-phosphorus functional foods specifically designed for renal patients that may help maintain a diverse choice of foods, e.g. low-phosphorus milk (49).

In summary, dietary intake of phosphorus should be limited and dietary modification can be used in conjunction with phosphate binders, whose dose and timing should be individually adjusted according to the dietary phosphate content of meals and snacks. According to the KDIGO and K/DOQI guidelines, serum phosphate in patients with CKD stage 3–5 should be

maintained within the laboratory normal limits and reduced toward the normal range in CKD Stage 5D.

In terms of managing calcium and nutritional vitamin D intake, the dietitian should recommend a total elemental calcium intake no greater than the IOM age-adjusted recommendations, including dietary calcium, calcium supplementation and calcium-based phosphate binders. Serum calcium in patients with CKD stages 3–5D should be kept within the normal reference range for the laboratory used, with avoidance of hypercalcemic episodes in dialysis patients. The dietitian should recommend vitamin D supplementation to maintain adequate levels of vitamin D if the serum level of 25-hydroxyvitamin D is less than 30 ng/mL (75 nmol/L). Nutritional interventions during each stage of CKD are summarized in Table 6 (40).

## Impact of mineral management interventions on outcomes

Multiple studies have shown that hyperphosphatemia and calcification worsen outcomes for patients on dialysis and that interventions to normalize mineral metabolism have the potential to improve survival. In the 2009 Accelerated Mortality on Renal Replacement (ArMORR) study, the use of any phosphate binder during the first 90 days of HD was independently associated with improved 1-year survival (50).

In another study, coronary artery calcification independently predicted mortality among 127 incident HD patients randomized to receive sevelamer or calcium-based phosphate binders, and sevelamer use improved survival over calcium binder use after a median follow-up of 44 months (51). Treatments to maintain or achieve normal targets are likely to maximize clinical benefit, but since normal levels are not always achievable in CKD Stage 5D, control of serum phosphorus

**Table 6.** Nutritional interventions in chronic kidney disease (adapted from the K/DOQI guidelines) (40)

Stage	Description	GFR (ml/min/1.73m <sup>2</sup> )	Possible nutritional intervention
1	Kidney damage with normal or elevated GFR	> 90	<ul style="list-style-type: none"> <li>• Therapeutic lifestyle changes and reduced intake in saturated fat and sodium to slow risk of CVD</li> <li>• Adequate protein to prevent malnutrition and delay progression</li> </ul>
2	Kidney damage with mild decrease in GFR	60–89	<ul style="list-style-type: none"> <li>• As above</li> </ul>
3	Moderate decrease in GFR	30–59	<ul style="list-style-type: none"> <li>• Continue as above</li> <li>• As intact PTH or phosphorus increases, maintain controlled amount of phosphorous in the diet</li> <li>• Check vitamin D levels</li> <li>• Iron-rich foods adjunctive to erythropoietin therapy</li> </ul>
4	Severe decrease in GFR	15–29	<ul style="list-style-type: none"> <li>• Dietary phosphorus restricted to 800 mg to 1000 mg/day, adjusting for protein intake</li> <li>• Protein &gt; 0.6 g/kg of body weight or as needed to maintain protein status</li> </ul>
5	Kidney failure	< 15	<ul style="list-style-type: none"> <li>• When dialysis is started, protein is increased</li> <li>• Potassium, phosphorus, sodium and fluids may need to be limited or adjusted</li> </ul>

“towards” the normal range is emphasized (30). Once again, treatment of bone and mineral abnormalities may be affected by financial considerations when phosphate binders and calcimimetics are included in the bundle. Within the clinical care team, especially in the area of bone and mineral metabolism, dietitians become advocates for individualized, evidence-based patient care to promote optimal outcomes.

## Conclusions

KDIGO has delineated a complex of metabolic anomalies (CKD-MBD) that develop as CKD progresses, including dysregulation of phosphate, calcium, vitamin D metabolism, and PTH synthesis and secretion. Multiple therapies are available to prevent or treat these abnormalities; many of these therapies are included in physician-approved, dietitian-driven protocols. Dietary modification, phosphate binder therapy, nutritional vitamin D repletion, active vitamin D administration, and calcimimetic therapy are employed, often in combination, to address abnormal bone and mineral metabolism. Clinical practice guidelines provide evidence-based recommendations for clinicians. KDIGO guidelines expand the upper and lower limits of PTH from those cited by K/DOQI 2003, but suggest that changes in PTH, even within those expanded limits, should prompt evaluation and action. KDIGO continues the focus on phosphate control and avoidance of hypercalcemia. KDIGO’s recommendations for monitoring and management of mineral metabolism have the potential to improve patient outcomes by helping to reduce vascular calcification, hyperparathyroidism, cardiovascular disease, and mortality.

Nephrology dietitians are well placed to implement KDIGO mineral management recommendations through patient education,

dietary modification, and per-protocol dosing of active vitamin D agents, calcimimetics, and phosphate binders. Dietitians can also foster treatment adherence and adequacy of dialysis, which are important for phosphorus control. Nephrology dietitians have more recently been involved with repletion of nutritional vitamin D, advocacy against the food industry’s use of phosphate food additives and the promotion of kidney-friendly foods that are low in phosphorus. Each of these efforts has the potential to help minimize bone and mineral abnormalities and to improve patient outcomes, but MIPPA-related changes will force all clinicians to evaluate and manage resources appropriately. Dietitians, as part of the healthcare team, have an opportunity to provide leadership in the area of bone and mineral management by applying KDIGO guidelines and advocating for individual patients’ needs in accordance with the totality of current evidence.

## Acknowledgments

The author determined the article’s conception and design, contributed multiple rounds of substantive scientific revisions, approved the final version for publication, and acknowledges the writing assistance of Kim Coleman Healy, PhD, CMPP, and Stuart Murray, BSc MSc, of Envision Scientific Solutions; writing assistance was supported by Sanofi.

## References

1. Iglehart JK. Bundled payment for ESRD—including ESAs in Medicare’s dialysis package. *N Engl J Med*. 2011;364(7):593-595.

# Feature Article...

2. Uhlig K, Berns JS, Kestenbaum B, et al. K/DOQI US commentary on the 2009 KDIGO Clinical Practice Guideline for the Diagnosis, Evaluation, and Treatment of CKD-Mineral and Bone Disorder (CKD-MBD). *Am J Kidney Dis*. 2010;55(5):773-799.
3. US Centers for Medicare and Medicaid Services. Part II. Department of Health and Human Services. Centers for Medicare and Medicaid Services. 42 CFR Parts 410, 413 and 414. Medicare Program; End-Stage Renal Disease Prospective Payment System; Final Rule and Proposed Rule. *Federal Register*. 2010;75(155):49029-49214.
4. KDIGO clinical practice guideline for the diagnosis, evaluation, prevention, and treatment of Chronic Kidney Disease-Mineral and Bone Disorder (CKD-MBD). *Kidney Int Suppl*. 2009(113):S1-130.
5. Uhlig K, Macleod A, Craig J, et al. Grading evidence and recommendations for clinical practice guidelines in nephrology. A position statement from Kidney Disease: Improving Global Outcomes (KDIGO). *Kidney Int*. 2006;70(12):2058-2065.
6. Atkins D, Best D, Briss PA, et al. Grading quality of evidence and strength of recommendations. *BMJ*. 2004;328(7454):1490.
7. Moe S, Drueke T, Cunningham J, et al. Definition, evaluation, and classification of renal osteodystrophy: a position statement from Kidney Disease: Improving Global Outcomes (KDIGO). *Kidney Int*. 2006;69(11):1945-1953.
8. Juppner H. Phosphate and FGF-23. *Kidney Int*. 2011;79(Suppl 121):S24-S27.
9. Isakova T, Gutierrez OM, Wolf M. A blueprint for randomized trials targeting phosphorus metabolism in chronic kidney disease. *Kidney Int*. 2009;76(7):705-716.
10. Isakova T, Xie H, Yang W, et al. Fibroblast growth factor 23 and risks of mortality and end-stage renal disease in patients with chronic kidney disease. *JAMA*. 2011;305(23):2432-2439.
11. Moe SM, Drueke T. Improving global outcomes in mineral and bone disorders. *Clin J Am Soc Nephrol*. 2008;3 Suppl 3:S127-130.
12. Gauci C, Moranne O, Fouqueray B, et al. Pitfalls of measuring total blood calcium in patients with CKD. *J Am Soc Nephrol*. 2008;19(8):1592-1598.
13. Souberbielle JC, Cavalier E, Jean G. Interpretation of serum parathyroid hormone concentrations in dialysis patients: what do the KDIGO guidelines change for the clinical laboratory? *Clin Chem Lab Med*. 2010;48(6):769-774.
14. Boudou P, Ibrahim F, Cormier C, Chabas A, Sarfati E, Souberbielle JC. Third- or second-generation parathyroid hormone assays: a remaining debate in the diagnosis of primary hyperparathyroidism. *J Clin Endocrinol Metab*. 2005;90(12):6370-6372.
15. Ljungdahl N, Haarhaus M, Linder C, Magnusson P. Comparison of 3 third-generation assays for bio-intact parathyroid hormone. *Clin Chem*. 2006;52(5):903-904.
16. el-Hajj Fuleihan G, Klerman EB, Brown EN, Choe Y, Brown EM, Czeisler CA. The parathyroid hormone circadian rhythm is truly endogenous--a general clinical research center study. *J Clin Endocrinol Metab*. 1997;82(1):281-286.
17. Maeda SS, Kunii IS, Hayashi LF, Lazaretti-Castro M. Increases in summer serum 25-hydroxyvitamin D (25OHD) concentrations in elderly subjects in Sao Paulo, Brazil vary with age, gender and ethnicity. *BMC Endocr Disord*. 2010;10:12.
18. Kestenbaum B, Sampson JN, Rudser KD, et al. Serum phosphate levels and mortality risk among people with chronic kidney disease. *J Am Soc Nephrol*. 2005;16(2):520-528.
19. Noordzij M, Korevaar JC, Boeschoten EW, Dekker FW, Bos WJ, Krediet RT. The Kidney Disease Outcomes Quality Initiative (K/DOQI) Guideline for Bone Metabolism and Disease in CKD: association with mortality in dialysis patients. *Am J Kidney Dis*. 2005;46(5):925-932.
20. Block GA, Hulbert-Shearon TE, Levin NW, Port FK. Association of serum phosphorus and calcium x phosphate product with mortality risk in chronic hemodialysis patients: a national study. *Am J Kidney Dis*. 1998;31(4):607-617.
21. Block GA, Klassen PS, Lazarus JM, Ofsthun N, Lowrie EG, Chertow GM. Mineral metabolism, mortality, and morbidity in maintenance hemodialysis. *J Am Soc Nephrol*. 2004;15(8):2208-2218.
22. Kalantar-Zadeh K, Kuwae N, Regidor DL, et al. Survival predictability of time-varying indicators of bone disease in maintenance hemodialysis patients. *Kidney Int*. 2006;70(4):771-780.
23. Kimata N, Albert JM, Akiba T, et al. Association of mineral metabolism factors with all-cause and cardiovascular mortality in hemodialysis patients: the Japan dialysis outcomes and practice patterns study. *Hemodial Int*. 2007;11(3):340-348.
24. Tentori F, Blayney MJ, Albert JM, et al. Mortality risk for dialysis patients with different levels of serum calcium, phosphorus, and PTH: the Dialysis Outcomes and Practice Patterns Study (DOPPS). *Am J Kidney Dis*. 2008;52(3):519-530.
25. Barreto FC, Barreto DV, Moyses RM, et al. K/DOQI-recommended intact PTH levels do not prevent low-turnover bone disease in hemodialysis patients. *Kidney Int*. 2008;73(6):771-777.
26. Kanis JA. FRAX(R) WHO Fracture Risk Assessment Tool. <http://www.shef.ac.uk/FRAX/index.htm>. Accessed March 3, 2008.
27. Ross AC, Manson JE, Abrams SA, et al. The 2011 report on dietary reference intakes for calcium and vitamin D from the Institute of Medicine: what clinicians need to know. *J Clin Endocrinol Metab*. 2011;96(1):53-58.
28. Martin CJ, Reams SM. The renal dietitian's role in managing hyperphosphatemia and secondary hyperparathyroidism in dialysis patients: a national survey. *J Ren Nutr*. 2003;13(2):133-136.



# Feature Article...

29. Martin KJ, Gonzalez EA. Prevention and control of phosphate retention/hyperphosphatemia in CKD-MBD: what is normal, when to start, and how to treat? *Clin J Am Soc Nephrol*. 2011;6(2):440-446.
30. Bhan I, Dubey A, Wolf M. Diagnosis and management of mineral metabolism in CKD. *J Gen Intern Med*. 2010;25(7):710-716.
31. Dusso AS, Brown AJ, Slatopolsky E. Vitamin D. *Am J Physiol Renal Physiol*. 2005;289(1):F8-28.
32. Brancaccio D, Bommer J, Coyne D. Vitamin D receptor activator selectivity in the treatment of secondary hyperparathyroidism: understanding the differences among therapies. *Drugs*. 2007;67(14):1981-1998.
33. US National Academy of Sciences. Institute of Medicine. Food and Nutrition Board. Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride. 1997. [http://www.nal.usda.gov/fnic/DRI/DRI\\_Calcium/146-189.pdf](http://www.nal.usda.gov/fnic/DRI/DRI_Calcium/146-189.pdf). Accessed February 22, 2012.
34. 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*. 2011;6(2):257-264.
35. Uribarri J. Phosphorus homeostasis in normal health and in chronic kidney disease patients with special emphasis on dietary phosphorus intake. *Semin Dial*. 2007;20(4):295-301.
36. Sullivan CM, Leon JB, Sehgal AR. Phosphorus-containing food additives and the accuracy of nutrient databases: Implications for renal patients. *J Ren Nutr*. 2007;17(5):350-354.
37. Oenning LL, Vogel J, Calvo MS. Accuracy of methods estimating calcium and phosphorus intake in daily diets. *J Am Diet Assoc*. 1988;88(9):1076-1080.
38. Zhang ZW, Shimbo S, Miyake K, et al. Estimates of mineral intakes using food composition tables vs measures by inductively-coupled plasma mass spectrometry: Part 1. calcium, phosphorus and iron. *Eur J Clin Nutr*. 1999;53(3):226-232.
39. Moreno-Torres R, Ruiz-Lopez MD, Artacho R, et al. Dietary intake of calcium, magnesium and phosphorus in an elderly population using duplicate diet sampling vs food composition tables. *J Nutr Health Aging*. 2001;5(4):253-255.
40. K/DOQI clinical practice guidelines for bone metabolism and disease in chronic kidney disease. *Am J Kidney Dis*. 2003;42(4 Suppl 3):S1-201.
41. McCann L. Calcium in chronic kidney disease: recommended intake and serum targets. *Adv Chronic Kidney Dis*. 2007;14(1):75-78.
42. Heaney RP, Holick MF. Why the IOM recommendations for vitamin D are deficient. *J Bone Miner Res*. 2011;26(3):455-457.
43. Steingrimsdottir L, Gunnarsson O, Indridason OS, Franzson L, Sigurdsson G. Relationship between serum parathyroid hormone levels, vitamin D sufficiency, and calcium intake. *JAMA*. 2005;294(18):2336-2341.
44. Reid IR, Avenell A. Evidence-based policy on dietary calcium and vitamin D. *J Bone Miner Res*. 2011;26(3):452-454.
45. Morey B, Walker R, Davenport A. More dietetic time, better outcome? A randomized prospective study investigating the effect of more dietetic time on phosphate control in end-stage kidney failure haemodialysis patients. *Nephron Clin Pract*. 2008;109(3):c173-180.
46. American Dietetic Association Academy of Nutrition and Dietetics. Chronic kidney disease evidence-based nutrition guideline. June 2010. <http://www.adaevidencelibrary.com/topic.cfm?cat=3927&auth=1>. Accessed February 14, 2012.
47. Caldeira D, Amaral T, David C, Sampaio C. Educational strategies to reduce serum phosphorus in hyperphosphatemic patients with chronic kidney disease: systematic review with meta-analysis. *J Ren Nutr*. 2011;21(4):285-294.
48. Taylor LM, Kalantar-Zadeh K, Markewich T, et al. Dietary egg whites for phosphorus control in maintenance haemodialysis patients: a pilot study. *J Ren Care*. 2011;37(1):16-24.
49. Oliverio S, Atcher L. Regular use of low-phosphorus milk significantly improves dietary satisfaction of patients without changing their serum phosphorus. *Dialysis & Transplantation*. 2006;35(4):215-219.
50. Isakova T, Gutiérrez OM, Chang Y, et al. Phosphorus binders and survival on hemodialysis. *J Am Soc Nephrol*. 2009;20(2):388-396.
51. Block GA, Raggi P, Bellasi A, Kooienga L, Spiegel DM. Mortality effect of coronary calcification and phosphate binder choice in incident hemodialysis patients. *Kidney Int*. 2007;71(5):438-441.

## Outstanding Service Award Applications Due by April 30th!

RPG offers one Outstanding Service Award (OSA) per year to a Renal Dietitian RPG member who has demonstrated leadership, promoted the dietitians' role with CKD, and has shown initiative and dedication to renal patients.

This prestigious award includes a sponsorship to the Academy's Food and Nutrition Conference and Expo (FNCE) including FNCE registration with 2 night's lodging/3 day's meals and transportation. The recipient will also be featured at a RPG sponsored event. All applications are due by April 30th.

Applications can be found at:  
[http://renalnutrition.org/members\\_only/awards.php](http://renalnutrition.org/members_only/awards.php)

## Protein-Energy Wasting in CKD Patients: Are We Wasting an Opportunity to Improve Outcomes?

**Abby C. Sauer, MPH, RD, LD**

Research Scientist, Abbott Nutrition  
Columbus, Ohio  
abby.sauer@abbott.com

**Anne Coble Voss, PhD, RD, LD**

Associate Research Fellow, Abbott Nutrition  
Columbus, Ohio  
anne.voss@abbott.com

**This article has been approved for 1 CPE units. The online CPEU quiz and certificate of completion can be accessed in the Members Only section of the RPG web site via the My CPEU link. This CPE offering is available to current RPG members only and the expiration date is April 15, 2013.**

For the general public, overnutrition and obesity are a major public health challenge. In contrast, for individuals with chronic kidney disease (CKD), especially those on dialysis, undernutrition (as defined as deficient energy or protein intake) and protein-energy wasting (PEW) (as defined as a loss of body protein and fuel reserves) is a primary concern (1,2). Research has shown that approximately 45% of pre-dialysis patients and up to 75% of dialysis patients experience undernutrition and/or PEW (2,3). Research has consistently shown that PEW is the strongest risk factor for adverse outcomes and increased mortality in dialysis patients (1). Evidence indicates that surrogates of PEW, such as low serum albumin level or inadequate protein intake, correlate with mortality (1). Approximately two-thirds of all patients on dialysis in the US exhibit hypoalbuminemia with a serum albumin level < 4 mg/dl (1). The pathogenesis of PEW in patients with CKD is complex and multifactorial. The primary causes include poor dietary intake, abnormal lipid and carbohydrate metabolism, amino acid imbalances, abnormal hormonal response, losses of nutrients, uremic toxicity and catabolism (4). Although there are numerous causes of PEW, decreased nutrient intake is often the most frequent cause (2,5). Reasons for poor nutrient intake include anorexia, poor food choice behavior, lack of knowledge of nutrient goals, skipping meals, nutrient losses during dialysis treatment, the dialysis procedure, concurrent illness, hypercatabolism, and inflammation (5). Inadequate intake is also caused by comorbid physical illnesses affecting gastrointestinal function, depression, other psychiatric illness, or socioeconomic factors (5). Many patients are not meeting their recommended energy and protein needs through their dietary intake. The recommended dietary energy intake (DEI) for

patients on hemodialysis and peritoneal dialysis is 30-35 kcal/kg ideal body weight per day, while the recommended dietary protein intake (DPI) is 1.2 g/kg per day for hemodialysis patients and 1.3 g/kg per day for peritoneal dialysis patients (5-7). Many dialysis patients have a lower DEI and DPI than the recommendations. Burrowes et al showed that for dialysis patients, the average DEI is 23.2±9.5 kcal/kg per day and the average DPI is 0.96±0.43 g/kg per day, clearly demonstrating inadequate protein-energy intake (8).

With all the challenges facing dialysis patients with regards to their dietary intake, and with the high prevalence of undernutrition and PEW, nutrition support is often needed. Nutrition support is a viable option to help patients reach the current recommendations for energy and protein intake, and subsequently improve their nutritional status and related clinical outcomes. Numerous studies have examined the effects of various forms of nutrition support including oral nutritional supplementation, intradialytic nutrition and in-center meals. The research has shown positive outcomes, including improvements in body weight, serum protein levels, nutritional status, and dietary energy and protein intake (1, 9).

The use of oral nutritional supplements (ONS), particularly renal-specific ONS, is supported by multiple research studies, expert guidelines, and a recent opinion paper by expert nephrologists in nutrition (1, 6-9).

A recent systematic review and meta-analysis of 18 studies (5 randomized controlled trials and 13 non-randomized controlled trials) on the use of multi-nutrient oral supplements and tube feeding in maintenance hemodialysis patients showed that oral and enteral nutritional support improves protein and energy intake, increases serum albumin concentrations by 0.23 g/dL ( $p<0.05$ ), and improves total energy intake (9). The analysis further showed that enteral support has an insignificant effect on electrolyte status, particularly serum phosphate and potassium, and may even improve clinical outcomes, especially in malnourished patients (9). Caglar et al showed in 2005 that a renal-specific ONS provided three times a week during dialysis treatments for a period of 6 months to malnourished subjects resulted in significant improvements in serum albumin, serum prealbumin, and subjective global assessment (SGA) scores (10). Additionally, a 2009 study by Williams showed that renal-specific ONS are preferred over standard ONS by hemodialysis patients due to the lower fluid content and decreased phosphate binder requirements (11). A recent 2011 study compared an intradialytic renal-specific ONS with and without resistance exercise in chronic hemodialysis patients for 6 months (11). Interestingly, the results of this study did not show any further benefits of resistance exercise on long-term protein accretion above and beyond the intradialytic renal-specific ONS alone (12).

Expert groups also have issued recommendations for nutrition support in dialysis patients, particularly in malnourished patients. Table 1 illustrates these recommendations, which emphasize that nutritional support should be provided to patients who cannot meet their nutritional needs through diet. Additionally, they recommend that when ONS is used, specialized formulas for dialysis patients are the best choice (5-7).

**Table 1: Expert Recommendations:**

	National Kidney Foundation Kidney Disease Outcomes Quality Initiative (K/DOQI) (6)	European Society for Clinical Nutrition and Metabolism (ESPEN) (7)	European Best Practice Guidelines (EBPG) (5)
Nutrition Support	Individuals undergoing maintenance dialysis who are unable to meet their protein and energy requirements with food intake for an extended period of time should receive nutrition support.	Special formula products for HD treatment can be useful especially in malnourished patients who are not able to increase their nutrient intake.	Oral nutritional supplements should be prescribed if nutritional counseling does not achieve an increase in nutrient intake to a level that covers minimum recommendations. Products specifically designed for dialysis patients should be prescribed.

Recently, Kalantar-Zadeh et al published a review paper on the importance of diet and nutrition support in improving outcomes in patients with CKD (1). They concluded that nutrition intervention can increase serum albumin levels or correct PEW in patients with CKD, and subsequently improve quality of life and survival (1). Based on the data from clinical research on nutrition intervention in CKD patients, these experts proposed an algorithm for nutrition support in patients with CKD to help improve nutritional status, dietary energy and protein intake, and serum albumin levels. The algorithm suggests that for dialysis and non-dialysis patients who are at nutritional risk, a renal-specific ONS should be started and given 1-2 times per day (1). These experts believe that providing renal-specific ONS is an effective and inexpensive strategy to improve nutritional status and outcomes in these patients (1). Renal-specific ONS can provide an additional 7-10 kcal/kg per day of energy and 0.3-0.4 g/kg per day of protein, which helps make it possible to meet the recommended targets of both DEI and DPI (1).

Undernutrition and PEW remain a significant problem for patients with CKD, especially dialysis patients. There is a consistent and strong association between nutritional status, serum albumin level and mortality. Research, along with expert opinion, supports the use of nutritional support in these patients, particularly the use of renal-specific ONS. The provision of ONS is an inexpensive and effective strategy to help improve nutritional status, and ultimately quality of life and outcomes in the CKD patient population. For those who work in the area of renal nutrition, it is imperative that recent

research and recommendations are utilized to guide practice, to help find patients at nutritional risk, intervene with the appropriate nutritional therapy, and continuously monitor patients' progress to ultimately improve outcomes.

## References

1. Kalantar-Zadeh K, Cano NJ, Budde K, et al. Diets and enteral supplements for improving outcomes in chronic kidney disease. *Nat Rev Nephrol*. 2011; 7 (7): 369-384.
2. 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*. 2008; 73 (4): 391-398.
3. Kovesdy CP, George SM, Anderson JE, Kalantar-Zadeh K. Outcome predictability of biomarkers of protein-energy wasting and inflammation in moderate and advanced chronic kidney disease. *Am J Clin Nutr*. 2009; 9 (2): 407-414.
4. Toigo G, Aparicio M, Attman PO, et al. Expert Working Group report on nutrition in adult patients with renal insufficiency (part 1 of 2). *Clin Nutr*. 2000; 19 (3): 197-207.
5. Fouque D, Vennegoor M, Wee PT, et al. EBPG Guideline on Nutrition. *Nephrol Dial Transplant*. 2007; 22(Suppl 2): ii45-ii87.
6. Clinical practice guidelines for nutrition in chronic renal failure. K/DOQI, National Kidney Foundation. *Am J Kidney Dis*. 2000; 35(Suppl 2): S1-S140.
7. Cano N, Fiaccadori E, Tesinsky P, et al. ESPEN Guidelines on Enteral Nutrition: Adult Renal Failure. *Clin Nutr*. 2006; 25 (2): 295-310.
8. Burrowes JD, Larive B, Cockram DB, et al. Effects of dietary intake, appetite, and eating habits on dialysis and non-dialysis treatment days in hemodialysis patients: cross-sectional results from the HEMO study. *J Ren Nutr*. 2003; 13 (3): 191-198.
9. 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*. 2005; 46 (3): 387-405.
10. Caglar K, Fedje L, Dimmitt R, Hakim RM, Shyr Y, Ikizler TA. Therapeutic effects of oral nutritional supplementation during dialysis. *Kidney Int*. 2002; 62 (3): 1054-1059.
11. Williams RF, Summers AM. Do hemodialysis patients prefer renal-specific or standard oral nutritional supplements? *J Ren Nutr*. 2009; 19 (2): 183-188.
12. Dong J, Sundell MB, Pupim LB, Pingsheng W, Shintani A, Ikizler TA. The effect of resistance exercise to augment long-term benefits of intradialytic oral nutritional supplementation in chronic hemodialysis patients. *J Ren Nutr*. 2011; 21 (2): 149-159.



# 2010 Dietary Guidelines and My Plate

## 2010 Dietary Guidelines for Americans and MyPlate

Linda S. Eck Mills, MBA, RD, LDN, FADA

*Reprinted from Connections Newsletter (Summer 2011, Vol. 36, Issue 1) of the Dietetics in Health Care Communities, a practice group of the Academy of Nutrition and Dietetics, used with permission.*

The *Dietary Guidelines* recommendations traditionally have been intended for healthy Americans ages two years and older. The 7th edition of *Dietary Guidelines for Americans* places stronger emphasis on reducing calorie consumption and increasing physical activity. This emphasis is because more than one-third of children and more than two-thirds of adults in the United States are overweight or obese, the *2010 Dietary Guidelines for Americans* focus on balancing calories with physical activity, and encourage Americans to consume more healthy foods like vegetables, fruits, whole grains, fat-free and low-fat dairy products, and seafood, and to consume less sodium, saturated and trans fats, added sugars, and refined grains.

Poor diet and physical inactivity are the most important factors contributing to an epidemic of overweight and obesity affecting men, women, and children in all segments of our society. Even in the absence of overweight, poor diet and physical inactivity are associated with major causes of morbidity and mortality in the United States. Therefore, the *Dietary Guidelines for Americans, 2010* is intended for Americans ages 2 years and older, including those at increased risk of chronic disease.

The *2010 Dietary Guidelines for Americans* include 23 Key Recommendations for the general population and six additional Key Recommendations for specific population groups, such as women who are pregnant. (See Figures 1, 2) Key recommendations are the most important messages within the *Guidelines* in terms of their implications for improving public health. The recommendations are intended as an integrated set of advice to achieve an overall healthy eating pattern. To get the full benefit, all Americans should carry out the Dietary Guidelines recommendations in their entirety.

### What is new in the 2010 Dietary Guidelines for Americans?

Two new chapters were included: "The Total Diet" considers various health-promoting dietary patterns and "Translating and Integrating the Evidence" addresses the broader environmental and social change needed to support healthy eating. A flexible approach to a total diet was encouraged, incorporating individual tastes and food preferences into the report's individual recommendations for a dietary pattern that is acceptable without exceeding calorie needs.

#### In addition:

- \* Eating *behaviors* were addressed (e.g., breakfast, snacking, fast food) and the association of screen time with increased body weight was assessed.
- \* Recommendations to increase consumption of plant foods (vegetables, cooked dry beans and peas, fruits, whole grains and nuts and seeds) were included.
- \* Seafood consumption of 8 oz (two servings) per week was encouraged. The Report noted that the benefits of consuming seafood far outweigh the risks, even for pregnant women.

### What is different in the 2010 Dietary Guidelines for Americans from the 2005 Dietary Guidelines for Americans?

The overarching differences include emphases on managing body weight through all life stages and on proper nutrition for children throughout. Also, research on eating patterns is incorporated for the first time, and the eating patterns presented now include vegetarian adaptations. A chapter acknowledges the influence of the broader food and physical activity environment on Americans' daily food, beverage and physical activity choices. This section calls for improvements to the environment via systematic and coordinated efforts among all sectors of influence.

#### Additional differences include:

- \* The 2010 Key Recommendations for food group intake are directional rather than providing the precise quantitative amounts that should be consumed, as were included as examples in 2005. Although the 2010 Key Recommendations do not specify quantities, an entire chapter and several appendices discuss eating patterns that include specific quantities.
- \* Specific foods that should be limited because they are substantial sources of sodium, saturated fat, cholesterol, trans fat, and added sugars are identified.
  - Reduce daily sodium intake to less than 2,300 mg and further reduce intake to 1,500 mg among persons who are 51 and older and those of any age who are African American or have hypertension, diabetes, or chronic kidney disease. The 1500 mg recommendation applies to about half of the U.S. population, including children, and the majority of adults.
  - There is a focus on nutrients of public health concern, (potassium, dietary fiber, calcium, and vitamin D), rather than on nutrients with intakes below recommended levels.
  - A new appendix table includes key consumer behaviors and potential strategies for professionals to use in implementing the *Dietary Guidelines*.
  - New guidance for alcohol consumption by breastfeeding women is included.

The two major themes of balancing calories to manage body weight and focusing on nutrient-dense foods and beverages are included in the Selected Messages for Consumers. (See Figure 3) Balancing calories



# 2010 Dietary Guidelines and My Plate

to manage body weight includes the concepts of controlling total calorie intake to manage body weight, increasing physical activity, and avoiding inactivity. Focusing on nutrient-dense foods and beverages includes the concepts of eating vegetables, fruits, whole grains, fat-free or low-fat dairy products, and seafood more often, and eating foods and beverages high in solid fats (major sources of saturated and trans fats), and added sugars less often, and reducing sodium intake.

More consumer-friendly advice and tools, including a next generation Food Pyramid are still to be released by USDA and HHS. Below is a preview of some of the tips that will be provided to help consumers translate the Dietary Guidelines into their everyday lives:

- Enjoy your food, but eat less.
- Avoid oversized portions.
- Make half your plate fruits and vegetables.
- Switch to fat-free or low-fat (1%) milk.
- Compare sodium in foods like soup, bread, and frozen meals – and choose the foods with lower numbers.
- Drink water instead of sugary drinks.

The *Dietary Guidelines* form the basis of nutrition education programs, Federal nutrition assistance programs such as school meals programs and Meals on Wheels programs for seniors, and dietary advice provided by health professionals.

Additional information on the Dietary Guidelines will be available for professionals at [www.dietaryguidelines.gov](http://www.dietaryguidelines.gov) and [www.health.gov/dietaryguidelines](http://www.health.gov/dietaryguidelines). Consumer information will be available at [www.choosemyplate.gov](http://www.choosemyplate.gov) and at [www.healthfinder.gov/prevention](http://www.healthfinder.gov/prevention).

## Balancing calories to manage weight

- Prevent and/or reduce overweight and obesity through improved eating and physical activity behaviors.
- Control total calorie intake to manage body weight. For people who are overweight or obese, this will mean consuming fewer calories from foods and beverages.
- Increase physical activity and reduce time spent in sedentary behaviors.
- Maintain appropriate calorie balance during each stage of life—childhood, adolescence, adulthood, pregnancy and breastfeeding, and older age.

## Foods and food components to reduce

- Reduce daily sodium intake to less than 2,300 milligrams (mg) and further reduce intake to 1,500 mg among persons who are 51 and older and those of any age who are African American or have hypertension, diabetes, or chronic kidney disease. The 1,500 mg recommendation applies to about half of the U.S. population, including children, and the majority of adults.
- Consume less than 10 percent of calories from saturated fatty acids by replacing them with monounsaturated and polyunsaturated fatty acids.
- Consume less than 300 mg per day of dietary cholesterol.

- Keep trans fatty acid consumption as low as possible by limiting foods that contain synthetic sources of trans fats, such as partially hydrogenated oils, and by limiting other solid fats.
- Reduce the intake of calories from solid fats and added sugars.
- Limit the consumption of foods that contain refined grains, especially refined grain foods that contain solid fats, added sugars, and sodium.
- If alcohol is consumed, it should be consumed in moderation—up to one drink per day for women and two drinks per day for men—and only by adults of legal drinking age.

## Foods and nutrients to increase

Individuals should meet the following recommendations as part of a healthy eating pattern while staying within their calorie needs.

- Increase vegetable and fruit intake.
- Eat a variety of vegetables, especially dark-green and red and orange vegetables and beans and peas.
- Consume at least half of all grains as whole grains. Increase whole-grain intake by replacing refined grains with whole grains.
- Increase intake of fat-free or low-fat milk and milk products, such as milk, yogurt, cheese, or fortified soy beverages.
- Choose a variety of protein foods, which include seafood, lean meat and poultry, eggs, beans and peas, soy products, and unsalted nuts and seeds.
- Increase the amount and variety of seafood consumed by choosing seafood in place of some meat and poultry.
- Replace protein foods that are higher in solid fats with choices that are lower in solid fats and calories and/or are sources of oils.
- Use oils to replace solid fats where possible.
- Choose foods that provide more potassium, dietary fiber, calcium, and vitamin D, which are nutrients of concern in American diets. These foods include vegetables, fruits, whole grains, and milk and milk products.

## Recommendations for specific population groups

### *Women capable of becoming pregnant*

- Choose foods that supply heme iron, which is more readily absorbed by the body, additional iron sources, and enhancers of iron absorption such as vitamin C-rich foods.
- Consume 400 micrograms (mcg) per day of synthetic folic acid (from fortified foods and/or supplements) in addition to food forms of folate from a varied diet.

### *Women who are pregnant or breastfeeding*

- Consume 8 to 12 ounces of seafood per week from a variety of seafood types.
- Due to their high methyl mercury content, limit white (albacore) tuna to 6 ounces per week and do not eat the following four types of fish: tilefish, shark, swordfish, and king mackerel.
- If pregnant, take an iron supplement, as recommended by an obstetrician or other health care provider.

# 2010 Dietary Guidelines and My Plate

## Individuals ages 50 years and older

- Consume foods fortified with vitamin B12, such as fortified cereals, or dietary supplements.

## Building healthy eating patterns

- Select an eating pattern that meets nutrient needs over time at an appropriate calorie level.
- Account for all foods and beverages consumed and assess how they fit within a total healthy eating pattern.
- Follow food safety recommendations when preparing and eating foods to reduce the risk of foodborne illnesses.



U.S. Department  
of Agriculture

**MyPlate** - the new icon released June 2nd by the US Department of Agriculture will replace the complex MyPyramid Food Guidance System that was introduced in 2005. MyPlate was designed to remind Americans to eat healthy and illustrates the five food groups – fruit, vegetable, grains, protein, and dairy foods - using a familiar

mealtime visual, a place setting. The United States joins countries such as Great Britain, Denmark, Spain, Belgium, and Norway to use a plate/wheel/pie/compass food guide approach.

The new icon shows a dinner plate divided into four slightly different sized quadrants, with fruits and vegetables taking up half the plate. Grains and protein make up the other half of the plate. Vegetables and grains are the largest portions of the four groups. There is also a circle to the side representing dairy. They form the basis of the federal government's nutrition education programs, federal nutrition assistance programs, and dietary advice provided by health and nutrition professionals.

"This is (icon) a quick, simple reminder for all of us to be more mindful of the foods that we're eating and as a mom, I can already tell you how much this is going to help parents across the country," said First Lady Michelle Obama during the unveiling of MyPlate. "When mom or dad comes home from a long day of work, we're already asked to be a chef, a referee, a cleaning crew. So it's tough to be a nutritionist, too. But we do have time to take a look at our kid's plates. As long as they're half full of fruits and vegetables, and paired with lean proteins, whole grains and low-fat dairy, we're golden. That's how easy it is."

In addition to the new icon consumer materials have been released. *Let's Eat for the Health of It* is a consumer brochure that highlights themes from the Guidelines such as Balancing Calories, Foods to Reduce, and Food to Increase. The 10 Tips Nutrition Education Series provides consumers and professionals with easy-to-follow tips in a convenient format. There are fourteen topics released so far.

1. choose MyPlate
2. add more vegetables to your day
3. focus on fruits

4. make half your grains whole
5. got your dairy today?
6. with protein foods, variety is key
7. build a healthy meal
8. healthy eating for vegetarians
9. smart shopping for veggies and fruits
10. liven up your meal with vegetables and fruits
11. kid-friendly veggies and fruits
12. be a healthy role model for children
13. cut back on your kid's sweet treats
14. salt and sodium

*Let's Eat for the Health of It* and the 10 Tips Nutrition Education Series are available as a PDF at [ChooseMyPlate.gov](http://ChooseMyPlate.gov). The site provides practical information to individuals, health professionals, nutrition educators, and the food industry to help consumers build healthier diets with resources and tools for dietary assessment, nutrition education, and other user-friendly nutrition information. Additional resources will be available in the fall. MyPyramid will remain available to interested health professionals and nutrition educators in a special section of the new website.

A multi-year campaign will focus on one action-prompting message at a time starting with "Make Half Your Plate Fruits and Vegetables". As part of this new initiative, the USDA wants to see how consumers are putting MyPlate in to action by encouraging consumers to take a photo of their plates and share on Twitter with the hash-tag #MyPlate. What a great way to share the exciting meals your facility serves!

Before incorporating the new icon into print materials, make sure to read the MyPlate Graphics Standards (terms of use) at <http://www.cnpp.usda.gov/Publications/MyPlate/MyPlateGraphicsStandards.pdf>.

## Source:

<http://www.cnpp.usda.gov/dietaryguidelines.htm>  
[www.Choosemyplate.gov](http://www.Choosemyplate.gov)

Linda S. Eck Mills, MBA, RD, LDN, FADA is a career and life coach and author of the book *From Mundane to Ah Ha! Effective Training Objects*. Linda has experience working in hospice, corrections and long-term care environments. Contact her at [Linda@dycomserv.com](mailto:Linda@dycomserv.com) or [www.dycomserv.com](http://www.dycomserv.com)

## Calling All Authors!

### Renal Nutrition Forum Article Submissions Needed!

If you have ever considered submitting an article to the RNF now is the time!

Please consider sharing your work with fellow RPG members or reaching out to colleagues to inquire about work they may be interested in submitting.

### We guarantee publication!

For more information, please contact RNF Editor  
Sara Erickson @ [saraericksonrd@gmail.com](mailto:saraericksonrd@gmail.com)

# 2010 Dietary Guidelines and My Plate

Figure 1



**Key Recommendations**

**BALANCING CALORIES TO MANAGE WEIGHT**

- Prevent and/or reduce overweight and obesity through improved eating and physical activity behaviors.
- Control total calorie intake to manage body weight. For people who are overweight or obese, this will mean consuming fewer calories from foods and beverages.
- Increase physical activity and reduce time spent in sedentary behaviors.
- Maintain appropriate calorie balance during each stage of life—childhood, adolescence, adulthood, pregnancy and breastfeeding, and older age.

**FOODS AND FOOD COMPONENTS TO REDUCE**

- Reduce daily sodium intake to less than 2,300 milligrams (mg) and further reduce intake to 1,500 mg among persons who are 51 and older and those of any age who are African American or have hypertension, diabetes, or chronic kidney disease. The 1,500 mg recommendation applies to about half of the U.S. population, including children, and the majority of adults.
- Consume less than 10 percent of calories from saturated fatty acids by replacing them with monounsaturated and polyunsaturated fatty acids.
- Consume less than 300 mg per day of dietary cholesterol.
- Keep *trans* fatty acid consumption as low as possible by limiting foods that contain synthetic sources of *trans* fats, such as partially hydrogenated oils, and by limiting other solid fats.
- Reduce the intake of calories from solid fats and added sugars.
- Limit the consumption of foods that contain refined grains, especially refined grain foods that contain solid fats, added sugars, and sodium.
- If alcohol is consumed, it should be consumed in moderation—up to one drink per day for women and two drinks per day for men—and only by adults of legal drinking age.<sup>5</sup>

Figure 2

**FOODS AND NUTRIENTS TO INCREASE**

Individuals should meet the following recommendations as part of a healthy eating pattern while staying within their calorie needs.

- Increase vegetable and fruit intake.
- Eat a variety of vegetables, especially darkgreen and red and orange vegetables and beans and peas.
- Consume at least half of all grains as whole grains. Increase whole-grain intake by replacing refined grains with whole grains.
- Increase intake of fat-free or low-fat milk and milk products, such as milk, yogurt, cheese, or fortified soy beverages.<sup>6</sup>
- Choose a variety of protein foods, which include seafood, lean meat and poultry, eggs, beans and peas, soy products, and unsalted nuts and seeds.
- Increase the amount and variety of seafood consumed by choosing seafood in place of some meat and poultry.
- Replace protein foods that are higher in solid fats with choices that are lower in solid fats and calories and/or are sources of oils.
- Use oils to replace solid fats where possible.
- Choose foods that provide more potassium, dietary fiber, calcium, and vitamin D, which are nutrients of concern in American diets. These foods include vegetables, fruits, whole grains, and milk and milk products.

**Recommendations for specific population groups**

*Women capable of becoming pregnant<sup>7</sup>*

- Choose foods that supply heme iron, which is more readily absorbed by the body, additional iron sources, and enhancers of iron absorption such as vitamin C-rich foods.
- Consume 400 micrograms (mcg) per day of synthetic folic acid (from fortified foods and/or supplements) in addition to food forms of folate from a varied diet.<sup>8</sup>

*Women who are pregnant or breastfeeding<sup>7</sup>*

- Consume 8 to 12 ounces of seafood per week from a variety of seafood types.
- Due to their high methyl mercury content, limit white (albacore) tuna to 6 ounces per week and do not eat the following four types of fish: tilefish, shark, swordfish, and king mackerel.
- If pregnant, take an iron supplement, as recommended by an obstetrician or other health care provider.

*Individuals ages 50 years and older*

- Consume foods fortified with vitamin B<sub>12</sub>, such as fortified cereals, or dietary supplements.

**BUILDING HEALTHY EATING PATTERNS**

- Select an eating pattern that meets nutrient needs over time at an appropriate calorie level.
- Account for all foods and beverages consumed and assess how they fit within a total healthy eating pattern.
- Follow food safety recommendations when preparing and eating foods to reduce the risk of foodborne illnesses.

**Source:** <http://www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/PolicyDoc/ExecSumm.pdf>

Figure 3



## Dietary Guidelines 2010 Selected Messages for Consumers

Take action on the Dietary Guidelines by making changes in these three areas.

Choose steps that work for you and start today.

### Balancing Calories

- Enjoy your food, but eat less.
- Avoid oversized portions.

### Foods to Increase

- Make half your plate fruits and vegetables.
- Switch to fat-free or low-fat (1%) milk.

### Foods to Reduce

- Compare sodium in foods like soup, bread, and frozen meals—and choose the foods with lower numbers.
- Drink water instead of sugary drinks.



Figures 1 – 3 Reprinted from *Center for Nutrition Policy and Promotion, Dietary Guidelines 2010*

January 2011

**Source:** <http://www.cnpp.usda.gov/dietaryguidelines.htm>



# Member Spotlight

## Adele Huls, PhD, RD, LMNT

Recipient of RPG stipend for the 2011 Food & Nutrition Conference & Expo (FNCE) in San Diego, CA.  
Huls Professional Nutrition Services, Inc.  
Renal Dietitian, Chandron Community Hospital  
Chadron, NE  
drhulsrd@yahoo.com

*The following is a summary of the Medical Nutrition Therapy (MNT) in the Primary Care Settings for Chronic Kidney Disease (CKD) session. The speakers for the session are: Andrew Narva, MD & Theresa Kuracina, MS, RD.*

As an RD in a rural community who practices in many areas of dietetics, this topic was of interest because I teach diabetic classes and have the opportunity to review the labs of diabetic referrals. Speakers Andrew Narva, MD and Theresa Kuracina, MS, RD, presented startling statistics and offered great resources for carrying out recommendations to decrease the burden of CKD in the US. They provided ways of decreasing barriers in order to improve the care of kidney disease.

Dr. Narva started out by stating “we will be discussing some disturbing adult material”. This was followed by a slide entitled “Type 2 Diabetes is Driving the Increase in ESRD”. The slide showed the incidence of ESRD from 1980-2004 with rates adjusted for age, gender and race. He explained that type 2 diabetes is driving the increase in ESRD, with hypertension still being an important contributor, but is not nearly increasing at the rate of ESRD. He explained that delivering appropriate care to those who need it, rather than defining appropriate care, is the barrier to improved outcomes.

The challenges to improving CKD care were outlined as being 1) under-diagnosed, 2) poor implementation of recommendations of care, and 3) feelings of inadequacy by many clinicians, including

dietitians. Feelings of inadequacy were identified as:

- ▶ being uncertain about interpretation of diagnostic tests,
- ▶ being unclear about clinical recommendations,
- ▶ having low confidence in ability to successfully manage CKD, and
- ▶ having poor understanding of indications for and process of referral.

To help with the challenges and feelings of inadequacy in CKD care, Kuracina urged health care providers to go beyond the ABCs of diabetes care (A1C, Blood pressure, Cholesterol) for better diagnosis of CKD. The importance of obtaining glomerular filtration rate (eGFR) and urine albumin creatinine ratio (UACR) and their interpretation were stressed. These and many other helpful resources for diagnosis were also given and more can be obtained by going to the National Kidney Disease Education Program (NKDEP) web site, <http://nkdep.nih.gov>.

Recommendations of care to improve confidence in management in regard to phosphorus, potassium, protein and sodium intake were also presented; these patient education materials are not copyrighted and can be reproduced without permission.

Additionally the NKDEP website has information to help with the process of referral. All of the above references to the NKDEP can be found on the web site listed above (<http://nkdep.nih.gov>).

The information I obtained at this session helped me to understand the importance of early diagnosis and intervention when CKD is identified. This information gives direction to educating health care providers regarding referrals. We do not currently run UACRs in our lab. I could have said that about eGFR a few years ago also, but, after many requests, I am happy to report RD input does yield results. UACR requests have begun as a result of this session. Additionally, I plan to include more information regarding the importance of the nutrition-focused physical exam in CKD on my website, [www.nutritionfocusedexam.com](http://www.nutritionfocusedexam.com).

## November 2011 Board Certified Specialists in Renal Nutrition (CSR)

### Congratulations to Registered Dietitians:

#### California

Deborah Benner  
Marla Garbat  
Nicole Gilles  
Vidyut Lingamneni  
Darcie Nagel  
Lori Robinson  
Lori Spears  
Terri Verone

#### Colorado

Patricia Kulbeth  
Marcella McLaughlin

#### Connecticut

Margaret Vaghi

#### Delaware

Christina Wotton

#### Florida

Janet Coder  
Elizabeth Emison

#### Illinois

Susan Eysenbach  
Dawn Hebert

#### Kansas

Rebecca Hayek

#### Michigan

Tina Kennedy  
Sarah Kruger  
Kathleen Wreford

#### Nevada

Christie Giordano

#### New York

Joy DeCaro  
Susan Indilicato

Lisa Lawkins

Mary Schanler

#### Ohio

Julie Hempfling  
Connie Schultz  
Nancy Wischmeyer-Schaub

#### Oregon

Leslie Dilley

#### Pennsylvania

Allyn Evans  
Alice Thureau

#### South Carolina

Rebecca Parnell

#### Texas

Jeanne Wittmayer

#### Virginia

Silvia Figueiroa da Cruz

#### West Virginia

Katie Hoover



# Rena start

**An innovative and unique formulation for supplementary feeding or complete nutrition support in the dietary management of pediatric kidney disease in individuals one year and older.**



*Medical food intended  
for use under medical supervision.*

- Suitable for use in Acute or Chronic Kidney Disease
- Low levels of potassium, phosphorus, chloride, calcium and vitamin A to compensate for disease specific defects in the metabolism of these micronutrients
- Lower in specific trace elements (manganese, selenium, chromium, molybdenum) due to compromised ability to excrete them in kidney disease
- Contains DHA and AA
- Great tasting

For more information on Renastart or to place an order, please call  
**1-888-VITAFLO or visit [www.VitaFloUSA.com](http://www.VitaFloUSA.com).**



Innovation in Nutrition

Code: RS212 Feb 2012

# Member Spotlight

## **Cheryl Y. Montgomery, RD, LDN**

Recipient of RPG stipend for the 2011 Food & Nutrition Conference & Expo (FNCE) in San Diego, CA.

Clinical Nutrition Manager

SkyRidge Medical Center

Cleveland, TN

cymonty@yahoo.com

I attended the 2011 American Dietetic Association's (ADA) Food & Nutrition Conference & Expo (FNCE) in San Diego, California. The conference offered an abundant amount of information for the dietetic professional to choose from in pre-conference workshops and cutting-edge sessions in eight informational tracts. The Expo allowed individuals to meet with company representatives who provide information on new products and services, emerging trends, and the latest technologies in the nutrition industry. Along with the ADA Bookstore, the new ADA Pavilion offered a centralized location for professionals to browse through the Commission on Dietetic Registration (CDR), ADA Foundation, Grassroots & Political Action Center, Kids Eat Right Corner, Commission on Accreditation for Dietetics Education (CADE), and Member Services to have questions answered or obtain information.

There were many highlights of FNCE, but two stand out. As a breast cancer survivor, one of the highlights of FNCE for me was the Keynote address "The Art of Seeing the Invisible" by Nancy Brinker, founder of Susan G. Komen for the Cure Foundation. Also, during the opening session of FNCE, ADA President Sylvia Escott-Stump announced the name change of "American Dietetic Association" to the "Academy of Nutrition and Dietetics" as of January 2012. She stated the name change will communicate "we are the nutrition experts."

Although I attended many excellent sessions on diverse topics during FNCE, one of my favorite lectures was entitled "Navigating Intestinal Surgery: How to Assess and Feed the Altered GI Tract" and was presented by Neha Parekh, MS, RD, LD, CNSC and Kathy Barco, RD, LD, CNSC. While I find it challenging as a clinical dietitian to meet the nutritional and educational needs of renal, diabetic, and oncology patients, it is even more difficult to maintain the nutritional needs of patients who have undergone some type of GI resection. Neha Parekh began the session by reviewing the basics of gastrointestinal (GI) absorption and how an altered GI tract can cause mechanisms of malabsorption. The mechanisms may include rapid gastric emptying / rapid intestinal transit, acid hypersecretion, loss of surface area / ileocecal valve (ICV), bacterial overgrowth, and / or bile acid wasting. Short bowel syndrome may occur if large portions of the small bowel are resected due to illness or injury. Assessment of the bowel length and condition of the remaining GI tract are the initial steps in the nutritional management of the patient. If the approximate bowel length cannot be obtained from surgery documentation, other methods such as a small bowel

follow through can be used to determine the estimated length of the remaining bowel. Nutritional management takes into consideration the patient's absorption function which can be measured through nutritional assessments of the appearance of GI output (stool), dietary intake & daily fluid balance, laboratory values, and changes in body weight. GI function can increase the risk of malnutrition due to delayed gastric emptying, atrophy of intestinal mucosa, increased risk post-op fistulas, and overall poor healing; therefore, many patients require TPN initially to help restore nutritional status. Kathy Barco continued the session by evaluating the factors that influence intestinal adaptation: site of resection, length of remaining bowel, presence of ICV & colon, age of patient, luminal nutrients, GI hormones, and / or pancreaticobiliary secretions. The final objective was to analyze the methods used to develop an enteral feeding plan for patients with an altered GI tract if they are unable to maintain nutritional status. This lecture was especially relevant, as I recently had a patient with end-stage renal disease (ESRD) admitted to the hospital that had undergone a Roux-en-Y Gastric Bypass (RYGB) surgery to aid with weight loss so she could be eligible for a kidney transplant. The patient had experienced rapid weight loss with nausea and vomiting in the six weeks since the surgery. Not only did I have the challenge of meeting her nutritional needs with post op RYGB complications but also providing renal restrictions.

The Expo offered opportunities to interact with over 350 representatives of specialty foods, alternative medicine, nutritional supplements, assessment tools and resources, food delivery equipment, and much more. The aisles of the Expo were filled with individuals tasting samples of new food products and obtaining information on the latest technologies and products.

There were many other events besides the sessions and the Expo that were offered. For example, individuals could browse through poster sessions of fellow professionals that rotated throughout the three days. Additionally, The Renal Dietitians Practice Group (RPG) provided a Member Appreciation Breakfast on Sunday morning which was a great opportunity to network with other professionals and place faces with names of RPG Executive Committee members.

Several excursions were offered before and during FNCE. Friends and I ventured out via the Old Town Trolley tours to discover some of the fascinating history of the San Diego area such as Old Town, the Gaslamp Quarter, Balboa Park, San Diego Zoo, USS Midway, and Coronado. The two hour narrative tour allowed us to disembark and discover each site as we wanted. Although shuttles were available between the conference center and our hotels, I enjoyed the beautiful San Diego climate; and I recorded over 22,000 steps on my pedometer on two of the three days of FNCE.

I am very fortunate that my hospital administration understands the benefits of attending FNCE and allows me to take time off from work. Not only is FNCE a very valuable and enjoyable experience, networking with old friends and making new friends in the dietetic profession is a great occurrence as well. Next year FNCE will be held on October 6-9, 2012 in Philadelphia, PA. I encourage everyone to try to attend. FNCE is an enormous experience!

# Member Spotlight

## **Marianne Wolfe-Hutton, RD, CSR, CDE**

Recipient of RPG stipend for the 2011 Food & Nutrition Conference & Expo (FNCE) in San Diego, CA.  
Director Food & Nutrition Services and Environmental Services  
Healdsburg District Hospital  
Healdsburg, CA  
finelyfit1@gmail.com

*The following is a summary of the Medical Nutrition Therapy (MNT) in the Primary Care Settings for Chronic Kidney Disease (CKD) session. The speakers for the session are Andrew Narva, MD & Theresa Kuracina, MS, RD.*

CKD is not well managed in the primary care setting; CKD is often under-diagnosed or mis-diagnosed. The implementation of recommended care is poor. Specifically, many clinicians, including Registered Dietitians (RDs), feel inadequately educated.

Clinicians in the primary care setting report they are:

- Uncertain about how to interpret diagnostic tests.
- Unclear about clinical recommendations.
- Not confident in their ability to successfully manage CKD.
- Unclear about the process and indication for referral.

Generalist RDs can play a significant role in early diagnosis, treatment and patient education in primary care settings. Early intervention and appropriate care can assist patients at risk, including those with hypertension and diabetes, to slow CKD progression and treat complications. Dr. Narva reminded us that guidelines for treating CKD reflect international consensus and that the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of Hypertension (JNC 8) is about to come out with new guidelines. We are not doing well controlling hypertension in the U.S. (USRDS 2008 ADR). One third of patients needing dialysis initially met their nephrologist for first time in the emergency room.

Dr. Narva described the burden of CKD in the United States and identified how RDs in primary care settings can provide care to CKD patients. Dr. Narva said, "The greatest opportunity for improving outcomes is through self-management," and "Dietitians are key players in improving CKD care." The chronic care model is an empiric model proven to reduce disparities in CKD. What can primary care providers do? Recognize and test at-risk patients: monitor eGFR and urine albumin to creatinine ratio (UACR). UACR and much more was explained by Dr. Narva and Theresa and is explained on the National Kidney Disease and Education Program (NKDEP) website at <http://nkdep.nih.gov/>.

Lessons learned and messages we need to convey:

- CKD is part of primary care.
- Changing patterns of care require changing the system.
- Improvement in care results from changes implemented by physician and non-physician health professionals.

Theresa described her practice experience, the new CKD certificate training program and clinical tools to provide care to pre-dialysis CKD patients. She explained in detail the metabolic function of the kidneys. She informed the audience that in September 2011, the NKDEP revised an overview guide for dietitians – *Chronic Kidney Disease and Diet: Assessment, Management and Treatment*. This can be downloaded from their website listed under the heading 'Health Professionals' section at <http://nkdep.nih.gov>.

The Academy's Center for Professional Development has launched a new online certificate of training program entitled, Chronic Kidney Disease Nutrition Management.

This curriculum is comprised of five modules.

- Module 1: Chronic Kidney Disease Basics
- Module 2: Slow Progression
- Module 3: Complications
- Module 4: The "Diet" for Chronic Kidney Disease
- Module 5: The Transition from Chronic Kidney Disease to Kidney Failure

The program includes activities, case studies and tools that can be used when counseling people with CKD. This program was developed in partnership with the NKDEP, an initiative of the National Institute of Diabetes and Digestive and Kidney Diseases of the National Institutes of Health.

Participants receive the certificate associated with the training, if the five modules are completed and participants pass a final exam in Module 5 with an 80 percent score. More information about this certificate program can be obtained at [www.eatright.org/cpd/online](http://www.eatright.org/cpd/online).

I'd like to take this opportunity to thank the Renal Practice Group and Stipends & Awards Chair, Sandra McDonald-Hangach, RD, for this stipend to hear these two excellent presenters at this very informative, interesting and timely session.

## ***Congratulations***

### **To the New RPG Fifty-Year Members (Joined in 1962)**

Lenore S. Hodges • Clarissa S. London  
Sandra M. Pechous • Concetta J. Schroeffer

## ***Congratulations***

### **RPG Fifty-Year Plus Members (Joined before 1962)**

Muriel Bradburn • Drena Damascos • Marilyn Goska  
Annie Jelks • Maureen Kachinski • Barbara Ketay  
Marilyn Lawson • Norma Ramirez-Kent • Rachel Stern  
Emma Weigley • Despina Zerdes





**Rachael Majorowicz, RD, LD**

RPG Chair

majorowicz.rachael@mayo.edu

**If you don't like something, change it**

*~Mary Engelbreit*

The number one complaint I hear from renal patients is that they are frustrated with constantly being told what they can't do. Like our patients, renal dietitians also grow tired of being told what we cannot do: We cannot have sufficient time or resources to provide optimal patient care and we cannot have adequate time to provide the quality education our patients need. Despite our protestations, the end result is always the same; we are told there is nothing we can do about it.

Well, RPG and CRN are teaming up to find out just what we CAN do! We have decided to tackle two issues that have plagued renal dietitians. First, disparities in providing care to dialysis patients, such as cut-backs to RD time or responsibilities as "cost-saving" measures in the bundled environment. RDs report decreased time spent with patients, patient care, the quality of education, and outcomes. These cutbacks are unacceptable and we argue that there IS something you can do! RPG and CRN are working to create a toolkit that will outline what steps you can take to address the concerns you may see in your dialysis clinic or how to pursue RD:patient ratios at your state level. Also, I hope you have completed the recent RPG survey to help us identify the specific concerns and the extent of problems being experienced in dialysis units. We are teaming up with the National Kidney Foundation and the Academy of Nutrition and Dietetics to address these concerns.

Second, we also cannot help but be concerned about the difficulty our patients face when trying to make food choices. The renal diet is complicated enough, but trying to balance this knowledge with staying abreast of label reading and food additives... our patients don't stand a chance. With the lower sodium initiatives pushing through the food industry, we are already seeing an increased use of potassium additives. Additionally, phosphorus additives continue to confound our patients. And now research is showing that phosphorus additives may be harmful to the general population as well. RPG plans to bring this issue to greater light by sponsoring a session at FNCE 2012 regarding the health concerns that phosphate additives are creating for everyone... not only those with renal disease.

Additionally, RPG and CRN are teaming up to further the work of the Kidney Friendly Food Shelf. We are partnering with the Academy's Food & Culinary Professionals Supermarket subgroup to better educate grocery store RDs regarding renal diets, create an education resource for patients to help simplify their food choices, and hopefully more to come!

I hope to have the opportunity to meet many of you this year!

**Lisa Gutenkunst, MEd, RD, CSR, CDN**

NKF-CRN Chair

2012 will be an eventful year for us all. Not only is it a leap year, a presidential year, and, possibly if you believe all of doomsday reports, the end of the world, but also the year in which when the National Kidney Foundation Clinical Meetings (NKF-CM12) are held in Washington, D.C. and the year of the biennial meeting of the International Congress of Renal Nutrition and Metabolism (ICRNM) in Honolulu, HI!

The NKF-CM12 will be held May 9-13, 2012 at the Gaylord National in Washington, D.C. The event is expected to draw hundreds of renal professionals from around the nation and will include some international guests. The Dietitian Track has been developed to incorporate not only nutritional aspects of treating renal disease but also medical advances and psycho/social aspects of the disease. Pre-conference seminars include Foundation of Nutrition Practice for Kidney Disease (Strategies I) and Advance Practice in Renal Nutrition (Strategies II). Strategies I is designed for new dietitians to the field in addition to allied health professionals who want to know more about the basics of renal nutrition. It is led by national leaders in our field. Strategies II takes a closer look at specific topics within the field of renal nutrition. This year the focus is on inflammation and anabolism.

The 16th meeting of the ICRNM takes place June 26-30, 2012 in Honolulu, HI. This biennial event draws an international group of researchers and practitioners in the field of renal nutrition and metabolism. There is over 50 seminars planned focusing on medical nutrition therapy, emerging treatments for bone mineral metabolism, patient education strategies, and early stage chronic kidney disease management. Though Honolulu seems a long distance to travel, the opportunity to meet the most advanced leaders in our field is invaluable. For more information on this meeting, please visit: <http://www.renalnutritionweek.com>.

Happy New Year to all and hope that I have the opportunity to meeting many of you this year!

**WANT TO GET INVOLVED?**

***Let us know!***

Contact Membership Chair:  
**Cynthia Terrill, RD, CSR, CD**  
**[cindy.terrell@hsc.utah.edu](mailto:cindy.terrell@hsc.utah.edu)**



# Calendar of Events

## April 2012

### 2012 American Society of Pediatric Nephrology Annual Meeting

Boston, MA

April 28-May 1, 2012

<http://www.aspneph.com/>

## May 2012

### National Kidney Foundation 2012 Spring Clinical Meetings

Gaylord National; Washington, DC

May 9-13, 2012

[www.kidney.org/news/meetings/clinical/index.cfm](http://www.kidney.org/news/meetings/clinical/index.cfm)

## June 2012

### American Transplant Congress 2012

Boston, MA

June 2-5, 2012

[www.atcmeeting.org/2012/](http://www.atcmeeting.org/2012/)

### 1st World Renal Nutrition Week

16th International Congress on Renal Nutrition and Metabolism (ICRNM)

Honolulu, HI

June 26-30, 2012

[www.renalnutrition.com](http://www.renalnutrition.com)

## July 2012

### 24th International Congress of the Transplantation Society

Berlin, Germany

July 15-19, 2012

<http://transplantation2012.org>

## August 2012

### NATCO 37th Annual Meeting

Grand Hyatt Washington DC, Washington DC

August 12-15, 2012

<http://www.natco1.org>

## October 2012

### ADA Food & Nutrition Conference and Expo

Philadelphia, PA

October 6-8, 2012

[www.eatright.org/fnce/](http://www.eatright.org/fnce/)

### ASN Kidney Week 2012

San Diego Convention Center; San Diego CA

October 30-November 4, 2012

[www.asn-online.org](http://www.asn-online.org)

## Recently Published . . . .

Don't have time to search recently published articles in nephrology nutrition? Consider this your one-stop-shop for research articles pertinent to your practice. We've reviewed the following articles from a variety of publications in an effort to help keep our RPG members informed – we hope you find this list helpful and, as always, would appreciate your feedback and suggestions!

### January 2012

Figueredo-Dias V, Cuppari L, Ghedini Garcia-Lopes M, et al. Risk factors for hypovitaminosis D in nondialyzed chronic kidney disease patients. *J Ren Nutr*. 2012; 22(1):4-11.

Hasuiki Y, Hama Y, Nonoguchi H, et al. Persistent homocysteine metabolism abnormality accelerates cardiovascular disease in hemodialyzed patients – the Nishinomiya study. *J Ren Nutr*. 2012; 22(1):12-18.

Shoben AM, Levin G, deBoer IH, et al. Variation in oral calcitriol response in patients with stages 3-4 CKD. *Am J Kidney Dis*. 2012 Jan 30. doi:10.1053/j.ajkd.2011.11.041.

Wyskida K, Witkowicz J, Chudek J, et al. Daily magnesium intake and hypermagnesemia in hemodialysis patients with chronic kidney disease. *J Ren Nutr*. 2012; 22(1): 19-26.

Bossola M, Ciciarelli C, Di Stasio E, et al. Relationship between appetite and symptoms of depression and anxiety in patients on chronic hemodialysis. *J Ren Nutr*. 2012; 22(1): 27-33.

Kant KS, Gonzalez AR, Hariachar S, et al. Converting to doxercalciferol capsules from intravenous paricalcitol or doxercalciferol. *J Ren Nutr*. 2012; 22(1):34-40.

Chen YW, Chen HH, Pan CF, et al. Interdialytic weight gain does not influence the nutrition of new hemodialysis patients. *J Ren Nutr*. 2012; 22(1): 41-49

### February 2012

Gill JS and Tonelli M. Penny wise, pound foolish? Coverage limits on immunosuppression after kidney transplantation. *N Eng J Med*. 2012 Feb 1. doi:10.1056/NEJMp1114394.[Epub ahead of print]

Wasse H, Huang R, Long Q, et al. Efficacy and safety of a short course of very-high-dose cholecalciferol in hemodialysis. *Am J Clin Nutr*. 2012;95:522-528.

Alssema M, Newson RS, Bakker SJL, et al. One risk assessment tool for cardiovascular disease, type 2 diabetes, and chronic kidney disease. *Diab Care*. 2012 Feb 14. doi:10.2337/dc11-1417 [Epub ahead of print]

Fiorina P, Vezzoli P, Bassi R, et al. Near normalization of metabolic and functional features of the central nervous system in type 1 diabetic patients with end-stage renal disease after kidney-pancreas transplantation. *Diab Care*. 2012;35:367-374.

# 2011-2012 RPG Executive Committee

**Mission:** Renal dietitians practice group is leading the future of dietetics by promoting and supporting its members working in nephrology nutrition.

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

## OFFICERS:

### Chair

Rachael Majorowicz, RD, LD  
majorowicz.rachael@mayo.edu

### Immediate Past Chair

Kathy Madigan, MS, RD, LDN, MBA  
kmmnutrfit@verizon.net

### Chair-Elect

Sarah Kruger, MS, RD, CSR  
Kruger\_sarah@yahoo.com

### Secretary

Jane Louis, RD, CSR, LD  
louisjl@att.net

### Treasurer

Stacey C. Phillips, MS, RD  
staceycphillips@yahoo.com

### HOD Member

Pam Kent, MS, RD, CSR, LD  
pamkentrd@yahoo.com

## RNF EDITORIAL BOARD:

### RNF Managing Editor

Megan Sliwa, RD, LDN, MBA  
megansliwa@aol.com

### Electronic Media Manager

Cathy M. Goeddeke-Merickel, MS, RD, LD  
cmgmerickel@gmail.com

### RNF Editor

Sara Erickson, RD, CSR, LDN, CNSC  
saraericksonrd@gmail.com

### RNF Assistant Editor

Jackie Abels, MA, RD, LD  
jackie.abels@aol.com

### RNF Advertising Editor

Emily R. Cutler, MS, RD, LDN  
emilycreamer@aol.com

## NOMINATING COMMITTEE:

### Nominating Chair

Kathy Schiro Harvey, MS, RD, CSR  
kathyh.rd@pskc.net

### Nominating Member

Elizabeth Neumann, RD, LD  
eneum1961@gmail.com

### Nominating Member

Betty Parry Fisher, MS, RD  
Betty.Fisher@Genzyme.com

### Membership Chair

Cynthia J. Terrill, RD, CSR, CD  
cindy.terrill@hsc.utah.edu

## COMMITTEE CHAIRS:

### Awards/Scholarship Chair

Sandy McDonald-Hangach, RD

### Professional Resource Center Coordinator

Nadiya Lakhani, RD, LD

### Public Policy Chair

Sarah Mott, MS, RD, LDN

## ADA CONTACT:

### Manager, DPG Relations

Susan DuPraw, MPH, RD  
800/877-1600 ext. 4814  
sdupraw@eatright.org

## RNF Guidelines for Authors

### Article length:

Article length is determined by the Editor for each specific issue. The feature article (including abstract) is approximately 3000 words (not including tables/graphs). Other articles are usually 1000-1500 words; member highlights and reports are approximately 400-500 words.

### Text format:

Times New Roman font, 12 point, double space.

### Tables/Illustrations:

Tables should be self-explanatory. All diagrams, charts and figures should be camera-ready. Each should be accompanied by a title and brief caption that clearly explains the table, chart, diagram, figure, illustration, etc.

### References:

References should be cited in the text in consecutive order parenthetically. At the end of the text, each reference should be listed in order of citation. The format should be the same as the Journal of the Academy of Nutrition and Dietetics.

### Reference citation examples:

#### Article in periodical:

Knowler WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Eng J Med*. 2002;346:393-403.

#### Book:

Institute of Medicine. *Dietary Reference Intakes: Applications for Dietary Assessment*. Washington, D.C.: National Academy Press; 2001.

#### Chapter in a book:

Walsh J. Which insulin to use and how to start. In: *Using Insulin*. San Diego, Calif.: Torrey Pines Press; 2003.

#### Web site:

Medscape drug info. Available at [www.medscape.com/druginfo](http://www.medscape.com/druginfo). Accessed August 15, 2011.

#### Author information:

List author with first name, middle initial (if any), last name, professional suffix and affiliation below the title of the article. Also include the primary author's complete contact information including affiliation, phone, fax and email address.

All submissions for publication should be submitted to the editor as an email attachment (MS Word file). The feature articles from the Renal Nutrition Forum will be posted on the Members Only Section of the RPG website (password protected). Thus, please include a brief abstract and 2-3 key words along with feature article submissions.

For all inquiries please email: [helpU@renalnutrition.org](mailto:helpU@renalnutrition.org)



# Nutrition designed for people on dialysis



- Excellent source of high-quality protein to help meet nutritional needs and replace **protein** lost during dialysis
- Low in **phosphorus, potassium and sodium**
- Has **Carb Steady**<sup>®</sup> carbohydrate blend designed to help manage blood glucose response

## NEW KIDNEY CLUB!

Money Saving Offers. Education. Support.

Help Enroll patients today at [Nepro.com](http://Nepro.com)

Available Through

- [www.abbottstore.com](http://www.abbottstore.com)
- Home Delivery (1-800-986-8502)

Also in pharmacy section next to Glucerna<sup>®</sup> at:

*Walgreens*

**H-E-B**

**RITE  
AID  
PHARMACY**

 **Giant**

 **Stop&Shop**

Product	Flavor/Description	Item #
Nepro <sup>®</sup> with CarbSteady <sup>®</sup>	Homemade Vanilla (24ct)	62094
	Mixed Berry (24ct)	62090
	Butter Pecan (24ct)	62092

\* Homemade Vanilla bottles only available at retail locations.

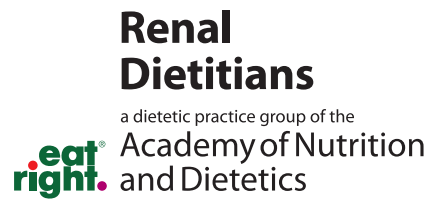
Use under medical supervision.

©2011 Abbott Laboratories Inc.  
79565/May 2011 LITHO IN USA

 **Abbott  
Nutrition**

Sara Erickson, RD, CSR, LDN, CNSC  
Editor, *Renal Nutrition Forum*  
8954 Meadowmont View Drive  
Charlotte, NC 28269

PRESORTED  
STANDARD  
US POSTAGE PAID  
ROCHESTER MN  
PERMIT NO. 289



2012 Copyright by Renal Dietitians  
Dietetic Practice Group of the Academy of  
Nutrition and Dietetics. All rights reserved.

**Visit the Renal Dietitians "Members Only Section"**  
**for valuable patient and professional resources @**  
**[www.renalnutrition.org](http://www.renalnutrition.org)**

---