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In This Issue

Feature Article

Hidden Phosphorus: A New Challenge for the Nephrology Dietitian

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This article has been approved for 2 CPE units and the CPEU insert can be accessed in the Members Only Section of the website from the CPEU Inserts link.

The nephrology healthcare team has struggled to help patients with chronic kidney disease (CKD) control serum phosphorus since the 1960's. It was determined from a National Institutes of Health MedLine literature search that articles linking elevated serum phosphorus levels with secondary hyperparathyroidism (1) and with soft tissue-calcification (2) appear as early as 1966. Associations between "persistently high (over 60) calcium phosphorus product" and cardiac calcification appear as early as 1975 (3). By 1975 sufficient evidence supported the association between elevated phosphorus levels and renal osteodystrophy. The American Dietetic Association (ADA) subsequently issued a recommendation for restricting phosphorus intake in the CKD population in hopes of minimizing and even preventing the bones lesions associated with secondary hyperparathyroidism (4). Today, the nephrology community has shifted the focus to protecting this population from the systemic effects of hyperphosphatemia, such as cardiac calcification.

The National Kidney Foundation (NKF) issued the Kidney Disease Outcome Quality Initiative (K/DOQI) Clinical Practice Guidelines for Bone Metabolism and Disease in 2003 (5). Since that time much emphasis has been placed on achieving serum calcium, phosphorus, calcium-phosphate product (CaxP) and parathyroid hormone (PTH) goals. Medications such as sevelamer hydrochloride, lanthanum carbonate, and calcimimetic agents have helped to acheive the established calcium and PTH goals. However hyperphosphatemia remain an ongoing problem and challenge for the nephrology healthcare team and patients.

In an effort to address this problem, dietitians emphasize adherence with phosphate binder prescriptions and provide extensive instruction on low phosphorus diets. In addition to the basic advice to limit those foods high in phosphorus such as dairy products, legumes, and colas, nephrology dietitians also advise patients to choose low phosphorus protein foods. Despite more options in phosphate binding medications and aggressive diet education, dietitians do not always see the results that they expect. In many cases, the culprit can be traced back to the diet in the form of hidden phosphorus.

With the demand for high quality convenience foods, food manufacturers have increased the use of phosphorus additives. They help ensure the quality and flavor that Americans demand and expect from prepared foods. Traditional products using phosphorus additives include restructured meats (chicken nuggets

RPG

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From the Editor's Desk

Winston Churchill said, "We make a living by what we do, but we make a life by what we give." As I took a step back to review the various articles in this issue, I am reminded of how many various roles nephrology dietitians play. As well as clinicians, we can be counselors, detectives, media spokespersons, and patient advocates, etc.

Our feature article by Lisa Gutekunst MSEd, RD, CSR, CDN, addresses hidden phosphorus. In addition to taking phosphorus binders and following a low phosphorus diet of "standard" low phosphorus foods, patients need to also identify hidden sources of phosphorus in our food supply. This article addresses why it is used, how to identify sources, and how to relay this information to patients and grocery store personnel. From my experiences with my hemodialysis patients, phosphorus is that one lab value that I am constantly discussing with the patients and staff, month after month. I found this article very practical, and have already implemented some of the suggestions with my patients. We are pleased to offer 2 CPEUs for this article, and you can find more information on the web at www.renalnutrition.org.

> "Volunteering can be an exciting, growing, enjoyable experience. It is truly gratifying to serve a cause, practice one's ideals, work with people, solve problems, see benefits, and know one had a hand in them."
> *Harriet Naylor*

Our advances in practice article by Lynn K. Munson, RD, LD approaches a case study and associated literature review on the management of a patient with chronic kidney disease stage 5, type 2 diabetes, and obesity. The author reviews her approach to treating the patient, while also discusses the many challenges and unknowns there are with treating these patients. Questions include: which weight do you use for dosing and which predictive equation do you use for estimating calorie and protein needs? Note that the CRN chair message also addresses a call for research regarding predictive equations for chronic kidney disease patients.

It is really hard to believe that we are embarking on a new calendar year with ADA! It has been my pleasure to serve as your editor of the Renal Nutrition Forum, and with completion of this issue that I officially hand off the role to our incoming editor, Rachael Majorowicz, RD, LD. I would like to thank the RPG Executive Committee members, authors, reviewers, proof readers, and test writer for all of your hard work and support this past year. I would especially like to thank Cathy M. Goeddeke-Merickel, MS, RD, LD, who collaborated with me as managing editor this past year. She has been the driving force of numerous advances on the Renal Nutrition Forum and our website (www.renalnutrition.org), and has been such a wonderful mentor to me.

As we begin our new year with ADA, I sincerely ask you to consider contributing to nephrology nutrition within our practice group. For me, being a part of the RPG Executive Committee has really expanded my networking with dietitians across the country, allowed me to attend educational sessions, kept me "in the loop" of what is going on behind the scenes at ADA, and most importantly given me a sense of pride for contributing to our profession. Without submissions of articles from nephrology professionals, our publication cannot continue to grow. In addition to the Forum, RPG is also involved in numerous task forces, including those focused on education and research. We are also interested in expanding our mentoring program, so look for more details in the future. We are open to new ideas! No matter how big or small a contribution, it counts. ◆

Aimee Zajc, RD LD

Table 1
Non-traditional foods containing phosphate additives

Beverages	• Dr. Pepper Red Fusion® • Hawaiian Punch® All Flavors • Sunny Delight® All Flavors • Hire's® Root Beer • Mountain Dew® Code Red • Mountain Dew® Amp • Nestea Cool® Iced Tea All Flavors • Non-Dairy Creamers All Flavors	• Fruitworks® All Flavors • Aquafina Essentials® • Dansani® Flavored • Tropicana® Fruit Drinks • Fanta® Orange • Slice® Cherry • Some calcium fortified juices • Canned and plastic bottled Iced Tea
Cereal/Breakfast Bars Greater than 100mg/serving	• Kellogg's® Special K-Low Carb • Kellogg's® Complete Bran Flakes • Kellogg's® Cracklin Oat Bran • Kellogg's® Healthy Start - Heart Smart • Kellogg's® Healthy Start - Soy Protein • General Mills® Multibran Chex • General Mills® Oatmeal Crisp Almond	• Quaker® Squares • Quaker® Life - All Flavors • Quaker® Breakfast Bars • Malt-O-Meal® Graham Squares • Milk 'n Cereal® Breakfast Bars • General Mills® Shrek • General Mills® Wheat Chex
Meat	• Frozen Turkey • Hormel Always Tender® Pork • Frozen Beef	• Frozen Chicken • Chicken Nuggets

and hotdogs), processed/spreadable cheeses, "instant" products (puddings and sauces), refrigerated bakery products, and beverages (6). Now foods containing additives extend into all segments of the food pyramid.

Additives appear in products once considered innocuous. Many brands of flavored water now contain a phosphate additive. Canned and plastic bottled iced teas may contain phosphorus. Even some brands of "fresh" meat contain phosphorus additives. Table 1 lists other foods normally considered "safe" for the CKD patient that may now may contain phosphate additives.

Phosphate salts are used for a variety of reasons. They are an inexpensive and effective way for manufactures to ensure the quality of and enhance their products. The U.S. Food and Drug Administration consider phosphate additives Generally Regarded as Safe (GRAS). Table 2 lists the most common phosphate salts used in today's foods and their functions.

Phosphate salts act not only as acids, as in the case of phosphoric acid, but also as buffers. They ensure the creaminess and meltability of dairy products, promote coagulation or prevent coagulation, emulsify, preserve the texture of frozen meats, and increase flavor in lean meats. They also tenderize tough meat, ensure the pliability of refrigerated and frozen bakery products, and can be used to add selected nutrients to a product, such as calcium.

Enhanced meat products are now available in commercial and discount supermarkets nationwide. Enhanced meat is fresh meat that has been injected with a solution containing water and other ingredients. The "other ingredients" consist of a mixture of sodium salts, phosphate salts, potassium salts, antioxidants, and/or flavorings. In appearance, enhanced meat resembles fresh meat, however it differs in nutritional value. Because the meat has been altered from its natural composition, manufactures must affix a food label stating that the meat product has been enhanced. Enhanced meats are significantly higher in sodium compared to fresh, unadulterated meat. On the average, fresh meats contain about 50-75 mg of sodium for a 3 ounce serving whereas enhanced meats contain over 300 mg for the same serving size.

The additional sodium found in enhanced meat products is a concern for individuals with hypertension who follow a low sodium diet. It is common for dietitians to encourage the use of fresh meat over processed meats. Enhanced meats look like fresh meat and are sold right next to fresh, unenhanced meats. Thus many individuals who hope to select the fresh meat end up with the enhanced high sodium meat product.

Enhanced meat products are not new to the American food supply; they have been made in homes and found in supermarkets for a long time. The self-basting turkey is one example of an enhanced product that has been a staple in many American homes

Table 2
List of Phosphate Additives and Their Uses

Phosphate Additive Common Name	Alternate Names	Uses	Products
Dicalcium Phosphate Anhydrous	Calcium phosphate dibasic; Calcium phosphate, secondary; Dicalcium orthophosphate; Dical; Calcium monohydrogen phosphate; Secondary calcium phosphate; DCP; DCPA	Mineral source; dough conditioner	Bakery mixes; yeast-raised bakery products; cereals; dry powder beverages; flour; food bars; infant food; milk-based beverages; multivitamin tablets; yogurts. Used in powder form as an abrasive in toothpaste.
Dicalcium Phosphate Dihydrous	Dicalcium phosphate duohydrate; Calcium phosphate dihydrate; Calcium phosphate,Secondary dihydrate; Dicalcium orthophosphate dihydrate; Dical; Calcium monohydrogen phosphate dihydrate; Secondary calcium phosphate dihydrate; DCPD	Mineral source; leavening agent	Bakery mixes; cereals; dry powder beverages; flour; food bars; infant food; milk-based beverages; multivitamin tablets; yogurt.
Dipotassium Phosphate	Dipotassium monophosphate; Potassium phosphate, dibasic; Dikalium phosphate; Dipotassium hydrogen phosphate; DKP	Nutrient in yeast culturing; sequestrant; buffer.	Yeast-containing products; non-dairy creamers; casein based creamers; processed cheese, meat products, mineral supplements, starter cultures.
Disodium Phosphate Anhydrous	Disodium monohydrate phosphate; Sodium phosphate, dibasic; Neutral sodium phosphate; Dibasic sodium phosphate; Disodium hydrogen phosphate; Disodium orthophosphate; Phosphate of soda; Secondary sodium phosphate; Exsiccated sodium phosphate; DSP; DSPA	Sequestrant; emulsifier; buffering agent; absorbant; pH control agent; protein modifier; source of alkalinity, stabilizer. Is used to adjust pH of cereal and pasta products to maintain quality color in final product. Accelerates the cook time of pasta and quick cooking cereals. Used during production of sprayed dry cheese and nonfat milk powders. Protects the milk proteins from heat dehydration allowing the proteins to remain dispersed during the spray drying process which assists in the solubility of powders upon reconstitution with water. Stabilizes the emulsion to enhance flavor, body and appearance of the final product.	Breakfast cereal;cheese; condensed milk; cream; evaporated milk; flavored milk powders; gelatin; half & half; ice cream; imitation cheese; infant food; instant cheesecake; instant pudding; isotonic drinks; nonfat dry milk; pasta; processed cheese; starch; vitamin capsules; whipped topping.

Information from: ICL Performance Products LP

Table 2, cont.
List of Phosphate Additives and Their Uses

Phosphate Additive Common Name	Alternate Names	Uses	Products
Disodium Phosphate Dihydrous	Disodium phosphate duohydrate; Disodium monohydrogen phosphate dihydrate; Sodium phosphate dibasic dihydrate; Neutral sodium phosphate dihydrate; Dibasic sodium phosphate dihydrated; Disodium orthophosphate dihydrate; Phosphate of soda dihydrate; Seconday sodium phosphate dihydrate; Sorensen's phosphate; Sorensen's sodium phosphate; DSPD	Same as Disodium Phosphate Anhydrous	Same as Disodium Phosphate Anhydrous
Monocalcium Phosphate Monohydrate	Monocalcium phosphate monohydrate; Calcium phosphate, monobasic or primary; Calcium acid phosphate; Calcium biphosphate; MCP, MCPM	Acidulant for foods and beverages; leavening acid; nutrient; dietary supplement; yeast food dough conditioner. Calcium source for fortification or enrichment.	Biscuits; cakes; donuts; muffins.
Magnesium Phosphate	DMPT	Nutritional source of magnesium and phosphorus; pH control agent; dietary supplement; flow aid	Magnesium source in infant formulas and diet beverages.
Monopotassium Phosphate	Acid potassium phosphate; Potassium phosphate monobasic; Potassium biphosphate; Potassium acid phosphate; Potassium dihydrogen phosphate; Sorensen's potassium phosphate; MKP.	pH control agent; buffering agent; acidulant; leavening agent; nutrient source.	Bread; doughs; dry powder beverages; eggs; isotonic beverages; mineral supplements; starter cultures; yeast cultures.
Phosphoric acid	Orthophosphoric acid; Acid monophosphoric	Acidulant; pH control agent; buffering agent; flavor enhancer; sequestrant; stabilizer; thickener; synergist.	Carbonated and noncarbonated beverages.
Sodium Hexametaphos-phate	SHMP; Graham's salt; Sodium phosphate glass	Sequestrant; neutral salt; deflocculant; curing agent; dough strengthener; emulsifier; firming agent; flavor enhancer; flavoring agent; humectant; nutrient supplement; processing aid; stabilizer and thickener; surface-active agent; synergist; texturizer and buffering agent.	Meat; seafood; poultry; vegetables; cream; half & half; ice cream; whey; processed cheese; eggs; table syrup; toppings; beverages.

Information from: ICL Performance Products LP

Table 2, cont.
List of Phosphate Additives and Their Uses

Phosphate Additive Common Name	Alternate Names	Uses	Products
Sodium Tripolyphosphate	Pentasodium triphosphate; Sodium triphosphate; Tripolyphosphate; Sodium polyphosphate; Triphosphoric acid; Pentasodium salt; STP; STPP.	Sequestrant; pH control agent; emulsifier; provides alkalinity; buffering agent; protein modifier; antioxidant; curing agent; flavor enhancer; humectant; thickener and stabilizer; texturizer, moisture retention.	Meat; seafood; poultry; vegetable proteins; processed cheese; sour cream; dips & yogurt; eggs; table syrups; whipped toppings; vegetables; whey.
Tetrasodium Pyrophosphate	Sodium pyrophosphate, tetrabasic; Tetrasodium diphosphate; Sodium diphosphate; TSPP.	Buffering agent; pH control agent; alkalinity source; dispersing agent; protein modifier; coagulant, sequestrant; moisture retention; antioxidant; color stabilizer.	Meat; poultry; seafood; processed cheese; potato products; ice cream; frozen desserts.
Tricalcium Phosphate	Calcium phosphate tribasic; Calcium phosphate tertiary; Hydroxy apatite; Tricalcium orthophosphate; TCP	Anti-caking agent; absorbant; calcium supplement; suspension polymerization agent.	Salt substitutes; dry beverage mixes; dry gravy mixes; spice blends; cereal; bakery mixes; flour; beverages; pharmaceuticals.
Trisodium Phosphate Anhydrous	Trisodium orthophosphate; Sodium phosphate tribasic; Basic sodium phosphate tertiary; Oakite; TSP; TSPA.	Buffer; emulsifying agent; stabilizer; protein modifier; provides "meltability" in processed cheese; quickens cooking time of cooked breakfast cereals; color agent.	Processed cheese and cheese products; cooked breakfast cereals; imitation cheese; isotonic beverages.
Monopotassium Phosphate Anhydrous	Monosodium dihydrogen phosphate; Sodium phosphate monobasic; Sodium biphosphate; Acid sodium phosphate; Sodium phosphate primary; Sodium dihydrogen phosphate; Monosodium orthophosphate; Primary sodium phosphate; MSP; MSPA.	Dry acidulant; buffering agent; emulsifier; leavening agent; protein modifier; sequestrant; gelling agent; color enhancer, flavor enhancer (tartness).	Cola beverages; dry powder beverages; egg yolks; gelatin; instant cheesecake; instant pudding; isotonic beverages; processed cheese; non-cola beverages; liquid egg mixtures.
SAPP	Acid sodium pyrophosphate; Disodium dihydrogen diphosphate; Dibasic sodium pyrophosphate; Disodium dihydrogen pyrophosphate; SAPP.	Emulsifier; formulation aid; humectant; leavening agent, pH control agent; acidulant; buffering agent; coagulant; dispersing agent; protein modifier; processing aid; sequestrant; stabilizer; thickener; synergist; texturizer.	Icing and frostings; processed meat; cured meats; processed chicken products; hotdogs; bologna; non-dairy creamers; processed potatoes; albacore tuna; processed cheese; vegetables; seafood; imitation cheese.
Tetrapotassium Pyrophosphate	Potassium pyrophosphate, tetrabasic; Tetrapotassium diphosphate; Potassium diphosphate; Diphosphoric acid tetrapotassium salt; TKPP.	Buffering agent; pH control agent; alkalinity source; dispersing agent; protein modifier; coagulant, sequestrant; nutrient source; antioxidant; texurizer.	Processed cheese; milk powders.

Information from: ICL Performance Products LP

Table 2, cont.
List of Phosphate Additives and Their Uses

Phosphate Additive Common Name	Alternate Names	Uses	Products
Pentasodium Triphosphate	Sodium triphosphate; Sodium triphosphate; Tripolyphosphate; Sodium polyphosphate; Triphosphoric acid, Pentasodium salt; STP; STPP.	Sequestrant; emulsifier; reduces oxidation; moisture retention; pH control; buffering agent; coagulant; dispersing agent; curing agent; flavor enhancer; humectant; thickener and stabilizer; texturizer.	Meat; poultry; seafood.
Tripotassium Phosphate	Basic potassium phosphate; Potassium phosphate tribasic; TKP	Alkalinity source; buffering agent; emulsifier; nutrient; protein modifier and stabilizer.	Starter cultures; processed cheese; dairy products; bread and dough conditioners; mineral supplements; isotonic beverages; cereals.

Information from: ICL Performance Products LP

around the holidays. Alternately, home chefs who soak their poultry, inject a roast, or marinade their meat before cooking are creating an enhanced meat product.

There are numerous benefits for the consumer as well as the grocer to sell and use enhanced meats. Consumer convenience is the first benefit. Americans are spending less time preparing meals, and, in our "grab and go" society, knowledge about meat selection and preparation is declining (7). Enhanced meat products provide a quick and easy solution to everyday meal planning.

Another benefit is being able to maintain the tissue integrity on an enhanced meat product. With increased awareness of food-borne illnesses, consumers tend to overcook meat (7). This produces a dry and tough product. Enhanced meats maintain their tenderness even under the most extreme cooking conditions.

Purge is a term used to describe the liquid that is released from raw meat as it ages. Many consumers find this unattractive when purchasing fresh meat and tend to shy away from choosing packages with high purge. These packages may be discarded thus resulting in waste and higher consumer prices. Purge also occurs when frozen meats thaw. Phosphate salt enhancers help to reduce the undesirable purge (7).

Phosphate salts also enhance a meat product's color, retain its moisture, and reduce rancidity from oxidation caused by the metals within the meat (7). Meat enhanced by phosphate salts has a longer shelf life, has a more attractive appearance for consumers, and maintains its moisture if over-cooked.

Finally, retailers can see increased profitability with enhanced meats. As discussed above, enhanced meats have less purge, longer shelf life, and longer lasting color resulting in more products being sold than wasted. Additionally, enhanced meats require little, if any, additional labor to bring it from processor to market. Many enhanced meats are package-ready for display while others require repackaging into individual sales units. Since no traditional butchering of the meat is required, labor costs are lower.

Despite federal guidelines requiring manufactures to include a notification statement of enhancement and a nutrition label, most consumers are not aware that they are purchasing an altered product. The notification statement is usually written in small print and often not noticed by the purchaser. If the product has been repackaged into individual selling portions, the store is responsible for affixing the provided nutrition label on each individual packet. This step may be overlooked and missed (8).

Employees and meat managers may not be certain if the product they are selling is enhanced. Some stores rely on central purchasing for their fresh meat inventory. One week they may receive non-enhanced meat; another week they receive enhanced meat. If the label is not on the individual selling unit, neither the public nor the store knows for certain (9).

There are individuals in the food purveyor industry who are concerned about enhanced meat products. In the words of Jeff Lyons, vice president and senior General Merchandise Manager of Costco, Inc., enhancing meat "is a short cut for manufacturers." He believes that there are alternate ways to provide a quality product without enhancing meat. Though Costco does provide some enhanced meat products, their fresh signature line, Kirkwood®, is not enhanced (10).

The impact of phosphate additives on the nephrology population is far reaching and immense. These phosphorus additives are highly absorbable. In a typical mixed diet of grains, meat, and dairy, approximately 40-60% of the dietary organic phosphates are absorbed, whereas phosphoric acid and other polyphosphates and pyrophosphates (inorganic) are almost 100% absorbed (6). Diets higher in these inorganic salts result in higher phosphorus absorption.

To cover the additional phosphate in the diet, patients must take more phosphate binders. Adherence with a phosphate binder prescription is already a challenge in this population. Increasing a patient's dose or adding an additional binder medication only adds to this challenge for the patient and clinician.

With the concern about calcium load and metastatic calcification, the NKF's K/DOQI Guidelines for Bone Metabolism and Disease recommended limiting the use of calcium based binders (5). Sevelamer hydrochloride increases the dietary acid load that can contribute to metabolic acidosis (11), and lanthanum carbonate use is limited to 3 grams/day (12). Binder therapies are often combined to maximize phosphate binding potency. Unfortunately, this only adds to the financial burden of the patient, as they now must pay for more than one prescription to achieve serum phosphorus control.

As more food comes to the market with phosphate additives, food choices and selection for the CKD population may diminish. Further limiting a renal diet can lead to malnutrition (a known complication within this population), non-adherence with all diet restrictions (which can lead to more life-threatening complications), and a breakdown of trust between the patient and the dietitian (dietitians may be viewed more as the "food police" instead of as a partner in healthcare).

Identifying these new, higher phosphorus foods is very challenging as well as daunting. Manufacturers are no longer required to list the phosphorus content on the food label. If the manufacturer does analyze the product for phosphorus, results are sometimes classified as "proprietary" information. Often the analysis is not readily accessible to customer service representatives who may turn over the request for information to one or more different departments. There is still no guarantee that the company can or will locate the information.

Another challenge faced by professionals and consumers is the practice of products being affiliated with one company and manufactured, packaged, and distributed by another company. For example, Country Time Lemonade® is considered a Kraft®-brand food, but some Country Time Lemonade® products are manufactured and distributed through Dr. Pepper/7-Up®. Also, each company formulates its products differently, and within each product, the individual delivery packages may be formulated differently. That is, the ingredients in a bottled iced tea may be different when compared to the same brand of canned iced tea. Nutrition labels listed on a company's website are not always accurate. When accessing a website for a product believed to be low in phosphorus, this author found a picture of the product's nutrition label that listed the phosphorus content as 0% DV. However, in a different area of the website, which provided a detailed description of the product, the phosphorus content was reported to be 230 mg per serving. When questioned, the company responded that they were not required by law to list the phosphorus content of their product. However, they did acknowledge the error and planned to correct it (13).

Regular monitoring of in-store food nutrition labels is the best way to keep abreast of the nutritional content of foods. In researching another article, this author found problems obtaining accurate analyses from widely used resources available to dietitians and patients. In some instances, there were as many as three conflicting analyses for the same product. In trying to clarify these discrepancies, limitations were found with nutrition analysis resources generally used by the public and the professional (14). In 2006, an effort to bring back the phosphorus content to the nutritional label was launched jointly by the NKF's Council on Renal Nutrition (CRN) and the ADA's Renal Dietitians Dietetic Practice Group (15). Together, ADA and NKF CRN conducted an internet survey of nephrology patients which revealed that more than half of the respondents do not buy a food product or beverage if potassium, calcium or phosphorus amounts are not listed on the label. Roughly 80 percent of the respondents indicated that phosphorus should be included on the label.

The NKF and ADA have shared the results of this survey in the form of comments submitted to the Food and Drug Administration (FDA). This was in response to a Federal Register notice requesting data to consider in updating the nutrition facts label (NFL). These comments are posted at http://www.eatright.org/ada/files/NKFADA_Comments.pdf. The results of this survey will be discussed at FNCE 2008 on Sunday, October 26, 2008, at 1:30 pm in the session entitled, "Dietitians Know Best: What We Told the FDA."

In an earlier conversation with the FDA, NKF CRN and ADA were advised that the NFL is designed to address public health issues-at-large, not for the treatment or purpose of specific disease populations, citing the Institute of Medicine's Dietary Reference Intakes for phosphorus (16). One significant issue is that the Nutrition Labeling and Education Act does not direct the FDA nor does it require phosphorus to be included on the nutrition facts label.

Undeterred by these barriers, efforts to bring "kidney friendly" foods to market are underway. Spearheaded by Dr. William Pordy, founder of the Delicious Milk Company, Inc., there is a pioneering effort to encourage manufacturers to make more kidney

Table 3
Shelf stable products that may be considered for a kidney friendly grocery shelf.
(Note: Use certain products in moderation depending on individual diet prescription.)

TYPE OF FOOD	SPECIFIC FOOD	AVAILABLE BRAND
VEGETABLES	Canned Fruit	SM*
FRUIT	Low Sodium Canned Vegetables:	SM*
	Green Beans, Wax Beans, Peas, Carrots, Corn, Asparagus, Beets	
	Rice, White	SM*
MILK/CHEESE	Milk (low phosphorus, low potassium)	DairyDelicious®
MILK SUBSTITUTE	Cheddar Cheese sauce	
	(very low phosphorus, low sodium)	DairyDelicious®
	Rice Milk (non-fortified)	SM*
CEREALS	Rice and Corn Squares	Chex®, SM*
	Corn Flakes	SM*
	Crisped Rice	SM*
	Puffed Rice	SM*
	Sweetend Cereals:	SM*
	Fruit Loops, Frosted Flakes, Sweetened Puffed Corn, Sweetened Puffed Wheat	
	Grits	SM*
	Rice Cereal - Hot	Cream of Rice®
	Wheat Cereal - Hot	Cream of Wheat®
BREADS	Sandwich Rolls	SM*
	Low Sodium Flour Tortillas	SM*
	English Muffins	SM*
	Hamburger Buns	SM*
	Bread Sticks, Plain	Progresso®
	All Purpose, Bread, Cake Flours (not self rising)	SM*
PASTAS	Macaroni	AM**
	Spaghetti	AM**
	Spirals [Fusilli] and Shells	AM**
	Deluxe Macaroni & Cheese Dinner	
	(very low phosphorus, low sodium)	DairyDelicious®
COOKIES	Animal Crackers	SM*
	Shortbread	Lorna Doone®
	Sugar Cookies	SM*
	Lady Fingers	SM*
	Vanilla Wafers	SM*
	Ginger Snaps	SM*
CAKES	Angel Food, Pound, Sponge, and Lemon Cake	Store Made
CAKES, cont.	Apple, Cherry and Blueberry Pie	SM*
	Doughnuts, Plain	SM*
SNACKS	Unsalted Popcorn, Pretzels, Rice Cakes	SM*
	Unsalted Crackers and Melba Toast	SM*
	Jelly Bean	SM*
	Marshmallows	SM*
	Gummies	SM*
	Sugar Free Chewing Gum	SM*
SEASONINGS, SPICES and SPREADS	Sweeteners	White Sugar, Splenda®, Saccharine, Aspartame
	Salt Replacers	Mrs. Dash®
	Pepper, Onion and Garlic Powder	AM**
	Cilantro, Oregano, Dill, Basil, Sage, Paprika,	
	Cinnamon, Nutmeg, Parsley, Rosemary, Curry	AM**
	Mayonnaise and Spreads (not low fat or fat free)	SM*
	Yellow Mustard, Organic Mustard	French's®, Annie's Naturals®
	Low Sodium Salad Dressings	SM*
	Low Sodium Bread Crumbs	SM*
	Hot Sauce	Frank's®, Tabasco®, Diamond®
	Lemon Juice	SM*
	Vanilla, Orange and Almond Extract	SM*
OIL and VINEGAR	Vegetable Oil	SM*
	Olive Oil	SM*
	Corn Oil	SM*
	White Vinegar	SM*
	Balsamic Vinegar	SM*
	Wine Vinegar	SM*
PUDDINGS, TOPPINGS and SYRUPS	Whipped Topping Mix - Dry	Dream Whip®
	Sugar Free Gelatin	SM*
	Light Chocolate Syrup	Hershey®
BEVERAGES	Coffee, Decaffeinated Coffee	AM**
	Regular and Diet Lemon-Lime Soda	AM**
	Regular and Diet Ginger Ale	AM**
	Regular and Diet Root Beer	A&W®
	Orange, Grape and Cherry Drink	Kool-Aid®, Crystal Light®
	Apple, Cranberry, and Grape Juice	SM*

Murphy-Gutekunst L. Hidden Phosphorus: Where Do We Go From Here? *J Ren Nutr.* 17(4): E31-E36, 2007.

friendly food products. Also, enticing food retailers to dedicate a "Kidney Friendly Shelf" in their retail stores would be a win-win arrangement. These would include products that are lower in phosphorus, sodium, potassium, and calcium. Patients with CKD win by having one convenient location to find foods that are appropriate for them and good for kidney health. The grocer wins by keeping current customers, gaining new ones, and delivering products that "address the current issues of condition specific nutrition," says Dr. Pordy (17). Dr. Joseph Vassalotti, Medical Director for the NKF concurred. He stated, "Kidney healthy foods would be an attractive way to avoid the challenge that food labels designed for the general population pose for CKD patients" (18). The food industry is driven by consumer demand, thus information regarding the need and desire for kidney friendly foods must get to industry leaders. Food retailers who have a high demand for these products will in turn ask for more kidney friendly foods from the manufacturer. Retailer information regarding the "Kidney Friendly Shelf," (Figure 1, which can be accessed via www.renalnutrition.org under CPEU Inserts or RNF Archives) and a list of kidney friendly foods (Table 3) to be included on this shelf have been included in this article as tools for healthcare professionals and CKD patients to use.

Education and creative repetition remains the key to serum phosphate control. Empowering the CKD population with knowledge can make a difference when they are faced with limited information about food choices. Without understanding the dangers of foods once thought to be "safe," the population cannot take further control of their healthcare and overall health. Education materials listing hidden phosphorus products can be displayed in waiting rooms. Low literacy posters showing pictures of new high phosphorus foods to limit can be utilized to reach those who cannot read nutrition ingredient labels. "Safe" food lists and pictures offer useful alternatives, and encourage variety and choice among patients.

In addition to these tools, healthcare professionals need to continue educating other healthcare providers on hidden dietary sources of phosphorus. The dietitian is only one part of a CKD patient's healthcare team. Nurses, dialysis technicians, social workers, and physicians can reinforce the dietitian's educational efforts, and aid the dietitian in gaining a more thorough understanding of a patient's lifestyle and needs.

Winning the war on phosphorus control is gained through each individual battle. It is a slow process, but one that benefits the lives of those who we serve as registered nephrology dietitians. ◆

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Nutrition Management of a Chronic Kidney Disease Patient with Obesity and Diabetes: A Case Study

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Nutrition care for patients with chronic kidney disease (CKD) involves continuous assessment of numerous nutritional and medical factors. Appropriate nutrition care can help patients maintain good health, and possibly prolong the time before renal replacement therapy (RRT) is initiated. In addition, bone and mineral metabolism control can be enhanced, dangerous alterations in serum potassium levels can be avoided, and fluid retention can be minimized (1). Nutrition intervention can also help patients avoid the protein-energy malnutrition (PEM) that often accompanies late stages of CKD (2,3).

If a patient also presents with diabetes and obesity, the complexity of nutrition care increases and priority-setting becomes vital. Similar to healthy individuals, CKD patients who are overweight or obese may improve blood pressure and cholesterol with weight loss of 5-10% (1). Weight loss may reduce both cardiovascular risk and risk of progression of CKD (4,5). However, it can be challenging to implement weight loss strategies along with the dietary modifications needed to help manage the metabolic alterations that occur in CKD. The following case illustrates an approach to the dietary management of a patient with diabetes and class III obesity.

Case Presentation

CJ was a 77 year old female with obesity (BMI >40 kg/m^2), type 2 diabetes and CKD stage 5 (not on dialysis). Medical history included partial parathyroidectomy for secondary hyperparathyroidism (SHPT), hypertension, cardiovascular disease and hypothyroidism.

CJ appeared well-nourished, despite her late stage of renal failure. Her weight was 251 pounds, and at her height of 5'3", her BMI was 44.5 kg/m^2. This is considered class III, or extreme obesity (6). Physical assessment indicated mild wasting of quadriceps and calf muscles, with mild edema present at the ankles. Decreased skin turgor was noted, indicating some degree of dehydration. In view of her decrease in leg muscle mass, it was not surprising that CJ had poor balance and poor endurance. She relied on a walker for ambulation. CJ was short of breath and easily fatigued. This was in part attributed to both iron and red blood cell deficiencies, as a consequence of the anemia of chronic kidney disease. As a result of her fatigue and obesity, CJ's activity level was very low. Despite the partial parathyroidectomy to correct her SHPT, her serum calcium was still above desirable levels for CKD. Her albumin of 3.9 g/dL did not point to a state of inflammation, yet the CO_2 of 16 mEq/L indicated presence of metabolic acidosis, which can lead to protein catabolism. Her potassium (K) was slightly elevated. Her HbA1C of 5.3%, along with her reports of fasting glucose of 90-100 mg/dL indicated excellent glycemic control. Urinary albumin excretion was not available.

Prior to her visit to the clinic, CJ was on a commercial weight loss program and lost 10 pounds. This program used a macronutrient distribution of 45% CHO, 35-40% protein and 20% fat (7). Because the counselors had no experience with adjusting her diet for kidney failure, there were no modifications made for the metabolic alterations related to her failing kidneys. Although CJ was pleased with her weight loss, she was more interested in following diet recommendations that would be appropriate for her kidney condition.

Her usual dietary habits included a simple breakfast at home (toast and fruit or a bowl of cereal), frequent restaurant lunches, and take-out or purchased pre-prepared foods for supper. Her usual diet was very high in sodium, and she often consumed baked goods and candy. She had received diabetic diet education several years earlier, but did not count carbohydrates or follow a meal pattern with exchanges. On the weight loss program, she had reduced her portions by sharing her restaurant lunch entrees with her daughter (who spent her days with CJ), and she had replaced evening sweets with fruit. She limited some foods high in phosphorus (P) upon her nephrologist's advice. CJ experienced early satiety, and noted a decreased appetite for meat.

CJ was not taking any vitamin or mineral supplements with the exception of Oscal with vitamin D. She did not take any nutritional or herbal supplements, and did not drink alcohol or smoke.

Treatment of CJ's anemia, caused by deficient production of erythropoietin and by iron-deficiency, was a primary reason for her referral to the CKD clinic. She was started on darbepoetin alpha (Aranesp) and iron sucrose (Venofer) to correct her iron deficiency, which, as noted in Table 1, was evidenced by a serum ferritin of 46 ng/mL.

Medications are listed in Table 2. CJ was on several medications commonly employed in patients with CKD to help control blood pressure, limit fluid retention and reduce proteinuria. These included furosemide (Lasix), valsartan (Diovan) and verapamil

Table 1
Laboratory Values

Year	Desired Values*	Date	Date	Date
Date		3/2/05	5/11/05	11/9/05
Weight	pounds	251	243	240
BMI	kg/m^2	44.5	43	42.5
Creatinine	mg/dL	3.9	3.5	3.0
BUN	mg/dL	97		
Estimated GFR	mL/min/1.73 m^2	12	14	16
CO_2	>22 mEq/L	**16**	**16**	24
Albumin	>/=4.0 g/dL	**3.9**	4.0	4.0
K	3.5-5.0 mEq/L	**5.1**	4.6	4.7
Calcium (corrected)	8.4-9.5 mg/dL	**10.5**	**9.8**	**9.9**
Phosphorus	3.5-5.5 mg/dL (stage 5)	5.2	4.0	4.8
Ca x P product	<55	54.6	39.2	47.5
PTH (bio-intact in 2005)	75-150 pg/mL for stage 5**	64	**192**	
Hgb	11-12 gm/dL	**9.2**	11.1	12.3
Transferrin Saturation (TSAT)	20-50%	21		21
Ferritin	100-800 ng/mL	**46**		390
HbA1C	<7%		5.3	
Blood Pressure	<130/80	132/60	128/64	
Cholesterol	mg/dL	159		
HDL Chol.	mg/dL	52		

*represent norms or desired values based on K/DOQI guidelines and/or institution guidelines. Patients with CKD do not achieve normal values for creatinine, BUN or GFR.
**Institution goals in 2005 were for bio-intact PTH: stage 4: 35-55 pg/mL; stage 5: 75-150 pg/mL

(Isoptin-SR). Her secondary hyperparathyroidism was managed with adjustments in the pro-active vitamin D doxercalciferol (Hectorol). Metabolic acidosis was managed with the use of sodium bicarbonate. This medication increases total sodium retention; thus dietary sodium restriction was warranted.

The nutrition strategy for this patient was to develop a meal plan that would help control blood pressure, reduce edema, and maintain appropriate serum K levels. Additionally, the plan's intention was also to normalize P and calcium balance so as to minimize exacerbation of the SHPT. CJ was instructed in a diet with 1800 calories, 48 gm protein, 2000 mg sodium, 800 mg P and 2730 mg K. Her calorie level was matched to that of the weight loss program on which she had been losing two pounds per week. Protein was restricted to 0.6 gm/kg desirable body weight, per K/DOQI guidelines for stage 5 CKD, but not on dialysis. This was to help minimize uremic waste products and potentially slow the progressive nature of her kidney failure (8). Sodium was restricted to help minimize fluid retention.

Although her serum P was not elevated, the dietary P restriction was continued as previously advised by her nephrologist. A level of 800 mg was prescribed, per K/DOQI guidelines (9). Calcium carbonate (OsCal), usually prescribed to bind P, was held due to the high serum calcium of 10.5 mg/dL. This presented another reason to limit dietary P intake, as no replacement phosphate-binder was prescribed. Finally, K was limited to approximately 70 mEq/day (2730 mg/day) to restore serum K levels to a normal range. Dietary measures to optimize lipid levels were also incorporated into the meal plan. To help maintain acceptable glycemic control, distribution of carbohydrate was discussed, along with food choices, portions and meal-timing. Continued weight loss was also encouraged, but not at the expense of impairing her nutritional status. Emphasis was placed on consuming adequate protein and the prescribed calories daily. A renal vitamin was prescribed, as patients on controlled-protein diets may need vitamin supplementation (9).

Selecting a weight to use for determining energy and protein needs is one of the most perplexing challenges in caring for the obese patient with CKD. As discussed further in this article, there are no validated methods for determining energy requirements in CKD patients. At this time, it is practical to make determinations based on the K/DOQI practice guidelines, also taking into consideration "the patient's values and preferences in the overall treatment plan (10)." Routine monitoring of a patient's progress and outcomes must serve as a guide for adjusting diet parameters (10). K/DOQI guidelines suggest using an "idealized body weight" for obese patients (11). When calculating needs for obese patients, adjusted body weight has historically been used, despite its lack of scientific foundation. Consistent with general practice at the CKD clinic at the time, both standard body weight (SBW) and adjusted body weight were considered in determining the body weight to use for calculations in obese patients. CJ's actual body wt was 114 kg. The SBW was

Table 2
Medications

Medication	Indication	Comments
Aranesp (darbepoetin alpha) 100 mcg Q 2 weeks	Anemia	
Dialyvite 800 1/day	Multivitamin	Contains water-soluble vitamins only
Diovan (valsartan), 80 mg bid	Angiotensin receptor blocker, relaxes blood vessels.	
Glucotrol (glipizide) AM: 10 mg PM 10 mg	Oral hypoglycemic agent	May lead to hypoglycemia if meals are skipped; can promote weight gain; needs close monitoring in pts with CKD
Hectorol (doxercalciferol) 0.5 mcg (begun in late 2005)	Active Vitamin D analog	Increases calcium absorption, reduces PTH stimulation Started in 2005; held in Oct 2007 as calcium levels were in high 9's
Lasix (furosemide) 80 mg and 160 mg alternating nights	Diuretic	Enhances K losses. Need to monitor K levels and adjust diet or provide K supplement if serum K too low.
Lipitor (atorvastatin) 20 mg	HMG-CoA reductase inhibitor, used to help lower cholesterol and TG	Grapefruit juice can increase levels in the blood.
Oscal with D (March 2005 only)	Calcium supplement and which can also perform as P-binder	Can raise calcium levels. Vitamin D in this form unlikely to be converted to active form in failing kidneys.
Sodium bicarbonate 650 mg tab 2 bid	Prevent and treat acidosis caused by kidney disease.	Contributes to total sodium intake
Synthroid (levothyroxine) 88 mcg	Hypothyroidism	Should be taken separately from calcium supplements, iron supplements, and antacids.
Venofer (iron sucrose) 200 mg q 2 weeks I.V.	Fe deficiency	Oral iron usually not adequate to meet needs in depleted CKD pt; thus IV Fe used.
Isoptin-SR (verapamil SR) 240 mg 1.5 tabs/day	Calcium channel blocker. Lowers blood pressure and reduces workload on heart, allowing it to work with less oxygen and blood flow.	

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70 kg, based on a medium frame size. Her adjusted body weight, using the K/DOQI method was 103 kg. These numbers were very far apart. Using too high a weight can result in overestimation of protein needs (11). A value between these numbers, 80 kg, was chosen, in part because this was the weight CJ reported being at when she was in her 30's.

Using this weight, her protein requirements were calculated at 48 grams/day and calorie needs at 2400 kcal/day (30 kcal/kg) (12). However, a pattern for 1800 calories was created, to help the patient continue with her weight loss efforts.

Implementation

To support her dietary change efforts, over the course of several visits, CJ was provided with *Dining Out with Confidence: A Guide*

for Kidney Patients (13), sample menus (developed with her input), and lists of high biological value protein, sodium, P and K food sources. Portion sizes were discussed, as well as strategies for continued weight loss.

During the time CJ was a patient at the clinic, her creatinine decreased from 3.9 to 3.0. Her anemia was corrected (see Table 1), and she reported that her energy and sense of well-being increased. She began to moderately increase her activity upon staff recommendations, but was limited due to her reliance on a walker. Dietary recalls indicated good adherence with restrictions in protein, phosphorus and potassium. Her sodium intake continued to be above recommended, as she continued her dependence on restaurant meals. Her calorie intake was variable and often greater than prescribed. Nevertheless, CJ had some success with continued weight loss, and reached 240 pounds by the end of 2005. Her nutritional status remained stable and albumin was maintained at 4.0 g/dL for several months.

Discussion

Obesity and CKD

Obesity may be the number one risk factor today for developing CKD, primarily because it is a risk factor for diabetes and hypertension (14). Together, diabetes and hypertension contribute to approximately 60% of the patients who develop CKD (15). Obesity may also have a direct impact on impairing renal function (14). Obesity increases the metabolic demands on the kidney, resulting in increased glomerular filtration rate (GFR) and renal plasma flow (RPF) and renal hypertrophy (14,16). As GFR and RPF rise, so does intracapillary pressure (14). Hyperfiltration and glomerulomegaly may result in glomerular damage and albuminuria in the severely obese (17).

Kambhan *et al* termed this condition obesity-related glomerulopathy (ORG). The course of ORG is somewhat indolent, and may not always progress to CKD stage 5. Although proteinuria is significant in over half the patients, hypoalbuminemia is present in only about 14% of patients. Kambhan *et al* reviewed 121 renal biopsies, including 71 patients with ORG and 50 patients with idiopathic focal segmental glomerulosclerosis. They found that the patients with ORG had a lower incidence of nephrotic syndrome (5.6% vs. 54%; $p<0.001$), higher serum albumin (3.9 vs. 2.9 g/dL; $p<0.001$) and less edema (35% vs. 68%; $p=0.003$). The mean BMI in ORG was 41.7. Kambham *et al* noted that the incidence of (ORG) increased from 0.2% in 1986-1990 to 2.0% in 1996-2000 (16). This parallels the increase in rates of obesity seen in the United States.

Lifestyle modifications that include weight loss and physical activity are recommended for the general public for improving blood pressure, blood glucose and lipid levels (4). Chagnac *et al* found improvements in glomerular hemodynamics in obese patients without overt renal disease following weight loss. Their findings suggest that weight loss may be helpful in reducing the metabolic demands on the kidney and potentially delaying progression of renal disease (17).

Diabetes and CKD

Diabetes is a primary cause of CKD with the first clue of diabetic nephropathy often being the presence of microalbuminuria (18). Glycemic control has long been emphasized as a goal in managing diabetes and preventing complications such as nephropathy. However, the level of protein in the diet may also play a significant role. High protein intakes promote glomerular hyperfiltration and increase pressure in the glomeruli. In contrast to the general population, higher protein intakes in diabetics may have more deleterious effects on kidney hemodynamics (11), and dietary protein limitation may help slow the loss of kidney function. "Competing needs for nutritional management of hyperglycemia, hypertension, and dyslipidemia can make determination of appropriate protein intake challenging," but even a modest decrease in protein intake to 0.89g/kg/d may be of benefit in reducing the progression to stage 5 CKD (11). A level of 0.6 to 0.8 gm protein/kg/day for CKD stages 3 and 4 with diabetes is recommended (11). Of this protein, 50-75% should be of high biological value (HBV), ideally from lean poultry, fish, and soy-based and vegetable-based proteins (11). "Protein of high biological value has an amino acid composition that is similar to human protein, is likely to be an animal protein, and tends to be utilized more efficiently by humans to conserve body proteins. The increased efficiency of utilization of high biological value protein is particularly likely to be observed in individuals with low protein intakes" (19).

With low protein levels, calories from protein may add up to only about 10% of total calories. To achieve adequate energy intake, non-protein calories should be derived from fat (~30% of calories) and carbohydrates (up to 60% of calories) (11). As patients reduce their protein intake, they may be increasing their baseline carbohydrate intake, leading to an increased need for insulin. Medications for glycemic control may need adjustment. Whenever possible, the patient should be encouraged to increase activity to improve insulin utilization and promote weight loss/prevent weight gain.

Carbohydrates that are considered low-glycemic should be emphasized to help decrease postprandial hyperglycemia and improve overall glycemic control (11) . Incorporating both soluble and insoluble fiber into the diet may help lower CVD risk (4); however, consideration needs to be given to the phosphorus and

potassium content of high fiber foods that are implemented in the diet.

Estimating Requirements for the Obese CKD Patient

The Metropolitan Life weight tables, which were developed to present weights associated with lowest mortality, were used in this population in the past, but no longer represent many Americans, as the data has tended to under represent those who do not purchase life insurance (20). The Hamwi optimum weight formula is familiar to most dietitians (20), but has no scientific data supporting its use. The NHANES II (National Health and Nutrition Evaluation Survey) SBW tables, recommended by K/DOQI, are not related to health outcomes. They also require measurement of frame size, which may not be feasible in some clinic settings, and they do not include values for adults over the age of 74 (20). The use of a formula for adjusted body weight in the obese also lacks research support (20).

A definitive approach is not currently available to estimate calorie requirements in the CKD population. At this time, the practitioner must rely on predictive equations. A survey of renal dietitians' practices conducted in 2005 illustrates the fact that there are no standardized methods employed across the country for assessing weight or determining calorie requirements in patients with CKD (21), though a large percentage of renal dietitians use the kcal/kg equation published by K/DOQI (21). This K/DOQI guideline is 35 kcal/kg/day for patients younger than age 60, and 30-35 kcal/kg/d for patients age 60 and older (12). Obese pts should be managed with lower calorie levels (1). In the end, the "practitioner must adopt a 'reasonable' approach," and let monitoring of patient's progress and outcomes provide a guide for adjusting medical nutrition therapy (10).

Conclusion

The number of patients with CKD and obesity is rising, in parallel with the increased incidence of obesity in the general public. Questions regarding the optimum treatment approaches in these patients will need to be addressed. For now, it appears that in early CKD (stages 1-3), weight loss should be aggressively pursued, as it may improve the hyperperfusion/hyperfiltration that accompanies obesity (17). However, the overweight and even obese patient with late stage CKD may have survival advantages once RRT has begun (22). It is unclear whether a higher BMI provides a survival benefit to the patient with a later stage of CKD, and not on RRT (23). Thus, weight loss in later stages of CKD must be addressed on an individual basis, taking into consideration the patient's personal goals, co-morbidities, their ability to make changes in activity level and food intake, and their future medical goals for RRT. Presently, no K/DOQI guidelines specifically address the treatment of obesity in CKD. Thus, recommendations from existing K/DOQI guidelines should be employed. Regardless of weight, every patient with CKD should receive close follow-up, re-education and adjustment of nutrition therapy as their clinical condition changes. ◆

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Have You Thought About Becoming a Provider?

Aimee Zajc, RD, LD
Editor *Renal Nutrition Forum*

I was fortunate to attend the Medicare 2008 Workshop on May 3, 2008 in Schaumburg, IL on behalf of the Renal Practice Group. It was entitled *Hot Topics for Medicare RDs: Effectiveness and Efficiency in 2008*. The presenters included Pam Michael, MBA, RD, ADA Nutrition Services Coverage (NSC) Team Director, Tori Bender, MSJ, NSC Communications Manager, professional biller Denise A. Alessi, CMA, CPC, CMM and ADA/RPG member and nephrology nutrition specialist Carolyn Cochran, RD, LD, MS, CDE. Attendees ranged from dietitians who were considering becoming a provider to those who had been providers for years. Topics included Medicare 101, policies, documentation considerations and billing concerns. Personally, I have considered becoming a provider, but was not familiar with how to do this before I attended the workshop.

Medicare Part B includes coverage of medical nutrition therapy (MNT) by a licensed or certified registered dietitian or licensed nutrition professional for patients with diabetes mellitus (DM), gestational DM, chronic kidney disease (CKD) with glomerular filtration rates 13-50 mL/min/1.73m² (pre-dialysis), and post renal transplant. Note that there are practice settings where MNT Medicare Part B benefits may not be separately billed. These locations where Medicare Part A services and payments are made such as: hospital inpatient settings, skilled nursing facilities, dialysis centers, home care (Medicare part A), and hospice.

How do you enroll?

1. Obtain a national provider number (NPI). The purpose of the NPI is to uniquely identify a health care provider in standard transactions, such as health care claims. Note that if you are enrolled in Medicare but have not submitted updated forms since 2003, you are required to submit an application. See the Centers for Medicaid and Medicare services (CMS) instructions. http://www.cms.hhs.gov/cmsforms/downloads/CMS10114.pdf

2. Complete Medicare enrollment application(s). There are up to 3 forms that you may need to fill out. It is vital to contact your local Medicare state carrier for a Medicare provider enrollment packet. Before downloading or printing enrollment application forms available online, call your local Medicare Part B carrier (Medicare intermediary) to verify which provider/supplier forms you should complete. If you are employed at a clinic or facility that will submit Medicare Part B claims forms and collect payment for your services, you will also need to complete a reassignment of benefits enrollment form. For your state contacts go to: http://www.eatright.org/ada/files/State_Carrier_Information_(2).pdf. For the CMS website, go to http://www.cms.hhs.gov/MedicareProviderSupEnroll/ "Application Forms and Instructions." Billing office personal may have experience filling out these forms.

3. Once you are enrolled, before providing MNT to qualifying seniors, you must obtain a referral from a treating physician. Nurse practitioners and physician assistants cannot send a Medicare MNT referral. The physician must include a specific diagnosis with a covered ICD-9 code, MD signature, and document in the patient's medical record the medical necessity for MNT services. An order that reads "see RD" is not enough. Maintain a copy of the referral in your files.

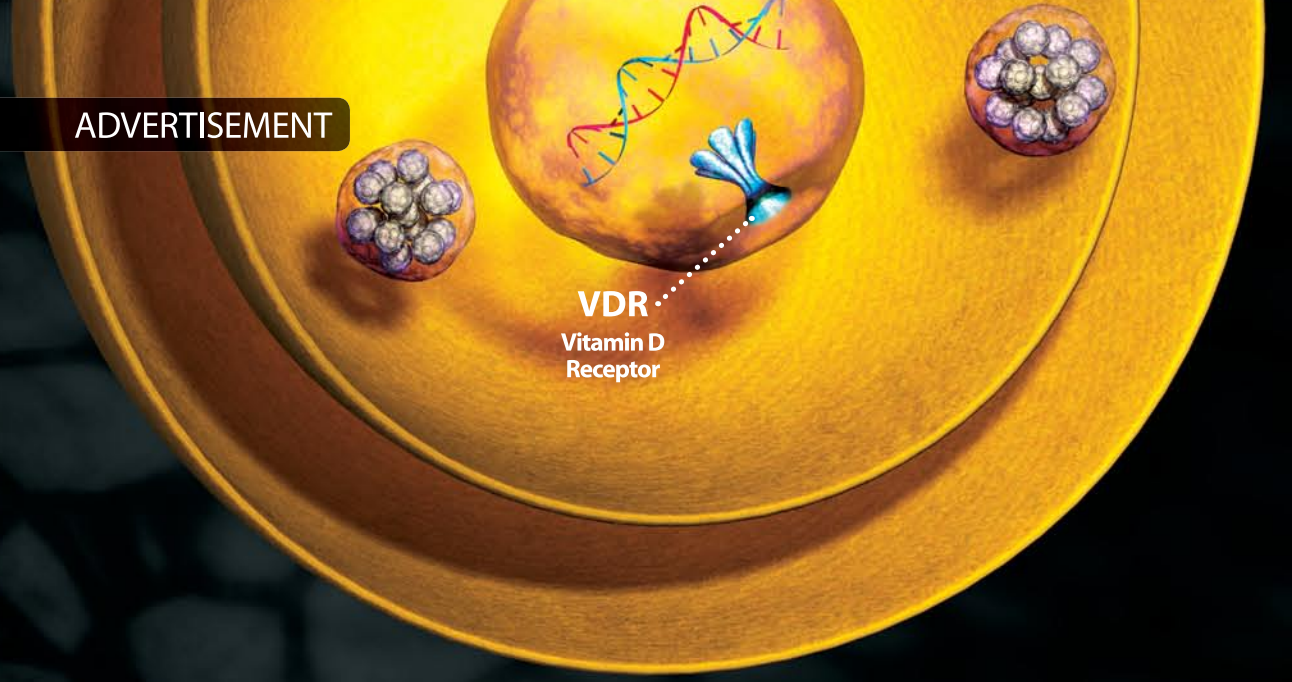
4. Basic coverage (year 1, per calendar year) = 3 hours. A 12-month period is called an "Episode of Care". Follow-up (year 2 and subsequent years) = 2 hours. Additional hours may be ordered if considered medically necessary by the treating physician. Examples: A patient may demonstrate a lack of understanding of the renal diet or they experience a significant clinical decrease in renal function. A patient with diabetes could develop CKD. An additional physician referral with chart documentation is needed for additional hours of MNT in the same calendar year, or for MNT in year 2 and subsequent years.

5. MNT CPT codes must be utilized by RD Medicare providers. Codes are billed in units of 15 minute increments for individual MNT, or 30 minute increments for group MNT classes.

A few practice tips:

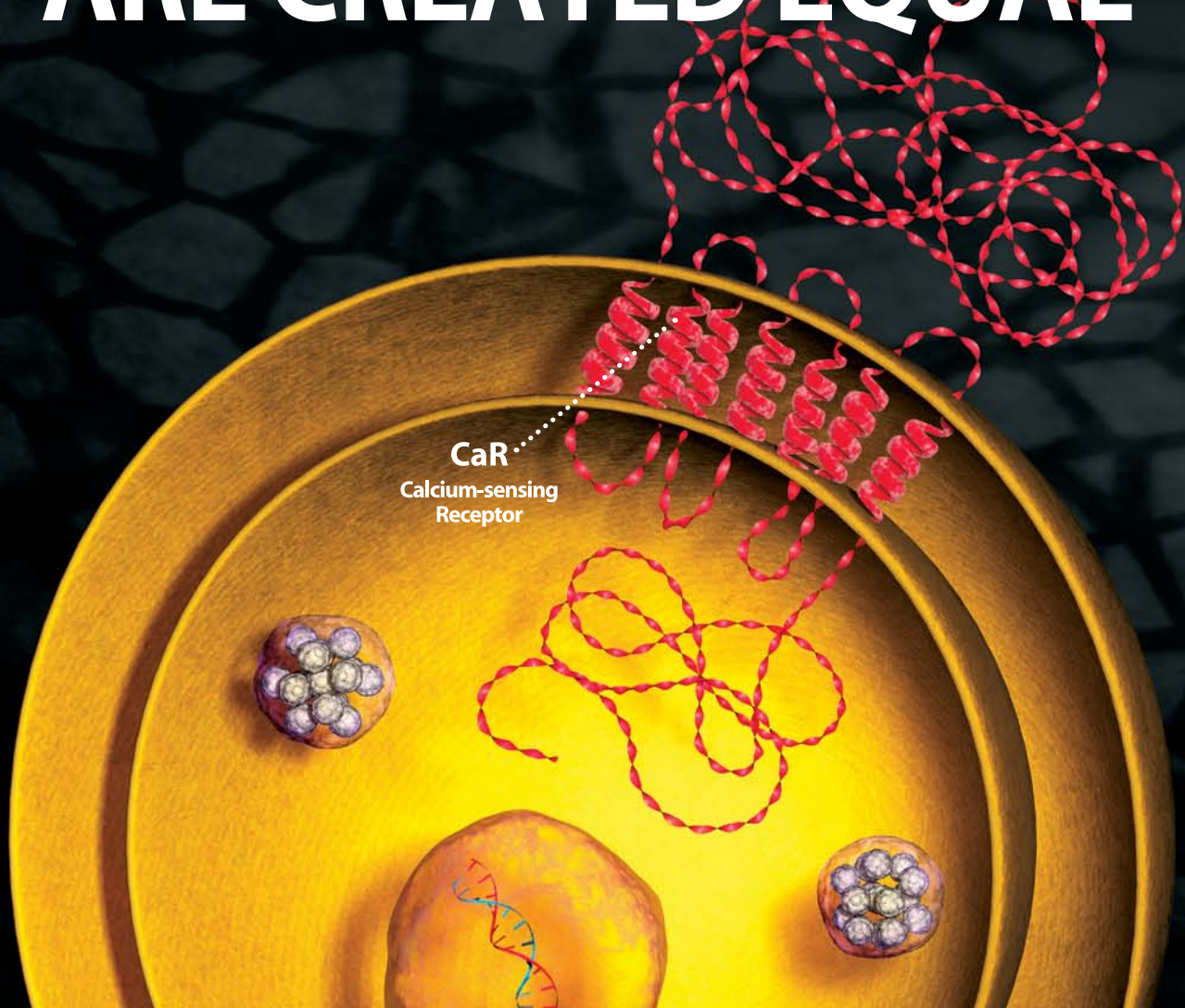
- Don't assume all individuals >65 years old are covered under Medicare Part B. Also, double check that the Medicare card matches the name of your patient. Disabled persons <65 years of age may also be Medicare beneficiaries.

- Make copies of the Medicare card and patient's photo identification card. Keep copies on file. You must also have a signed agreement on file, a "signature on file," that confirms the patient has received MNT services, and this must be signed prior to billing for the service.



In secondary HPT,

NOT ALL RECEPTORS ARE CREATED EQUAL



Data show that the vitamin D receptor and the calcium-sensing receptor play independent roles in the pathogenesis of secondary HPT

Secondary hyperparathyroidism (HPT) begins at early stages of chronic kidney disease and becomes increasingly severe over time.[1,2] Disease progression is characterized by parathyroid gland hyperplasia—defined as cell proliferation—and gland enlargement.[3,4] It is crucial, therefore, to understand the factors that mediate parathyroid gland hyperplasia and its role in disease progression.[3-7]

Calcium, acting through the calcium-sensing receptor (CaR), and vitamin D, acting through the vitamin D receptor (VDR), have diverse effects in a variety of tissues[8] and independently impact parathyroid gland function.[4-6,9] Vitamin D directly diminishes parathyroid hormone (PTH) gene expression and hormone synthesis and indirectly reduces PTH synthesis and secretion by raising blood calcium levels.[7,10] In contrast, calcium signaling through the CaR directly inhibits PTH secretion and reduces PTH gene expression.[3,6-8]

Moreover, recent evidence suggests that signaling through the CaR is a key determinant of parathyroid gland enlargement and cell proliferation.[3,6] Findings from preclinical studies by Li et al suggested that calcium-dependent signaling through the CaR was sufficient to prevent parathyroid gland hyperplasia even in mice lacking a functional VDR whose tissues cannot respond to vitamin D.[6,11]

Research suggests that there are 2 independent pathways involved in the pathogenesis of secondary HPT.[5,12] Signaling through the VDR inhibits PTH gene expression and hormone synthesis[12] while signaling via the CaR affects PTH secretion, PTH synthesis, and parathyroid cell proliferation[3,6,12]—the last impacting parathyroid gland hyperplasia.[3,6,8]

References: 1. Martínez I, Saracho R, Montenegro J, Llach F. The importance of dietary calcium and phosphorous in the secondary hyperparathyroidism of patients with early renal failure. *Am J Kidney Dis*. 1997;29:496-502. **2.** Goodman WG. The consequences of uncontrolled secondary hyperparathyroidism and its treatment in chronic kidney disease. *Semin Dial*. 2004;17:209-216. **3.** Rodriguez M, Nemeth E, Martin D. The calcium-sensing receptor: a key factor in the pathogenesis of secondary hyperparathyroidism. *Am J Physiol Renal Physiol*. 2005;288:F253-F264. **4.** Goodman WG. Calcium and phosphorus metabolism in patients who have chronic kidney disease. *Med Clin North Am*. 2005;89:631-647. **5.** Goltzman D, Miao D, Panda DK, Hendy GN. Effects of calcium and of the vitamin D system on skeletal and calcium homeostasis: lessons from genetic models. *J Steroid Biochem Mol Biol*. 2004;89-90:485-489. **6.** Chen RA, Goodman WG. Role of the calcium-sensing receptor in parathyroid gland physiology. *Am J Physiol Renal Physiol*. 2004;286:F1005-F1011. **7.** Silver J, Kilav R, Naveh-Many T. Mechanism of secondary hyperparathyroidism. *Am J Physiol Renal Physiol*. 2002;283:F367-F376. **8.** Brown EM, Pollak M, Hebert SC. The extracellular calcium-sensing receptor: its role in health and disease. *Annu Rev Med*. 1998;49:15-29. **9.** Cañadillas S, Canalejo A, Santamaría R, et al. Calcium-sensing receptor expression and parathyroid hormone secretion in hyperplastic parathyroid glands from humans. *J Am Soc Nephrol*. 2005;16:2190-2197. **10.** Silver J, Naveh-Many T, Mayer H, Schmeizer HJ, Popovtzer MM. Regulation by vitamin D metabolites of parathyroid hormone gene transcription in vivo in the rat. *J Clin Invest*. 1986;78:1296-1301. **11.** Li YC, Amling M, Pirro AE, et al. Normalization of mineral ion homeostasis by dietary means prevents hyperparathyroidism, rickets, and osteomalacia, but not alopecia in vitamin D receptor-ablated mice. *Endocrinology*. 1998;139:4391-4396. **12.** Goodman WG. Calcium-sensing receptors. *Semin Nephrol*. 2004;24:17-24.

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Medicare Part B 101...

- Even if you are not filling out billing forms directly, you are responsible for knowing the rules and regulations of the billing process. Billing can become time consuming based on multiple discussions from RDs with experience. You may consider hiring a biller to assist with this, or working in an environment where you have assistance with billing.

- Consider using an Advanced Beneficiary Notice (ABN) if extra hours of MNT for diabetes and CKD are provided in the same calendar year. Since there could be instances where the extra MNT hours might be questioned by CMS, and payment potentially could be denied, an ABN signed by the patient allows the RD to collect payment from the patient. (See www.eatright.org/mnt for more information on ABNs.)

- Co-pays, co-insurances and deductibles must be collected from the patients.

- RD payment for MNT services are listed in the Medicare physician fee schedule. Look on www.eatright.org/mnt for the RD specific fee schedule. Medicare pays 80% of the total, and the patient pays a 20% co-pay. The patient is also responsible for the Medicare annual deductible of $135.00 (for 2008).

- Document well. An auditor will evaluate why a visit took a specified amount of time and what was covered in the visit.

- MNT providers must utilize nationally recognized protocols (example: ADA MNT Evidence Based Guides for Practice) and collect outcomes data.

- Be sure to add your name to ADA's list of nutrition network providers. Be aware that providing nutrition services for free may be considered fraud.

Where can you go for help?

Contact your local Medicare Administrative Contractors (carrier) for Medicare provider enrollment questions. The carrier's names, phone numbers and addresses are listed on the ADA website www.eatright.org/mnt. In addition, the ADA website also includes Medicare information, and a frequently asked questions section to assist members. Refer to the ADA Medicare MNT Provider newsletter, Journal, Reimbursement Community of Interest and On the Pulse for additional Medicare information. Network with others who might have more experience.

The ADA Coding and Coverage Committee and staff from the Nutrition Services Coverage Team provide resources to members on codes, coverage and MNT practice. They are also sponsoring the Chicago FNCE 2008 session: "Billing and Coding 101: Tips for Successful Billing," on Sunday 10/26, 1:30 - 3:00 PM.
***Please see additional details in this publication for the RPG sponsored pre-FNCE workshop in Chicago this fall.
This discussion is meant to be an introduction to Medicare part B coverage. Please contact ADA, CMS, and local state contacts for more details. ◆

Which Patients Qualify for MNT per CMS Regulations?

CMS defines diabetes as: a condition of abnormal glucose metabolism diagnosed using the following criteria: A fasting blood sugar greater than or equal to 126 mg/dL on two different occasions; a 2 hour post-glucose challenge greater than or equal to 200 mg/dL on 2 different occasions; or a random glucose test over 200 mg/dL for a person with symptoms of uncontrolled diabetes.

- CMS regulations indicate a beneficiary who has been discharged from the hospital after a successful renal transplant within the last 6 months is eligible to receive MNT.

- Gestational diabetes is any level of glucose intolerance with onset or first recognition during pregnancy.

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A Renal Nutrition Forum Series with Practice-Based Examples of the NCP

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The nutrition diagnostic statement, or PES statement (P=Problem; E = Etiology; S = Signs and symptoms), has become the cornerstone of documenting nutrition diagnosis(es) using the standardized language (SL) for dietetics and nutrition. See Table 1 for a description of the various parts of the PES statement and a sample statement. (This is one example, independent of the case study that appears later in this article.)

The annual publication of updated reference manuals for SL demonstrates the continually evolving nature common to all phases of SL. Nutrition diagnosis terms were first introduced in September 2005 (1). Two years later, the International Dietetics and Nutrition Terminology (IDNT) Reference Manual included a revised list of nutrition diagnosis terms, reducing the list from its original 62 terms to 60 terms (2). Changes were based on research findings and on input received through a defined process for terminology maintenance and revision.

Composing a PES statement may be quite challenging initially. However, a well-written statement clearly and concisely conveys the dietetics professional's nutrition care plan for the specified patient. Therefore, an understanding of the purpose of each component of PES statements is important.

A few pointers about PES statements:

- The time it takes to write these statements is significant initially, but will decrease as the learning process advances. A good PES statement presents a succinct yet focused picture of the individual patient, and is worth the time invested.
- The PES statement creates a helpful context that helps the clinician to identify intervention.
- Finally, a clear and concise PES statement reflects the clinician's most sophisticated critical thinking skills. The PES statement presents the nutrition diagnosis in a context that demonstrates to others within health care team the diagnostic thinking involved in the NCP.

Most dietitians were not taught how to formulate diagnosis as a fundamental clinical skill, certainly not in the manner in which this skill is conveyed in medical education. In a 2006 New England Journal of Medicine (NEJM) article, Bowen compares diagnostic reasoning by a novice resident with that by an expert resident. The article provides examples of the language used by each after interviewing the same patient for the purposes of developing a list of differential diagnoses (3).

The contrast this article describes between individuals at two different levels of skill development provides an excellent example of what dietetics students and practitioners are encountering as they progress through various levels of experience with nutrition diagnosis in the NCP model. There is every reason to expect that the novice resident in the NEJM article will achieve a higher level of diagnostic thinking with continued practice. Eventually she/he will be able to express findings in more sophisticated diagnostic terms, identifying diagnoses more clearly.

Table 1
Components of PES Statement

	Definition	Example (P...E...S)
P	**Problem:** the actual nutrition diagnosis, taken from the standardized terms for nutrition diagnosis. Terms from the "intake" domain should be considered first.	Inadequate dietary protein *related to*
E	**Etiology:** the cause of the diagnosis, or factors contributing to the diagnosis	Knowledge deficit about dietary protein needs for dialysis patients *as shown by*
S	**Signs and symptoms:** defining characteristics of the diagnosis which will be monitored to evaluate outcomes.	Patient still following meal plans for 45-50 g protein/day

This is similar to our experiences in nephrology nutrition as we move forward in adopting the NCP model and SL. It is a challenge adding this into everyday practice, but it is also a rewarding process of professional growth---and that is a big part of what the NCP model is about.

The case study that follows describes an initial assessment for a patient who has chronic kidney disease (CKD) stage 5 secondary to Diabetes Mellitus Type 2 (DM2), and recently started peritoneal dialysis (PD). This is a fairly typical presentation, and without major co-morbidities. The case study is presented in the format of an initial assessment note to provide examples of a possible PES statement for the case, and to show how a chart note might be developed to demonstrate the NCP model. It also demonstrates that it is acceptable to have more than one diagnosis in one note. The important point is that any diagnosis that is presented must also be included in the intervention, monitoring and evaluation components of the NCP.

Future topics in this RNF series will include tools to support implementation of NCP and its terminology (including the IDNT Reference Manual and resources on the ADA web page), discussions of each component of the NCP, documentation of follow-up notes, and the application of NCP and SL to all stages of CKD.

CASE STUDY – New PD Patient

Assessment

57 yr old male with CKD stage 5 due to DM2

Food and Nutrition History: Previous education on low sodium, low phosphorus, low potassium, and calorie controlled diet while in hospital. Some education at hemodialysis (HD) unit before starting PD. Reports good appetite, 3 meals/day with 1 snack; no nutrition supplements. He does most food shopping and preparation for his family. Eating out more than usual lately due to PD training, but usually eats most meals at home. Believes he follows 2 g sodium diet, but uses some salt at the table; also uses salt substitute. Adjusting well to PD per patient.

Anthropometrics: Height 184.8 cm; medium frame; target weight 115.6 kg; UBW* 127.8 kg (pt is 90% of UBW)
SBW* 88 kg (pt is 131% of SBW); adj SBW* 109 kg; BMI = 34. Concerned about weight gain. Reports 18.2 kg wt loss in last 6 mo, and believes this was fluid weight.

Physical Exam: No gastrointestinal complaints; patient not sure about urine output. Normal dentition. No current exercise, but hopes to return to pre-dialysis routine of 30-44 minutes walking daily.

Clinical Data: CKD stage 5 due to DM2; hyperlipidemia, hx triple-vessel coronary bypass graft (CABG). Checks capillary blood glucose (CBG) 4x/day before meals and at HS. Chronic ambulatory peritoneal dialysis (CAPD) prescription: 4 - 25% 2 Liter exchanges qd. Meds with nutrition significance: Humalog, Protonix, Tums, Promethazine, Nephrovite, Lantus q HS, Amaryl, Actos, Lipitor, Furosemide, Lisinopril

Diagnosis

(Problem-Etiology-Signs and Symptoms or PES)

Problem (or Diagnosis): Excessive carbohydrate intake related to
Etiology: lack of education to date regarding how to adjust for dextrose load of PD, as shown by
Signs and Symptoms: elevated glucose and A1c since starting PD.
Problem (or Diagnosis): Excess dietary potassium due to
Etiology: knowledge deficit about potassium content of foods and seasonings as shown by
Signs and Symptoms: stated use of high potassium salt sub while of "low potassium" diet.

Intervention

Nutrition Prescription: 1.2-1.3 g protein/kg adj SBW or 130-140 g protien/day (4); 2 g sodium, 2 g potassium, low phosphorus, consistent CHO (5-6 carbohydrate choices/meal with 15 g CHO per carb choice)

Intervention 1: Comprehensive nutrition education re: advanced topic

Goal: Patient will understand the concept of 15 gm dietary carbohydrate equal to 1 serving of "carbs" and will be able to

BIOCHEMS	Results	Lab Norm	BIOCHEMS	Results	Lab Norm
Potassium	5.0 mEq/L	3.5-5.5	Calcium	9.4 mg/dl	8.5-10.5
CO2	24.0 mmol/L	23-29	ADJ Calcium	9.4 mg/dL	NA
BUN	49 mg/dL	6-20	Phosphorus	3.5 mEq/L	2.5-4.5
Creatinine	12.3 mg/dL	0.7-1.5	Kt/V*	2.1	NA
Glucose	247 mg/dL	70-110	nPCR*	0.73	NA
Albumin	4.0 g/dL	3.5-4.7	Hgb A1c	8.3	

evaluate carbohydrate content of some preferred meals.
Intervention 2: Comprehensive nutrition education re: skill development
Goal: Patient will be able to calculate carbohydrate servings from Nutrition Facts Panel information.
Intervention 3: Brief nutrition education re: survival information
Goal: Patient will stop using potassium-containing salt-substitutes.

Monitoring and Evaluation

Indicator	Criteria
Carbohydrate intake 5-5 carbohydrate choices/meal	Patient aware of CHO of choice/meal and reaches goal
Label Reading skills	Patient able to calculate carbohydrate/serving from nutrition label information.
CBGs and Hemoglobin A1c	Will meet goals set by primary care physician.
Serum Potasium	Serum potassium will be in acceptable range for CAPD patient

(See reference 4 for further information regarding terms used in this case study.) ◆

References:

1. The American Dietetic Association. *Nutrition Diagnosis: A Critical Step in the Nutrition Care Process*. Chicago, IL: American Dietetic Association; 2005.
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Access to the Feature Article: Hidden Phosphorus, Figure 1
http://www.renalnutrition.org/members_only/insert.php *OR*
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Access to Kidney Friendly Facts Columns:
http://www.renalnutrition.org/members_only/kff.php

Calendar/Meetings section for an extensive list of conferences & add'l CPEU opportunities:
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Current & archived PDF files of the Renal Nutrition Forum (RNF) Issues & RNF CPEU Inserts
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For more information about the Certification Specialty Exam in Renal (CSR)
www.renalnutrition.org/faq/index.php

To access a copy of the ADA/CDR code of ethics and reference list updates:
http://www.renalnutrition.org/resources/index.php

Member input and suggestions are a vital part of improving our member resources such as the website. Please submit your ideas and suggestions to Cathy M. Goeddeke-Merickel, Web Editor via **cmgmerickel@gmail.com**

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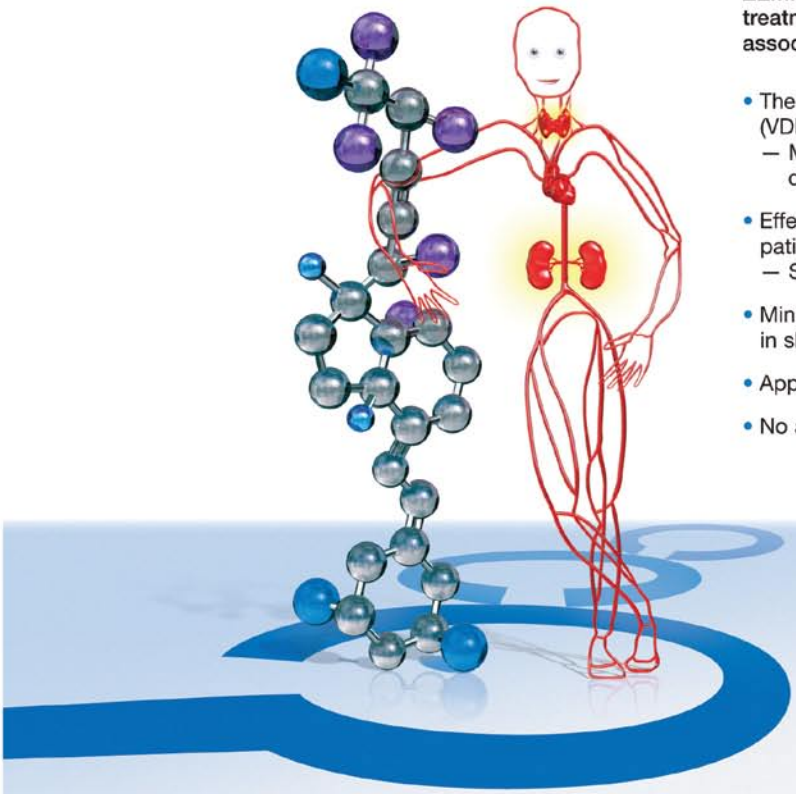

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- Withhold phosphate or vitamin D related compounds during treatment with ZEMPLAR
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- Adverse events with greater than 5% frequency with ZEMPLAR vs placebo, regardless of causality, were nausea (13% vs 8%), vomiting (8% vs 4%), and edema (7% vs 0%)

References: 1. ZEMPLAR Injection [package insert]. North Chicago, IL: Abbott Laboratories; 2005. 2. Data on file. Abbott Laboratories. 3. IMS data. December 2006. 4. Lindberg J, Martin KJ, González EA, Acchiardo SR, Valdin JR, Soltanek C. A long-term, multicenter study of the efficacy and safety of paricalcitol in end-stage renal disease. *Clin Nephrol.* 2001;56:315-323. 5. Martin KJ, González EA, Gellens M, Hamm LL, Abboud H, Lindberg J. 19-Nor-1-α-25-dihydroxyvitamin D₂ (paricalcitol) safely and effectively reduces the levels of intact parathyroid hormone in patients on hemodialysis. *J Am Soc Nephrol.* 1998;9:1427-1432. 6. National Kidney Foundation. K/DOQI clinical practice guidelines for bone metabolism and disease in chronic kidney disease. *Am J Kidney Dis.* 2003;42(suppl 3):S1-S201.

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Renal Dietitians Chair Message

Pamela S. Kent, MS, RD, CSR, LD
RPG Chair

"Vision without action is a daydream. Action without vision is a nightmare." Japanese proverb

As I begin my term as Chair of RPG, I would like to reflect on our Mission and Vision for the Practice Group.

Mission: Renal Dietitians dietetic practice group is leading the future of dietetics by promoting and supporting ADA members working in nephrology practice.

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

The task of leadership is to communicate clearly and repeatedly the organization's vision…all with the intent of helping every person involved understand what work needs to be done and why, and what part the individual plays in the overall effort.

For our action to be effective and efficient, communication is vital. With communication, we can put our ideas together and build upon our actions. In order to improve communication with our membership, the Renal Practice Group has utilized e-blasts messages to keep members aware of significant events and activities that require action.

RPG continues to have an impact on regulatory and legislative issues which continue to support the practice group mission and vision. Recommendations were provided to Centers for Medicare and Medicaid Services on the Proposed Rule for Medicare Conditions of Coverage of End-Stage Renal Disease providers and ADA worked closely with the Council on Renal Nutrition (CRN) to support a nutrition labeling petition to the Food and Drug Administration to mandate potassium and calcium in milligrams on food labels. Representatives from RPG have collaborated with CRN on the Standards of Practice/Standard of Professional Performance project which created a scope of practice for nephrology dietitians.

I look forward to working with new and returning members of the 2008-2009 RPG Executive Committee, and to ongoing partnership with the National Kidney Foundation CRN.

"Your vision of the future determines your action of today." ◆

CRN Chairperson Message

Let's End the Debate on "Which Weight?"

Maria Karalis, MBA, RD, LDN
CRN Chair

One of the key insights from the Council on Renal Nutrition (CRN) Strategic Session held last fall was to generate enthusiasm and an increased sense of urgency regarding the importance of research in clinical practice.

Whether to use actual weight or adjusted weight for the determination of energy requirements is a discussion that is routinely debated on the renalRD listserv. **Your CRN Executive Committee is stepping up to the challenge, and for the first time ever is placing a call for proposals in a specific area of research.** This is one area that is begging for more answers: determination of a predictive equation to estimate energy requirements in the hemodialysis population.

Ann Beemer Cotton, MS, RD, CNSD, and Karen Wiesen, MS, RD, LD, have led the way in the development of this area of research, which involves the pursuit of a predictive equation specific to the hemodialysis (HD) population. The development of this type of equation will provide a valuable tool that is essential to the nutrition assessment process, and may end the debate on "*which weight*" to use.

Predictive equations have been derived for many patient populations, including the elderly, those with a BMI > 40, people with cystic fibrosis, as well as hospitalized patients including an equation specific to the critically ill.

Each population has its unique characteristics that influence energy requirements and therefore benefit from a specific predictive equation. This is especially true of the HD patient based on these known factors:

- Hemodialysis is pro-inflammatory therapy;
- Presence of pro-inflammatory co-morbidities;
- Moderate to severe hyperparathyroidism linked to elevated resting energy expenditure (REE) in HD;
- Elevated serum interleukin-6 and C-reactive protein are linked to an elevated REE in HD patients.

We strongly encourage you and your colleagues to consider submitting a letter of intent for this research. The time has never been more important for Nephrology Dietitians to lead the way in enhancing our knowledge in **improving healthcare delivery to chronic kidney disease patients.** The letter of intent is due October 15th and grant proposals are due December 1st.

For more information, visit: http://www.kidney.org/professionals/research/profcouncil.cfm#crn or call Ann Beemer Cotton at 317-962-8676 or Karen Wiesen at 314-286-0832. ◆

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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 American Dietetic Association*.

Reference citation examples:
Article in periodical:
Knower 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.: Torry Pines Press; 2003.
Web site:
Medscape drug info. Available at www.medscape.com/druginfo. Accessed Feb. 3, 2004.
Author information: List author with first name, middle initial (if any), last name, professional suffix and affiliation (all in italics) 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 along with feature article submissions.

Aimee Zajc, RD, LD
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