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In This Issue

Feature Article

Diabesity, Fatabetes, or Metabolic Syndrome: Different Names … Same Condition?

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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 August 20, 2010.

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Despite advances in detection and treatment of cardiovascular disease (CVD) and diabetes, these conditions continue to result in serious health complications, disability, and premature death. In addition, the incidence rates of CVD, diabetes, and related complications are expected to increase as the United States (U.S.) population ages and the prevalence of obesity continues to rise. At present, approximately 24 million people in the U.S. are diagnosed with diabetes, but another 54 million are considered prediabetic. Since 1990, the prevalence of diabetes has increased by 61%. During 1999-2002, more than half (54.8%) of people with diabetes were obese (BMI > 30) and 85.2% were overweight (BMI of 25-29.9) (1,2). The state of health risk for these metabolic conditions is spelled out in Table 1 (3).

Table 1
The State of Health Risk

- Two out of three Americans are overweight or obese
- More than 70 million (nearly one in four) Americans have varying degrees of insulin resistance
- An estimated 54 million (more than one in six) Americans have prediabetes
- Nearly one in four U.S. adults have high cholesterol
- One in three American adults have high blood pressure

The state of prediabetes is often preceded by a condition which has been labeled by many names (Table 2) and defined by several different professional, national, and international organizations (Table 3). The criteria established by these organizations for the diagnosis of what has been called "Metabolic Syndrome" vary from one to another, with some overlap (Table 4). All organizations seem to agree on certain core components of Metabolic Syndrome: obesity, dyslipidemia, hypertension (HTN), and insulin resistance (IR), with classification of IR established using different laboratory diagnostics. However, these organizations

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From the Editor's Desk

Stacey C. Phillips, RD
Editor

As we start the 2009-2010 year with RPG, I sincerely appreciate this opportunity as incoming editor, and hope to produce editions of the Forum that not only strengthen your nutrition skills but also enhance your enthusiasm within the field of renal nutrition. A sincere thanks should be given to outgoing managing editor Aimee Zajc, RD, LD, for her hard work over the last several years and also to current managing editor, Rachael Majorowicz, RD, LD for her ongoing dedication to providing top notch Forums for you-the readers. Not to be forgotten and highly appreciated are web editor, Cathy Goeddeke-Merickel, MS, RD, LD and advertising editor, Marianne Hutton, RD, CSR, CDE who will continue on in their current roles for the upcoming year. We also welcome Megan Sliwa, RD, LDN to the editorial committee as our new Assistant Editor.

Over the last several months, the editorial team has been working diligently to provide increased opportunities to members through the Forum. To start, online recording of CPEUs has become a simple, yet cost-effective method of documenting these credit hours. Additionally, the editorial team has been able to compile and provide resources for members to use as educational tools with the special electronic issue sent out in early Summer and now available on the RPG web site. Next, as you read through the Summer Forum, you may notice that instead of the usual three continuing professional education credits available, this issue provides you with the chance to complete four. Lastly, I encourage you to visit the online quiz section of the RPG web site. The Nutrition Care Process articles from the Winter 2009 Forum and the Spring 2009 Forum are now being offered as one CPEU credit each. Careful note should be made of the CPEU expiration dates for each of these two articles which corresponds with the original Forum publication date.

This issue of the Forum offers a variety of different topics. The feature article, written by Susan Barlow, RD, CDE, provides an excellent overview of the Metabolic Syndrome, an intricate group of diseases which will more commonly be seen as the complexity of our patient population becomes more advanced. Criteria for the syndrome is specifically outlined which then can be used in everyday practice. The Advances in Practice article, by author Philippa Norton-Feiertag MEd, RD, LD, focuses on a new and developing area of nutrition-nutrigenomics. Along with a brief introduction, this article illustrates how the practice of renal nutrition can potentially benefit from ongoing research. Co-authors Maureen McCarthy, MPH, RD, CSR, LD and Jessie Pavlinac, MS, RD, CSR, LD also provide us another article in the Nephrology Nutrition and the Nutrition Care Process series with the examination and importance of using standardized language in everyday documentation.

As a member of RPG, plenty of opportunities are available for you to become more involved. One of these opportunities, writing an article for the Forum, is something that should be considered as a method of sharing your expertise or area of interest with fellow colleagues. First time or even seasoned authors are encouraged to contribute because without you, the Forum can not be a success. Please feel free to contact me or any member of the editorial team with ideas, comments, or suggestions. ◆

Stacey C. Phillips, RD

Table 2
Metabolic Syndrome, AKA...

Metabolic Syndrome	Several specific definitions using a composite of medical parameters to establish as a disease entity
Insulin Resistance	Pathological condition associated with many disease states; a state in which a given level of insulin produces a less than expected biological effect; associated with abnormalities in both glucose and lipid metabolism
Diabesity	Associates obesity as a strong underlying factor in the development of diabetes
Fatabetes	Like "diabesity"
Prediabetes	Condition preceding frank diagnosis of diabetes which has specific diagnostic criteria established based on blood glucose levels; impaired fasting glucose and impaired glucose tolerance are the two manifestations
Reaven's Syndrome	Named after Gerald Reaven, one of the first to examine this cluster of metabolic risk factors in both CVD and diabetes
Deadly Quartet	Obesity, HTN, hyperglycemia, and hypertriglyceridemia
Hypertriglyceridemic Waist	
Dysmetabolic Syndrome	
Syndrome "X"	

Table 3
Organizations Establishing Definitions of Metabolic Syndrome

WHO	World Health Organization (1999)
EGIR	European Group for the Study of Insulin Resistance
NCEP-ATPIII	National Cholesterol Education Program – Adult Treatment Panel III (2001)
AACE	American Association of Clinical Endocrinologists – AACE Position Statement on the Insulin Resistance Syndrome (2003/2006)
IDF	"The International Diabetes Federation Consensus Worldwide Definition of the Metabolic Syndrome" (1999; revised 2006 based on ATPIII)
AHA/NHLBI	American Heart Association and National Heart, Lung and Blood Institute of NIH "Scientific Statement: Diagnosis and Management of the Metabolic Syndrome" (2005)

apply the criteria differently to identify the cluster of metabolic derangements (4-7).

Of specific note to renal healthcare specialists, microalbuminuria is listed as a criterion in a couple of the organizational definitions of Metabolic Syndrome, i.e. WHO and AACE (8). Recent large-scale clinical trials exploring the value of diet and exercise as modifiable factors for management of diabetes and development of CVD, such as the Look AHEAD (Action for Health in Diabetes) Study, further established this link (9). Look AHEAD analyses have described that increased BMI and abdominal obesity are associated with albuminuria in overweight and obese adults with type 2 diabetes. This same bidirectional association has also been implicated in the development of nephropathy in type 1 diabetes, as assessed by the FinnDiane Study (10). Using the WHO, NCEP, and IDF definitions of Metabolic Syndrome, this study concluded that Metabolic Syndrome is a risk factor beyond albuminuria for cardiovascular morbidity and diabetes-related mortality in type 1 diabetes.

There are some distinct advantages to using the terminology Metabolic Syndrome, despite the differential definitions. It serves as an operational definition for risk factors predisposing one to various metabolic risks. Most clinicians do not measure global risk

Table 4
Different Organizational Definitions and Characterizations for Diagnosis of Metabolic Syndrome

AHA/NHLBI (2005)	**IDF** (2006)
Any three or more of the following criteria: 1. Elevated waist circumference >102 cm (40 in.) in men and >88 cm (35 in.) in women 2. Elevated triglycerides (TG) ≥150 mg/dL (1.7 mmol/L) or drug treatment for elevated TG 3. Elevated blood pressure (BP) ≥130 mmHg systolic BP or ≥85 mmHg diastolic BP or drug treatment for HTN 4. High density lipoprotein cholesterol (HDL-C) <40 mg/dL in men and <50 mg/dL in women or drug treatment for reduced HDL-C 5. Elevated fasting glucose ≥100 mg/dL or drug treatment for elevated glucose	Central obesity (defined as waist circumference with ethnicity-specific values) plus any two of the following factors: 1. Raised TG ≥150 mg/dL (1.7 mmol/L) or specific treatment for this lipid abnormality 2. Reduced HDL-C <40 mg/dL in males and <50 mg/dL in females or specific treatment for this lipid abnormality 3. Raised BP ≥130/85 mmHg or treatment of previously diagnosed HTN 4. Raised fasting plasma glucose ≥100 mg/dL or previously diagnosed type 2 diabetes
NCEP (2001)	**WHO** (1999)
1. Abdominal obesity defined by waist circumference >102 cm (40 in.) in men and >88 cm (35 in.) in women 2. TG ≥150 mg/dL 3. HDL-C <40 mg/dL in men and <50 mg/dL in women 4. BP ≥130/85 mmHg 5. Fasting plasma blood glucose ≥110 mg/dL	Diabetes, impaired fasting glucose, impaired glucose tolerance, or IR (assessed by clamp studies) and at least two of the following criteria: 1. Waist-to-hip ratio >0.90 in men and >0.85 in women 2. Serum TG ≥1.7 mmol/L or HDL-C <0.9 mmol/L in men and <1.0 mmol/L in women 3. BP ≥140/90 mmHg 4. Urinary albumin excretion rate >20 µg/min or albumin-to-creatinine ratio ≥30 mg/g

Table 5
Risk Factors of Cardiometabolic Risk (CMR)

Modifiable CMR Factors	Non-Modifiable CMR Factors
Overweight/obesity	Age
High blood glucose	Race/ethnicity
High LDL cholesterol	Gender
Low HDL cholesterol	Family history
High triglycerides	
Hypertension	
Hypercoagulation	
Inflammation	
Smoking	
Physical inactivity	
Unhealthy eating	
Psychosocial issues	
Health disparities	

or use multivariate predictive equations as "metabolic syndrome" is easier. Classification as an entity encourages clinicians to look for other abnormal factors, as Metabolic Syndrome is usually defined by at least three, and as many as five, factors. Finally, it has been established by the San Antonio Heart Study that Metabolic Syndrome is a better predictor of diabetes than of CVD (11).

If one were to characterize Metabolic Syndrome, it could be described by a cluster of factors which are all strongly correlated with Cardio-Metabolic Risk (CMR): IR, visceral distribution of body fat (aka. central or abdominal adiposity), dyslipidemia, HTN, and prothrombotic state. CMR is a clustering of risk factors or markers that predispose people to CVD and/or Type 2 diabetes. Intra-abdominal fat is high-risk fat linked to IR, dyslipidemia, HTN, and vascular inflammation. This factor also appears to be a common thread in all of the organizational definitions of Metabolic Syndrome. Table 5 lists both the modifiable and non-modifiable risk factors that are hallmarks of cardiometabolic risk (CMR) (3).

Assessing CMR offers several important and distinct advantages (3):

1. Provides clinicians with a comprehensive view of a patient's health and potential risk for future disease and complications
2. Recognizes that not all risk factors are created equal, given their differential effects on future CVD or diabetes risk
3. Refocuses clinical attention on the value of systematic evaluation, education, lifestyle behavior changes, disease prevention, and treatment
4. Supports an integrated approach to health care

This interpretation of CMR is further complicated, or perhaps more aptly justified, by the identification of another condition, known as Metabolically Obese, Normal Weight (MONW). MONW was first described in an article in the *American Journal of Clinical Nutrition* in 1981 as, "A great many disorders, including maturity-onset (Type 2) diabetes, hypertension, and hypertriglyceridemia, are frequently associated with adult-onset obesity and improve with caloric restriction. It is the premise of this brief review that there are patients with these disorders who are not obese according to standard weight tables or other readily-available criteria, but who would also respond favorably to caloric restriction. It is proposed that such individuals might be characterized by hyperinsulinism and possibly an increase in fat cell size compared to patients of similar age, height and weight and/or to themselves at an earlier time. The possibility is also discussed that inactivity is a contributing factor in some of these individuals and that for them, the appropriate therapy might be exercise"(12). MONW refers to individuals who have many of the predisposing risk factors characterizing CMR, but who are of normal weight, yet still may display the characteristic profile of large abdominal girth, or the "apple" shape.

Table 6
Summary of Concerns Regarding Metabolic Syndrome (13)

- Criteria are ambiguous or incomplete. Rationales for thresholds are ill-defined.
- Value of including diabetes in the definition is questionable.
- Insulin resistance as the unifying etiology is questionable.
- No clear basis for including/excluding other CVD risk factors.
- CVD risk value is variable and dependent on the specific risk factors present.
- The CVD risk associated with the syndrome appears to be no greater than the sum of its parts.
- Treatment of the syndrome is no different than the treatment for each of its components.
- The medical value of diagnosing the syndrome is unclear.

The inconsistencies inherent in this disorder have led to a controversial debate, questioning the existence of Metabolic Syndrome and favoring a default to CMR. "Metabolic Syndrome: Time for a Critical Appraisal" was a joint statement prepared by both the American Diabetes Association and the European Association for the Study of Diabetes in 2005. This statement puts forth arguments questioning the clarity of the existing definition(s) of Metabolic Syndrome. A summary of the expressed concerns is listed in Table 6 (13,14).

Despite the variability in definition and controversy questioning the existence of Metabolic Syndrome as an established disease state, there is one thing that is agreed upon. Whether a healthcare professional believes they are treating a "disease" or whether they diagnose even one of the CMR risk factors, treatment is essential. Recognition of even one factor should prompt the clinician to look for others as they cluster together. CVD varies tremendously depending on how many components of CMR are present and the extent above the normal range the patient's laboratory or anthropometric values fall (3).

CMR factor assessment integrates a broader approach to health management by giving clinicians the tools to assess patient's risk for both diabetes and CVD. As discussed, CMR is influenced by an array of risk factors, including traditional and emerging ones. This type of assessment looks beyond clinically evident problems or abnormalities to assess and manage underlying or subclinical processes. Focusing on evaluation, education, disease prevention, lifestyle, behavior change, and treatment of all related risk factors can optimize health outcomes for patients at risk. Two clinical trials that demonstrate the health benefits of diet and physical activity in both the prevention and improved management of diabetes include the Diabetes Prevention Program and the Look AHEAD studies, respectively (9,15). In the Diabetes Prevention Program, dietary intervention coupled with thirty minutes per day of physical activity resulted in a 58% reduction of progression from prediabetes to type 2 diabetes (15).

The goals for clinical management of CMR include weight control, treating the patient for both diabetes and CVD risks, prevention of type 2 diabetes, and prevention of cardiovascular events. The methods for achieving these goals are therapeutic lifestyle changes, for example the Mediterranean diet and physical activity, along with pharmacologic therapy. Medication goals are summarized in Table 7.

Currently, several new therapeutic drug classes based on naturally occurring gut hormones are being explored and studied

Table 7
CMR Clinical Management: Pharmacologic

- Overall Optimization of the Lipid Profile
  - ♦ Treat elevated TG
  - ♦ Improve low HDL-C
  - ♦ Reduction of LDL (lowers CVD risk, but does not impact metabolic syndrome)
- Achieve Blood Pressure Goals
- Achieve Blood Glucose Goals (Mediterranean Diet)
- Minimize Prothrombotic State (aspirin)
- Correct Insulin Resistance
  - ♦ Weight reduction
  - ♦ Increased physical activity
  - ♦ Drugs that decrease IR have not been proven to reduce coronary artery disease risk and no consensus exists on whether these insulin sensitizers should be used in non-diabetic individuals with Metabolic Syndrome

for the treatment of prediabetes and Metabolic Syndrome/CMR. These drug classes include the incretin mimetics, dipeptidyl peptidase-4 inhibitors, and other glucagon-like peptide-1 analogs. These classes seem to hold promise for potentially altering disease progression through various proposed mechanisms of β-cell preservation and reduction of inflammatory processes, at least in animal models.

A recurrent theme for treatment is that initial therapy for CMR/Metabolic Syndrome should consist of caloric restriction and increased physical activity. The role of the Registered Dietitian is clearly stamped on the management of these risks.

To quote Vicki K. Sullivan, PhD, RD, "It is important to continue aggressively emphasizing the management of those things that are under our control, such as diet, exercise and behavioral changes. Likewise it is important to take into account the individual differences in genetics and energy balance that are sometimes beyond our control"(16). This sounds like a prescription for job security … and it's guaranteed, when you circle back to the state of risk at the beginning of this article. ◆

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An Introduction to Nutrigenomics for the Renal Dietetics Professional

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Nutrigenomics has been identified as an important topic in renal dietetics practice and one that will demand new skills from the renal dietetics professional (1). The goal of this relatively new science is to understand the effects of specific nutrients on an individual's genes, and potentially apply research findings to decrease the incidence or manage the symptoms of chronic diseases (2).

The National Center for Minority Health and Health Disparities Center of Excellence for Nutritional Genomics designs research studies directed to delay, prevent and treat diseases which affect millions of Americans. These diseases include obesity, cardiovascular disease and type 2 diabetes (3). As the results of nutrigenomic research begin to appear in the media, dietetics professionals will need to help patients understand the relationship between nutrition and genes, and the impact of their interactions on health.

This article reviews the basic genetic principles underlying the science of nutrigenomics, examines the goals of nutrigenomic research and provides an example of possible applications for nutrigenomic research in the chronic kidney disease (CKD) population.

Basic Genetic Principles

Each individual's development and physiological functioning is controlled by genetic instructions contained in the double-stranded deoxyribonucleic acid (DNA) molecule. Genes are segments of DNA which carry genetic information in the form of a sequence of purine (adenine and guanine) and pyrimidine (cytosine and thymine) bases (4). Complementary base pairing between purines and pyrimidines (adenine to thymine and guanine to cytosine) stabilizes the DNA molecule and ensures that all information is duplicated on each strand.

In humans, DNA occurs in chromosomes within the cell nucleus. The set of 46 chromosomes containing approximately 20,500 genes and billions of DNA base pairs makes up the human genome (5). Genetic information in DNA is read and copied into ribonucleic acid (RNA) during the process of transcription. RNA specifies the sequence of amino acids assembled into proteins, such as enzymes and hormones, during the process called translation.

Free radicals and other oxidizing agents can change the sequence of bases or cause breakage in the DNA molecule (6). Even small changes in DNA, known as genetic variations or gene variants, may affect an individual's susceptibility to disease (7). Conversely, correct functioning of DNA depends on its interactions with various categories of protein. For example, enzymes called DNA ligases can repair broken DNA strands and are important in DNA replication (8). Polymerases bind DNA and copy the base sequence during transcription. Transcription factors are proteins that bind specific sets of DNA sequences, activating or inhibiting transcription of particular genes (9).

Previous research has shown that a number of food components influence health outcomes via genetic pathways and that genetic variation between individuals may determine how well a protein works (10). Supplementing the diet with selenium, an important component of the antioxidant enzyme glutathione peroxidase, has been linked with decreased incidence of some types of cancer in humans. However, due to genetic variation, some individuals have one amino acid substitution (leucine for proline) in their glutathione peroxidase molecules, which apparently increases their risk for lung cancer. Although the reason for this is unclear, it may reflect reduced ability of these individuals to utilize selenium.

Nutrigenomic Research Goals

Prevention of chronic disease is an underlying theme of nutrition research. However, most chronic diseases are probably the consequence of interactions among numerous environmental factors, multiple genes and their variants (11). An important goal of nutrigenomic studies is to identify the impact of nutrients on the genome for the purpose of promoting health (12). More specifically, nutrigenomics can increase our understanding of how nutrients affect gene expression and the role of genes in determining an individual's response to particular nutrients.

One of the challenges of nutrigenomic research is to separate the effects of large numbers of nutrients on each of the wide variety of targets within the human body. For this reason, simpler

organisms have been used as model systems in nutrigenomic research (12). The presence of adipose-like tissue and a lipid transport system has made the fruit fly a suitable model for studying obesity and related diseases. In addition, methodologies used in medical and pharmacological research are being applied in nutrigenomics. These research strategies focus on identifying the information contained in a nutrient's molecular structure that activates a specific molecular pathway.

Research of this type has led to the identification of transcription factors as important intermediates between nutrients and gene expression (11,12). Transcription factors are produced as a result of a series of biochemical reactions initiated by the binding of an extracellular molecule, such as oxidized-LDL cholesterol, to receptors on the surface of a cell (11). Subsequently, these transcription factors enter the nucleus and bind to specific sequences of the genome, selectively switching particular genes on or off.

The nuclear receptor superfamily of transcription factors has been identified as an important group of sensors for nutrients and their metabolites (12). Transcription factors in this group bind fatty acids and cholesterol metabolites, and then bind specific DNA segments thereby influencing DNA transcription. Thus, nutrients are able to impact protein synthesis and metabolic processes via their interactions with these receptors.

Potential Application of Nutrigenomics in the CKD Population

During the past decade, a number of studies have indicated that omega-3 fatty acids in fish oils may be beneficial for patients with CKD. Treatment with fish oils appears to slow progression of renal disease in patients with a commonly occurring form of glomerulonephritis, while fish consumption is associated with decreased cardiac symptoms and reduced mortality rates in patients undergoing maintenance hemodialysis (13-18).

Renal disease progression has been linked to hypertension and obesity, both of which may damage vascular endothelial tissue, induce inflammation and disrupt renal filtration (19). When the vascular endothelium lining blood vessels are damaged, monocytes and absorbed lipids that accumulate in the walls of affected vessels become macrophages and secrete pro-inflammatory cytokines (20). Inflammation activates endothelial tissue, stimulating secretion of adhesion molecules and resulting in the attraction of more monocytes. Macrophages bind oxidized lipoproteins producing foam cells that may occlude small vessels.

Studies indicate that adding the omega-3 fatty acid docosahexaenoic acid to endothelial cell cultures allows its incorporation into cell membrane phospholipids, resulting in inhibition of endothelial cell activity and decreased production of adhesion molecules (20). There is also evidence to suggest that products of omega-3 fatty acid oxidation inhibit cytokine activity via a mechanism involving transcription factors belonging to the nuclear receptor superfamily. Thus, omega-3 fatty acids may exert transcriptional control over a number of endothelial pro-inflammatory genes, including those that encode adhesion molecules and cytokines in the vascular endothelium. This may provide a basis for therapeutic application of omega-3 fatty acids in the management of inflammatory processes (20-22).

Conclusion

Although evidence from nutrigenomic research is promising, it is currently considered insufficient to warrant the formulation of personalized nutritional recommendations based on genetic information (23). However, it is anticipated that as this type of research proceeds, interventions tailored to specific molecular mechanisms underlying health conditions will become increasingly feasible (11). Such interventions might include helping patients to understand disease states, as well as integrating diet and other lifestyle choices into a treatment plan. Renal dietetics professionals will require an understanding of the influence of specific food components on metabolic pathways, and there will also be a need for continuing education programs focusing on genetics and the application of nutrigenomics in the treatment of chronic disease (24). ◆

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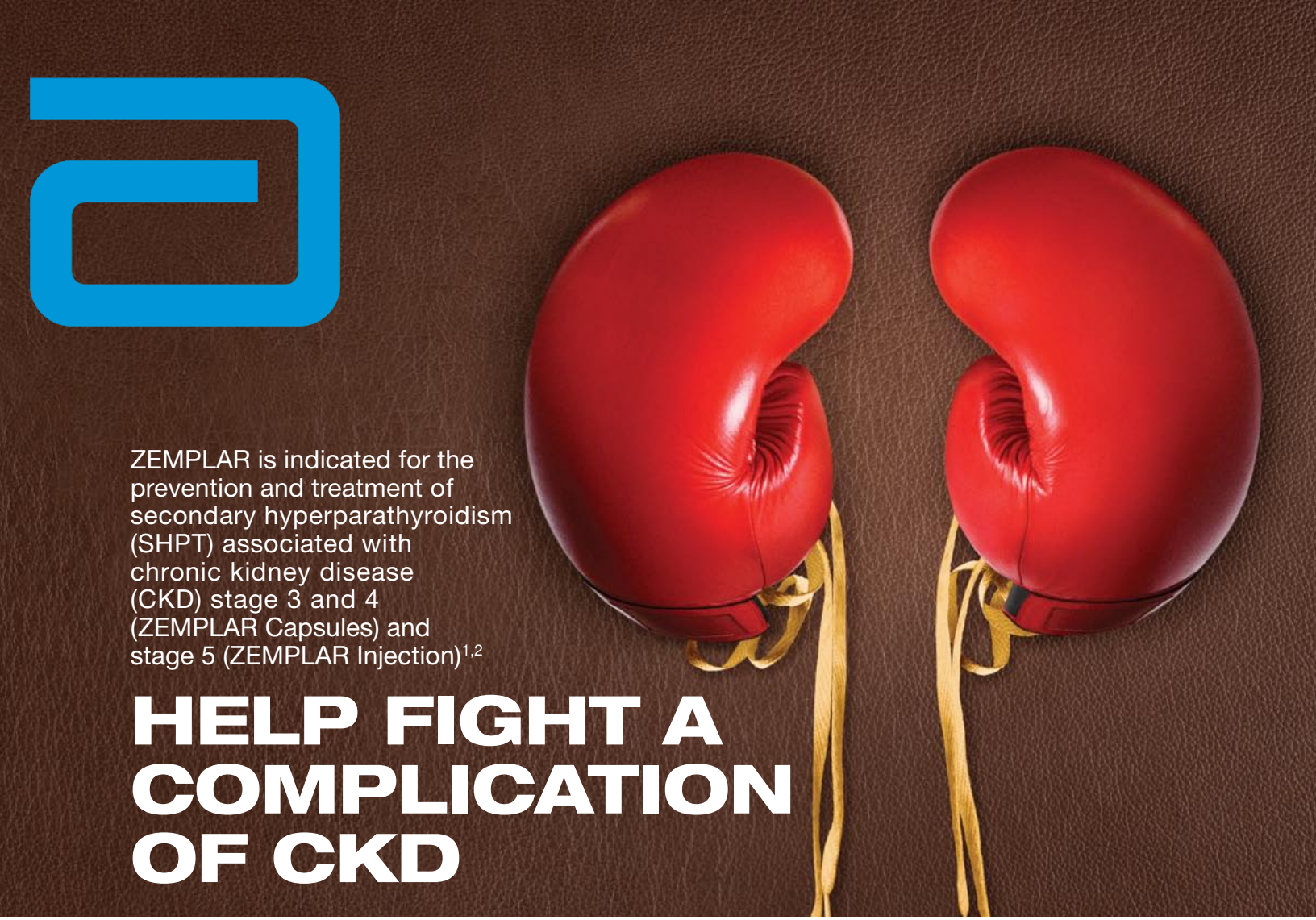

ZEMPLAR is indicated for the prevention and treatment of secondary hyperparathyroidism (SHPT) associated with chronic kidney disease (CKD) stage 3 and 4 (ZEMPLAR Capsules) and stage 5 (ZEMPLAR Injection)[1,2]

HELP FIGHT A COMPLICATION OF CKD

Important Safety Information[1,2]

- ZEMPLAR Capsules and Injection are contraindicated in patients with evidence of vitamin D toxicity, hypercalcemia, or hypersensitivity to any product ingredient.
- Excessive administration of vitamin D compounds can cause over suppression of parathyroid hormone (PTH), hypercalcemia, hypercalciuria, hyperphosphatemia, and adynamic bone disease. High intake of calcium and phosphate concomitant with vitamin D compounds may lead to similar abnormalities, and patient monitoring and individualized dose titration is required. Progressive hypercalcemia due to overdosage of vitamin D may require emergency medical attention. Acute hypercalcemia may exacerbate tendencies for cardiac arrhythmias and seizures and may potentiate the action of digitalis. Use caution when digitalis compounds are prescribed concomitantly with ZEMPLAR. Chronic hypercalcemia can lead to vascular and soft-tissue calcifications. Chronic administration of ZEMPLAR Injection may place patients at risk for hypercalcemia, elevated Ca x P product, and metastatic calcification.
- ZEMPLAR is partially metabolized by CYP3A. Care should be taken while dosing ZEMPLAR with ketoconazole and other strong cytochrome P450 3A inhibitors including atazanavir, clarithromycin, indinavir, itraconazole, nefazodone, nelfinavir, ritonavir, saquinavir, telithromycin or voriconazole.
- During ZEMPLAR Capsules therapy withhold pharmacologic doses of vitamin D compounds. PTH, calcium and phosphorus levels should be monitored at least every 2 weeks for 3 months after initiation or following dose adjustments, then monthly for 3 months, and every 3 months thereafter. Patient monitoring and individualized dose titration are required to maintain physiologic targets and optimum reduction/levels of PTH. The dose of ZEMPLAR Capsules should be reduced or interrupted if hypercalcemia or elevated Ca x P is observed.
- During ZEMPLAR Injection therapy withhold phosphate or vitamin D related compounds. PTH should be monitored at least every 3 months and more frequently at initiation and dosage changes. Calcium and phosphorus should be measured at least monthly and more frequently at initiation or following dosage changes. If clinically significant hypercalcemia develops or an elevated Ca x P product greater than 75 mg^2/dL^2 is noted, the dose should be immediately reduced or interrupted.
- Patients should be informed to adhere to their diet and phosphorus restriction, to take prescribed phosphate binders, and should be knowledgeable about the symptoms of hypercalcemia. While taking ZEMPLAR Capsules patients should be informed to comply with dosage instructions.
- Adverse events reported by at least 5% and at a frequency of at least twice that of placebo were allergic reaction, rash, arthritis, and vertigo for the ZEMPLAR Capsules Stage 3 and 4 treated patients and chills, fever, sepsis, gastrointestinal bleeding, vomiting, edema, light-headedness, and pneumonia for the ZEMPLAR Injection Stage 5 treated patients.

Please see brief summary of Prescribing Information for ZEMPLAR Injection and ZEMPLAR Capsules on following pages.

References: 1. ZEMPLAR (paricalcitol) Capsules [package insert]. North Chicago, IL; Abbott Laboratories. **2.** ZEMPLAR (paricalcitol) Injection [package insert]. Lake Forest, IL; Abbott Laboratories.

ZEMPLAR® (PARICALCITOL)

Goal achievement across the treatment continuum

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A Promise for Life

PROFESSIONAL BRIEF SUMMARY
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Zemplar®

(paricalcitol) Capsules

℞ only

INDICATIONS AND USAGE

Zemplar Capsules are indicated for the prevention and treatment of secondary hyperparathyroidism associated with chronic kidney disease (CKD) Stage 3 and 4.

CONTRAINDICATIONS

Zemplar Capsules should not be given to patients with evidence of vitamin D toxicity, hypercalcemia, or hypersensitivity to any ingredient in this product (see **WARNINGS**).

WARNINGS

Excessive administration of vitamin D compounds, including Zemplar Capsules, can cause over suppression of PTH, hypercalcemia, hypercalciuria, hyperphosphatemia, and adynamic bone disease. Progressive hypercalcemia due to overdosage of vitamin D and its metabolites may be so severe as to require emergency attention. Acute hypercalcemia may exacerbate tendencies for cardiac arrhythmias and seizures and may potentiate the action of digitalis. Chronic hypercalcemia can lead to generalized vascular calcification and other soft-tissue calcification. High intake of calcium and phosphate concomitant with vitamin D compounds may lead to similar abnormalities and patient monitoring and individualized dose titration is required.

Pharmacologic doses of vitamin D and its derivatives should be withheld during Zemplar treatment to avoid hypercalcemia.

PRECAUTIONS

General

Digitalis toxicity is potentiated by hypercalcemia of any cause, so caution should be applied when digitalis compounds are prescribed concomitantly with Zemplar Capsules.

Information for Patients

The patient or guardian should be informed about compliance with dosage instructions, adherence to instructions about diet and phosphorus restriction, and avoidance of the use of unapproved nonprescription drugs. Phosphate-binding agents may be needed to control serum phosphorus levels in patients, but excessive use of aluminum containing compounds should be avoided. Patients also should be informed about the symptoms of elevated calcium (see **ADVERSE REACTIONS**).

Laboratory Tests

During the initial dosing or following any dose adjustment of medication, serum calcium, serum phosphorus, and serum or plasma iPTH should be monitored at least every two weeks for 3 months after initiation of Zemplar therapy or following dose-adjustments in Zemplar therapy, then monthly for 3 months, and every 3 months thereafter.

Drug Interactions

Paricalcitol is not expected to inhibit the clearance of drugs metabolized by cytochrome P450 enzymes CYP1A2, CYP2A6, CYP2B6, CYP2C8, CYP2C9, CYP2C19, CYP2D6, CYP2E1 or CYP3A nor induce the clearance of drugs metabolized by CYP2B6, CYP2C9 or CYP3A.

A multiple dose drug-drug interaction study demonstrated that ketoconazole approximately doubled paricalcitol $AUC_{0-\infty}$. Since paricalcitol is partially metabolized by CYP3A and ketoconazole is known to be a strong inhibitor of cytochrome P450 3A enzyme, care should be taken while dosing paricalcitol with ketoconazole and other strong P450 3A inhibitors including atazanavir, clarithromycin, indinavir, itraconazole, nefazodone, nelfinavir, ritonavir, saquinavir, telithromycin or voriconazole. Dose adjustment of Zemplar Capsules may be required, and iPTH and serum calcium concentrations should be closely monitored if a patient initiates or discontinues therapy with a strong CYP3A4 inhibitor such as ketoconazole.

Drugs that impair intestinal absorption of fat-soluble vitamins, such as cholestyramine, may interfere with the absorption of Zemplar Capsules.

Carcinogenesis, Mutagenesis, Impairment of Fertility

In a 104-week carcinogenicity study in CD-1 mice, an increased incidence of uterine leiomyoma and leiomyosarcoma was observed at subcutaneous doses of 1, 3, 10 mcg/kg given three times weekly (2 to 15 times the AUC at a human dose of 14 mcg, equivalent to 0.24 mcg/kg based on AUC). The incidence rate of uterine leiomyoma was significantly different than the control group at the highest dose of 10 mcg/kg. In a 104-week carcinogenicity study in rats, there was an increased incidence of benign adrenal pheochromocytoma at subcutaneous doses of 0.15, 0.5, 1.5 mcg/kg (< 1 to 7 times the exposure following a human dose of 14 mcg, equivalent to 0.24 mcg/kg based on AUC). The increased incidence of pheochromocytomas in rats may be related to the alteration of calcium homeostasis by paricalcitol. Paricalcitol did not exhibit genetic toxicity *in vitro* with or without metabolic activation in the microbial mutagenesis assay (Ames Assay), mouse lymphoma mutagenesis assay (L5178Y), or a human lymphocyte cell chromosomal aberration assay. There was also no evidence of genetic toxicity in an *in vivo* mouse micronucleus assay. Paricalcitol had no effect on fertility (male or female) in rats at intravenous doses up to 20 mcg/kg/dose (equivalent to 13 times a human dose of 14 mcg based on surface area, mcg/m^2).

Pregnancy

Pregnancy category C

Paricalcitol has been shown to cause minimal decreases in fetal viability (5%) when administered daily to rabbits at a dose 0.5 times a human dose of 14 mcg or 0.24 mcg/kg (based on body surface area, mcg/m^2), and when administered to rats at a dose two times the 0.24 mcg/kg human dose (based on body surface area, mcg/m^2). At the highest dose tested, 20 mcg/kg administered three times per week in rats (13 times the 14 mcg human dose based on surface area, mcg/m^2), there was a significant increase in the mortality of newborn rats at doses that were maternally toxic and are known to produce hypercalcemia in rats. No other effects on offspring development were observed. Paricalcitol was not teratogenic at the doses tested.

Paricalcitol (20 mcg/kg) has been shown to cross the placental barrier in rats.

There are no adequate and well-controlled clinical studies in pregnant women. Zemplar Capsules should be used during pregnancy only if the potential benefit to the mother justifies the potential risk to the fetus.

Nursing Mothers

Studies in rats have shown that paricalcitol is present in the milk. It is not known whether paricalcitol is excreted in human milk. In the nursing patient, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

Geriatric Use

Of the total number (n = 220) of patients in clinical studies of Zemplar Capsules, 49% were 65 and over, while 17% were 75 and over. No overall differences in safety and effectiveness were observed between these patients and younger patients, and other reported clinical experience has not identified differences in responses between the elderly and younger patients, but greater sensitivity of some older individuals cannot be ruled out.

Pediatric Use

Safety and efficacy of Zemplar Capsules in pediatric patients have not been established.

ADVERSE REACTIONS

The safety of Zemplar Capsules has been evaluated in three 24-week (approximately six-month), double-blind, placebo-controlled, multicenter clinical studies involving 220 CKD Stage 3 and 4 patients. Six percent (6%) of Zemplar Capsules treated patients and 4% of placebo treated patients discontinued from clinical studies due to an adverse event. All reported adverse events occurring in at least 2% in either treatment group are presented in Table 3.

Table 3. Treatment - Emergent Adverse Events by Body System Occurring in ≥ 2% of Subjects in the Zemplar-Treated Group of Three, Double-Blind, Placebo-Controlled, Phase 3, CKD Stage 3 and 4 Studies; All Treated Patients

	Number (%) of Subjects			
Body System[a] COSTART V Term	**Zemplar Capsules (n = 107)**		**Placebo (n = 113)**	
Overall	**88**	**(82%)**	**86**	**(76%)**
Body as a Whole	**49**	**(46%)**	**40**	**(35%)**
Accidental Injury	10	(9%)	8	(7%)
Pain	8	(7%)	7	(6%)
Viral Infection	8	(7%)	8	(7%)
Allergic Reaction	6	(6%)	2	(2%)
Headache	5	(5%)	5	(4%)
Abdominal Pain	4	(4%)	2	(2%)
Back Pain	4	(4%)	1	(1%)
Infection	4	(4%)	4	(4%)
Asthena	3	(3%)	2	(2%)
Chest Pain	3	(3%)	1	(1%)
Fever	3	(3%)	1	(1%)
Infection Fungal	3	(3%)	0	(0%)
Cyst	2	(2%)	0	(0%)
Flu Syndrome	2	(2%)	1	(1%)
Infection Bacterial	2	(2%)	1	(1%)
Cardiovascular	**27**	**(25%)**	**19**	**(17%)**
Hypertension	7	(7%)	4	(4%)
Hypotension	5	(5%)	3	(3%)
Syncope	3	(3%)	1	(1%)
Cardiomyopathy	2	(2%)	0	(0%)
Congestive Heart Failure	2	(2%)	5	(4%)
Myocardial Infarct	2	(2%)	0	(0%)
Postural Hypotension	2	(2%)	0	(0%)
Digestive	**29**	**(27%)**	**31**	**(27%)**
Diarrhea	7	(7%)	5	(4%)
Nausea	6	(6%)	4	(4%)
Vomiting	6	(6%)	5	(4%)
Constipation	4	(4%)	4	(4%)
Gastroenteritis	3	(3%)	3	(3%)
Dyspepsia	2	(2%)	2	(2%)
Gastritis	2	(2%)	4	(4%)
Rectal Disorder	2	(2%)	0	(0%)
Hemic and Lymphatic System	**4**	**(4%)**	**10**	**(9%)**
Hypervolemia	2	(2%)	4	(4%)
Ecchymosis	2	(2%)	4	(4%)
Metabolic and Nutritional Disorders	**24**	**(22%)**	**34**	**(30%)**
Edema	7	(7%)	5	(4%)
Uremia	7	(7%)	9	(8%)
Gout	4	(4%)	6	(5%)
Dehydration	3	(3%)	1	(1%)
Acidosis	2	(2%)	1	(1%)
Hyperkalemia	2	(2%)	3	(3%)
Hyperphosphatemia	2	(2%)	4	(4%)
Hypoglycemia	2	(2%)	4	(4%)
Hypokalemia	2	(2%)	1	(1%)
Musculoskeletal	**12**	**(11%)**	**9**	**(8%)**
Arthritis	5	(5%)	1	(1%)
Leg Cramps	3	(3%)	0	(0%)
Myalgia	2	(2%)	5	(4%)
Nervous	**18**	**(17%)**	**12**	**(11%)**
Dizziness	5	(5%)	5	(4%)
Vertigo	5	(5%)	0	(0%)
Depression	3	(3%)	0	(0%)
Insomnia	2	(2%)	2	(2%)
Neuropathy	2	(2%)	1	(1%)
Respiratory	**26**	**(24%)**	**25**	**(22%)**
Pharyngitis	11	(10%)	12	(11%)
Rhinitis	5	(5%)	4	(4%)
Bronchitis	3	(3%)	1	(1%)
Cough Increased	3	(3%)	2	(2%)
Sinusitis	3	(3%)	1	(1%)
Epistaxis	2	(2%)	1	(1%)
Pneumonia	2	(2%)	0	(0%)
Skin and Appendages	**17**	**(16%)**	**10**	**(9%)**
Rash	6	(6%)	3	(3%)
Pruritus	3	(3%)	3	(3%)
Skin Ulcer	3	(3%)	0	(0%)
Skin Hypertrophy	2	(2%)	0	(0%)
Vesiculobullous Rash	2	(2%)	1	(1%)
Special Senses	**9**	**(8%)**	**11**	**(10%)**
Amblyopia	2	(2%)	0	(0%)
Retinal Disorder	2	(2%)	0	(0%)
Urogenital System	**10**	**(9%)**	**10**	**(9%)**
Urinary Tract Infection	3	(3%)	1	(1%)
Kidney Function Abnormal	2	(2%)	1	(1%)

a. Includes all patients with events in that body system.

Potential adverse effects of Zemplar Capsules are, in general, similar to those encountered with excessive vitamin D intake. The early and late signs and symptoms of hypercalcemia associated with vitamin D overdoses include:

Early: Weakness, headache, somnolence, nausea, vomiting, dry mouth, constipation, muscle pain, bone pain, and metallic taste.

Late: Anorexia, weight loss, conjunctivitis (calcific), pancreatitis, photophobia, rhinorrhea, pruritus, hyperthermia, decreased libido, elevated BUN, hypercholesterolemia, elevated AST and ALT, ectopic calcification, hypertension, cardiac arrhythmias, somnolence, death, and, rarely, overt psychosis.

OVERDOSAGE

Excessive administration of Zemplar Capsules can cause hypercalcemia, hypercalciuria, and hyperphosphatemia, and over suppression of PTH (see **WARNINGS**).

Treatment of Overdosage

The treatment of acute overdosage of Zemplar Capsules should consist of general supportive measures. If drug ingestion is discovered within a relatively short time, induction of emesis or gastric lavage may be of benefit in preventing further absorption. If the drug has passed through the stomach, the administration of mineral oil may promote its fecal elimination. Serial serum electrolyte determinations (especially calcium), rate of urinary calcium excretion, and assessment of electrocardiographic abnormalities due to hypercalcemia should be obtained. Such monitoring is critical in patients receiving digitalis. Discontinuation of supplemental calcium and institution of a low-calcium diet are also indicated in accidental overdosage. Due to the relatively short duration of the pharmacological action of paricalcitol, further measures are probably unnecessary. If persistent and markedly elevated serum calcium levels occur, there are a variety of therapeutic alternatives that may be considered depending on the patient's underlying condition. These include the use of drugs such as phosphates and corticosteroids, as well as measures to induce an appropriate forced diuresis.

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Revised: May, 2005

05E-131-J612-2 **MASTER**

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PROFESSIONAL BRIEF SUMMARY
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Zemplar®

(paricalcitol) Injection

Fliptop Vial

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INDICATIONS AND USAGE

Zemplar is indicated for the prevention and treatment of secondary hyperparathyroidism associated with chronic kidney disease Stage 5.

CONTRAINDICATIONS

Zemplar should not be given to patients with evidence of vitamin D toxicity, hypercalcemia, or hypersensitivity to any ingredient in this product (see **WARNINGS**).

WARNINGS

Acute overdose of Zemplar may cause hypercalcemia, and require emergency attention. During dose adjustment, serum calcium and phosphorus levels should be monitored closely (e.g., twice weekly). If clinically significant hypercalcemia develops, the dose should be reduced or interrupted. Chronic administration of Zemplar may place patients at risk of hypercalcemia, elevated Ca x P product, and metastatic calcification.

Treatment of patients with clinically significant hypercalcemia consists of immediate dose reduction or interruption of Zemplar therapy and includes a low calcium diet, withdrawal of calcium supplements, patient mobilization, attention to fluid and electrolyte imbalances, assessment of electrocardiographic abnormalities (critical in patients receiving digitalis), hemodialysis or peritoneal dialysis against a calcium-free dialysate, as warranted. Serum calcium levels should be monitored frequently until normocalcemia ensues.

Phosphate or vitamin D-related compounds should not be taken concomitantly with Zemplar.

PRECAUTIONS

General

Digitalis toxicity is potentiated by hypercalcemia of any cause, so caution should be applied when digitalis compounds are prescribed concomitantly with Zemplar. Adynamic bone lesions may develop if PTH levels are suppressed to abnormal levels.

Information for the Patient

The patient should be instructed that, to ensure effectiveness of Zemplar therapy, it is important to adhere to a dietary regimen of calcium supplementation and phosphorus restriction. Appropriate types of phosphate-binding compounds may be needed to control serum phosphorus levels in patients with chronic kidney disease (CKD) Stage 5, but excessive use of aluminum containing compounds should be avoided. Patients should also be carefully informed about the symptoms of elevated calcium (see **ADVERSE REACTIONS**).

Laboratory Tests

During the initial phase of medication, serum calcium and phosphorus should be determined frequently (e.g., twice weekly). Once dosage has been established, serum calcium and phosphorus should be measured at least monthly. Measurements of serum or plasma PTH are recommended every 3 months. An intact PTH (iPTH) assay is recommended for reliable detection of biologically active PTH in patients with CKD Stage 5. During dose adjustment of Zemplar, laboratory tests may be required more frequently.

Drug Interactions

Paricalcitol is not expected to inhibit the clearance of drugs metabolized by cytochrome P450 enzymes CYP1A2, CYP2A6, CYP2B6, CYP2C8, CYP2C9, CYP2C19, CYP2D6, CYP2E1, or CYP3A nor induce the clearance of drug metabolized by CYP2B6, CYP2C9 or CYP3A.

Specific interaction studies were not performed with Zemplar Injection.

A multiple dose drug-drug interaction study with ketoconazole and paricalcitol capsule demonstrated that ketoconazole approximately doubled paricalcitol $AUC_{0-\infty}$. Since paricalcitol is partially metabolized by CYP3A and ketoconazole is known to be a strong inhibitor of cytochrome P450 3A enzyme, care should be taken while dosing paricalcitol with ketoconazole and other strong P450 3A inhibitors including atazanavir, clarithromycin, indinavir, itraconazole, nefazodone, nelfinavir, ritonavir, saquinavir, telithromycin or voriconazole.

Digitalis toxicity is potentiated by hypercalcemia of any cause, so caution should be applied when digitalis compounds are prescribed concomitantly with Zemplar.

Carcinogenesis, Mutagenesis, Impairment of Fertility

In a 104-week carcinogenicity study in CD-1 mice, an increased incidence of uterine leiomyoma and leiomyosarcoma was observed at subcutaneous doses of 1, 3, 10 mcg/kg (2 to 15 times the AUC at a human dose of 14 mcg, equivalent to 0.24 mcg/kg based on AUC). The incidence rate of uterine leiomyoma was significantly different than the control group at the highest dose of 10 mcg/kg.

In a 104-week carcinogenicity study in rats, there was an increased incidence of benign adrenal pheochromocytoma at subcutaneous doses of 0.15, 0.5, 1.5 mcg/kg (<1 to 7 times the exposure following a human dose of 14 mcg, equivalent to 0.24 mcg/kg based on AUC). The increased incidence of pheochromocytomas in rats may be related to the alteration of calcium homeostasis by paricalcitol.

Paricalcitol did not exhibit genetic toxicity *in vitro* with or without metabolic activation in the microbial mutagenesis assay (Ames Assay), mouse lymphoma mutagenesis assay (L5178Y), or a human lymphocyte cell chromosomal aberration assay. There was also no evidence of genetic toxicity in an *in vivo* mouse micronucleus assay. Zemplar had no effect on fertility (male or female) in rats at intravenous doses up to 20 mcg/kg/dose [equivalent to 13 times the highest recommended human dose (0.24 mcg/kg) based on surface area, mg/m^2].

Pregnancy

Pregnancy Category C.

Paricalcitol has been shown to cause minimal decreases in fetal viability (5%) when administered daily to rabbits at a dose 0.5 times the 0.24 mcg/kg human dose (based on surface area, mg/m^2) and when administered to rats at a dose 2 times the 0.24 mcg/kg human dose (based on plasma levels of exposure). At the highest dose tested (20 mcg/kg 3 times per week in rats, 13 times the 0.24 mcg/kg human dose based on surface area), there was a significant increase of the mortality of newborn rats at doses that were maternally toxic (hypercalcemia). No other effects on offspring development were observed. Paricalcitol was not teratogenic at the doses tested.

There are no adequate and well-controlled studies in pregnant women. Zemplar should be used during pregnancy only if the potential benefit to the mother justifies the potential risk to the fetus.

Nursing Mothers

Studies in rats have shown that paricalcitol is present in the milk. It is not known whether paricalcitol is excreted in human milk. In the nursing patient, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

Pediatric Use

The safety and effectiveness of Zemplar were examined in a 12-week randomized, double-blind, placebo-controlled study of 29 pediatric patients, aged 5-19 years, with end-stage renal disease on hemodialysis and nearly all had received some form of vitamin D prior to the study. Seventy-six percent of the patients were male, 52% were Caucasian and 45% were African-American. The initial dose of Zemplar was 0.04 mcg/kg 3 times per week, based on baseline iPTH level of less than 500 pg/mL, or 0.08 mcg/kg 3 times a week based on baseline iPTH level of ≥ 500 pg/mL, respectively. The dose of Zemplar was adjusted in 0.04 mcg/kg increments based on the levels of serum iPTH, calcium, and Ca x P. The mean baseline levels of iPTH were 841 pg/mL for the 15 Zemplar-treated patients and 740 pg/mL for the 14 placebo-treated subjects. The mean dose of Zemplar administered was 4.6 mcg (range: 0.8 mcg – 9.6 mcg). Ten of the 15 (67%) Zemplar-treated patients and 2 of the 14 (14%) placebo-treated patients completed the trial. Ten of the placebo patients (71%) were discontinued due to excessive elevations in iPTH levels as defined by 2 consecutive iPTH levels > 700 pg/mL and greater than baseline after 4 weeks of treatment.

In the primary efficacy analysis, 9 of 15 (60%) subjects in the Zemplar group had 2 consecutive 30% decreases from baseline iPTH compared with 3 of 14 (21%) patients in the placebo group (95% CI for the difference between groups –1%, 63%). Twenty-three percent of Zemplar vs. 31% of placebo patients had at least one serum calcium level > 10.3 mg/dL, and 40% vs. 14% of Zemplar vs. placebo subjects had at least one Ca x P ion product > 72 $(mg/dL)^2$. The overall percentage of serum calcium measurements > 10.3 mg/dL was 7% in the Zemplar group and 7% in the placebo group; the overall percentage of patients with Ca x P product > 72 $(mg/dL)^2$ was 8% in the Zemplar group and 7% in the placebo group. No subjects in either the Zemplar group or placebo group developed hypercalcemia (defined as at least one calcium value > 11.2 mg/dL) during the study.

Geriatric Use

Of the 40 patients receiving Zemplar in the three phase 3 placebo-controlled CKD Stage 5 studies, 10 patients were 65 years or over. In these studies, no overall differences in efficacy or safety were observed between patients 65 years or older and younger patients.

ADVERSE REACTIONS

Zemplar has been evaluated for safety in clinical studies in 454 CKD Stage 5 patients. In four, placebo-controlled, double-blind, multicenter studies, discontinuation of therapy due to any adverse event occurred in 6.5% of 62 patients treated with Zemplar (dosage titrated as tolerated) and 2.0% of 51 patients treated with placebo for 1 to 3 months. Adverse events occurring with greater frequency in the Zemplar group at a frequency of 2% or greater, regardless of causality, are presented in the following table:

Adverse Event Incidence Rates For All Treated Patients In All Placebo-Controlled Studies

Adverse Event	Zemplar (n=62) %	Placebo (n=51) %
Overall	71	78
Body as a Whole		
Chills	5	0
Feeling unwell	3	0
Fever	5	2
Flu	5	4
Sepsis	5	2
Cardiovascular		
Palpitation	3	0
Digestive System		
Dry mouth	3	2
Gastrointestinal bleeding	5	2
Nausea	13	8
Vomiting	8	4
Metabolic and Nutritional Disorders		
Edema	7	0
Nervous System		
Light-headedness	5	2
Respiratory System		
Pneumonia	5	0

A patient who reported the same medical term more than once was counted only once for that medical term.

Safety parameters (changes in mean Ca, P, Ca x P) in an open-label safety study up to 13 months in duration support the long-term safety of Zemplar in this patient population.

Potential adverse events of Zemplar Injection are, in general, similar to those encountered with excessive vitamin D intake. Signs and symptoms of vitamin D intoxication associated with hypercalcemia include:

Early

Weakness, headache, somnolence, nausea, vomiting, dry mouth, constipation, muscle pain, bone pain, and metallic taste.

Late

Anorexia, weight loss, conjunctivitis (calcific), pancreatitis, photophobia, rhinorrhea, pruritus, hyperthermia, decreased libido, elevated BUN, hypercholesterolemia, elevated AST and ALT, ectopic calcification, hypertension, cardiac arrhythmias, somnolence, death, and rarely, overt psychosis.

Adverse events during post-marketing experience: Taste perversion, such as metallic taste, and allergic reactions, such as rash, urticaria, pruritus, facial and oral edema rarely have been reported.

OVERDOSAGE

Overdosage of Zemplar may lead to hypercalcemia, hypercalciuria, hyperphosphatemia, and over suppression of PTH. (see **WARNINGS**).

Treatment of Overdosage and Hypercalcemia

The treatment of acute overdosage should consist of general supportive measures. Serial serum electrolyte determinations (especially calcium), rate of urinary calcium excretion, and assessment of electrocardiographic abnormalities due to hypercalcemia should be obtained. Such monitoring is critical in patients receiving digitalis. Discontinuation of supplemental calcium and institution of a low calcium diet are also indicated in acute overdosage.

General treatment of hypercalcemia due to overdosage consists of immediate suspension of Zemplar therapy, institution of a low calcium diet, and withdrawal of calcium supplements. Serum calcium levels should be determined at least weekly until normocalcemia ensues. When serum calcium levels have returned to within normal limits, Zemplar may be reinitiated at a lower dose. If persistent and markedly elevated serum calcium levels occur, there are a variety of therapeutic alternatives that may be considered. These include the use of drugs such as phosphates and corticosteroids as well as measures to induce diuresis. Also, one may consider dialysis against a calcium-free dialysate.

Revised: September, 2005

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Report from the Management of Diabetes in Youth Conference

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The Barbara Davis Center for Childhood Diabetes presents its Management of Diabetes in Youth Conference every two years at the Keystone Resort and Conference Center in Keystone, CO, in July. Following is a summary of the "Top-10 Techniques for Attaining Glucose Goals" presented by Gary Scheiner, MS, CDE, who is the Owner/Director of Integrated Diabetes Services in Wynnewood, PA. At his facility, the staff are certified diabetes educators who also have type 1 diabetes; they work primarily with people with type 1 diabetes and insulin-requiring type 2 diabetes. These tips actually apply to individuals of all ages with all types of diabetes.

10. Agree on the Goals

What are the acceptable target blood glucose ranges for your patient? The health care provider(s) and patient need to agree on this. Should they be 70–150 mg/dL, 80–180 mg/dL, 80–200 mg/dL, or somewhere else? How about 1 hour postprandial: <180 mg/dL, 200 mg/dL, or 240 mg/dL?

What percentage of blood glucose values should be in the target range? This again is a discussion point for patients and their health care team. Often the initial goal is to improve on the past. Are we aiming for a value higher than 50% or 70% or something else to be within the target range?

What is an "acceptable" rate for the incidence of hypoglycemia? Which definition of hypoglycemia are we using: less than 80 mg/dL, less than 70 mg/dL or less than 60 mg/dL? How many low blood glucose values per week are acceptable: fewer than five, fewer than three or fewer than two? The allowable incidence of severe low hypoglycemic events is usually zero.

What is the target hemoglobin A1c concentration? In the short-term, we usually wish to improve upon the recent past. What is the ultimate long-term goal? Is this goal age-related?

9. Individualize the Plan of Care

No two patients—whether they are infants, toddlers, school-age children, adolescents, or adults—are the same. Also, no two caregivers are the same. Patients are your clients. We diabetes educators need to adapt to *their* needs and not vice versa. The patient is the boss. We are there to assist them. What is the nature of services provided? Our teaching style needs to adapt to their learning style. What is an appropriate rate of progression? What are the overall goals?

8. Don't Pigeon-Hole

Not everyone fits the usual formulas. Insulin sensitivity formulas using the numbers 1,500, 1,600 and 1,700 are a starting point and need to be individualized. Programs need to be tailored to the client's needs, interests and abilities. They need to be offered a "menu" of options along with the pros and cons of each, such as diet and exercise alone; oral agents; conventional insulin therapy; multiple daily injections; and insulin pumps.

7. Stay on the Cutting Edge

What are the latest medical treatments and are they a match for our patients—insulin cocktails, diluted insulin, oral agents, incretins? People with type 1 diabetes may be able to use oral agents if they have polycystic ovarian syndrome or insulin resistance.

Technology can be our friend. Learn and use the latest devices such as a continuous glucose monitoring system (CGMS), software to download and analyze meter and pump data, insulin pumps, insulin pens, injections ports, new meters, online resources, etc.

We need to think outside the box! Are you willing to try new things? Diabetes management is by its very nature trial and error. Try an approach and make adjustments as indicated.

6. Empower Thy Patients

Show your patients how to critically self-evaluate. Explain the process for making sound self-adjustments. Detail when and who should be contacted on their health care team.

Teach your patients well. We need to teach and foster self-management. Use diabetes tools fully and properly. Make sure they are experts at carbohydrate counting because this is often the weakest link in diabetes management and control. Prepare them for sick days. Can they properly manage hypoglycemia? Teach them to effectively troubleshoot and prevent complications. Scheiner recommends that his patients see their dentist regularly including four cleanings per year. ◆

5. Respect the Basals

Start with the basals. Basal insulin is the foundation of the management program. Make sure the basal insulin is on target before attempting to fine-tune boluses.

When initiating insulin pump therapy, determination of basal rate is the most important step. Establishing the patient's unique pattern of peaks and valleys is key as each individual has his or her own unique basal fingerprint.

Basal testing is infinitely easier with a CGMS. Obtaining loaner units can make this more feasible. Patients wear the unit for a week. They skip breakfast, lunch and supper, or delay their meal. The testing may be done over several days so as to not miss all meals in one day.

Conduct fasting tests to verify basal insulin level. Do this around the clock for those using insulin pumps and overnight for those receiving injections. Clients who use an insulin pump will need to fast to test their basals during the day.

Basal testing conditions include the intake of no food that will raise blood glucose. The last meal or snack should be about four hours before the start of the test and fairly low in fat content. No calories should be consumed during the test period. The test is discontinued if the blood glucose level is lower than 70 mg/dL because then hypoglycemia needs to be treated.

During basal testing, no bolus of insulin is to be given because it would lower the blood glucose level. The last bolus of insulin should be administered at least four hours before the test period. The test is discontinued if the blood glucose level is higher than 250 mg/dL and then a correction bolus may be administered. No temporary basal rate, suspension or disconnection from the insulin pump should be conducted during the testing. No unusual stress, illness or hormonal changes are to occur during the basal test. Patients go about their usual daily activities; however, they should not engage in heavy exercise during the test. They should also be advised to not consume any caffeine. During the time frame that is specified for the basal test period, they will check their blood glucose every one to two hours (every two to three hours overnight).

The grounds for basal rate adjustments are as follows:

- Consistent rise or fall of blood glucose levels through the test phase
- A change of more than 30 mg/dL through the test phase
- If the pattern is irregular, repeat the test before making adjustments
- Make changes one hour before the time needed if using an insulin pump
- If taking injections of long-acting insulin such as insulin glargine (Lantus) or insulin detemir (Levemir), then adjust 10% of the basal dose

Marian Rewers, MD, PhD, from the Barbara Davis Center for Childhood Diabetes suggested checking the basal rates of teenagers at least three times per year and preferably every three months. He noted that the 50-50 basal-bolus "rule" rarely applies to teenagers. For example, a teenage male may require up to 80% of their total daily dose of insulin as a bolus of insulin.

With patients using sensor-augmented insulin pumps, set the target for the percentage of time when the blood glucose levels are within range on the CGMS unit. Over time you can move to tighter targets without hypoglycemia. When evaluating postprandial blood glucose levels, Scheiner suggests using a number of readings at the same time of day to fine tune settings.

4. Plan for Communication

Who is involved in the communication regarding your patient—mother, father, grandparent, youth, husband, wife? Is it shared? Who receives and/or replies at your end? It is important to keep parents in the loop even with the teens. You may wish to suggest that the parent and teen have a short weekly meeting where they share information.

What is your plan for communication? Blood glucose levels only? Premeal, or pre- and postmeal? Include carbohydrate intake, insulin doses, activity? Do they have a programmable meter and/or pump? Written log sheets rule! Blood glucose values by themselves can tell us that something is wrong, but they cannot tell us why it is wrong.

When is communication to occur? Daily? Weekly? Monthly? In special circumstances? Before appointments? How is communication done? Fax, e-mail, Web site (CareLink, etc), transmission of pump/meter data, snail mail?

3. Think Activity

Encourage and support activity. Suggest that they think about it whenever dosing. Insulin does not always work the same in the body. Insulin is only as effective as the body is at using it. Encourage activity that is done daily as well as year-round because insulin sensitivity improves with activity. Tell your clients to think of exercise as medicine that they need daily.

2. Monitor and Adjust Often

Patients need to monitor their blood glucose levels and adjust often. Scheiner suggests that pediatric type 1 diabetic individuals monitor their blood glucose levels during the following:

- Fasting
- Before meals, snacks, and at bedtime
- One hour after meals (rotating)
- Before sports/exercise activity

Management of Diabetes in Youth Conference...

- Every hour during prolonged sports/exercise activity
- Every two to three hours during illness
- 3 a.m. (at least once weekly)

Scheiner feels that the bedtime blood glucose value is the most important one of the entire day. The data should be collected every week by patients and families and reviewed every month or quarter with the health care provider. Analyze the data by time of day and with questions such as, "Are more than 30% high or more than 10% low?" If there are concerns, find the culprit. Items to suspect include the following:

- The insulin program
- Basal insulin
- Insulin-to-carbohydrate ratio
- Correction factor
- Exercise variation
- Lifestyle issues
- Hormone changes
- "Sabotage"

1. Instill the Right Attitude

First impressions count, so be aggressive from the very start! Do remember to let kids be kids. You need to instill the right attitude, but know when to say when. Structure works and certain things *must* get done:

- Blood glucose monitoring
- Insulin administration
- Hypoglycemia prevention and management
- Timely professional care

The last slide in this memorable presentation was "Think Like a Pancreas!" which is included in the title of Gary Scheiner's book *Think Like a Pancreas: A Practical Guide to Managing Diabetes With Insulin.*

The entire Management of Diabetes in Youth Conference presented by the Barbara Davis Center for Childhood Diabetes exceeded my expectations with a large volume of useful, practical information that I am in the process of applying to my practice and sharing with my peers. ◆

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Thank You...

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Adding Sensipar® now?
Good thinking.

81%
of dialysis patients achieved PTH treatment goal when starting Sensipar® at
iPTH 300–500 pg/mL[1]

Waiting until now?
Think again.

22%
of dialysis patients achieved PTH treatment goal when starting Sensipar® at
iPTH > 800 pg/mL[1]

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Sensipar® simultaneously lowers[2]
PTH ↓ Ca ↓ P ↓ Ca x P ↓

Sensipar®
(cinacalcet) Tablets
30mg · 60mg · 90mg

Sensipar® is indicated for the treatment of secondary hyperparathyroidism (HPT) in patients with chronic kidney disease (CKD) on dialysis.

Important Safety Information

Significant reductions in calcium may lower the threshold for seizures. Secondary hyperparathyroidism (HPT) patients, particularly those with a history of seizure disorder, should be carefully monitored for the occurrence of low serum calcium or symptoms of hypocalcemia.

In Sensipar® postmarketing use, isolated, idiosyncratic cases of hypotension, worsening heart failure, and/or arrhythmia were reported in patients with impaired cardiac function. The causal relationship to Sensipar® therapy could not be completely excluded and may be mediated by reductions in serum calcium levels.

Sensipar® lowers serum calcium; therefore, it is important that patients have a serum calcium ≥ 8.4 mg/dL when initiating therapy.

Adynamic bone disease may develop if intact parathyroid hormone (iPTH) levels are suppressed below 100 pg/mL.

Patients with moderate to severe hepatic impairment should be monitored throughout treatment with Sensipar®, as cinacalcet exposure assessed by area under the curve (AUC) was higher than in patients with normal hepatic function.

Serum calcium and serum phosphorus should be measured within 1 week and PTH should be measured 1 to 4 weeks after initiation or dose adjustment of Sensipar®. Once the maintenance dose has been established, serum calcium and serum phosphorus should be measured approximately monthly, and PTH every 1 to 3 months.

The most commonly reported side effects were nausea, vomiting, and diarrhea.

References: 1. Moe SM, Chertow GM, Coburn JW, et al. Achieving NKF-K/DOQI™ bone metabolism and disease treatment goals with cinacalcet HCl. *Kidney Int.* 2005;67:760-771. **2.** Sensipar® (cinacalcet) prescribing information, Amgen.

Please see brief summary of prescribing information on next page.

Brief Summary
See package insert for full prescribing information
SENSIPAR® (cinacalcet) Tablets
INDICATIONS AND USAGE
Sensipar® is indicated for the treatment of secondary hyperparathyroidism in patients with Chronic Kidney Disease on dialysis.
CONTRAINDICATIONS
Sensipar® is contraindicated in patients with hypersensitivity to any component(s) of this product.
WARNINGS
Seizures: In three clinical studies of CKD patients on dialysis, 5% of the patients in both the Sensipar® and placebo groups reported a history of seizure disorder at baseline. During the trials, seizures (primarily generalized or tonic-clonic) were observed in 1.4% (9/656) of Sensipar®-treated patients and 0.4% (2/470) of placebo-treated patients. Five of the nine Sensipar®-treated patients had a history of a seizure disorder and two were receiving anti-seizure medication at the time of their seizure. Both placebo-treated patients had a history of seizure disorder and were receiving anti-seizure medication at the time of their seizure. While the basis for the reported difference in seizure rate is not clear, the threshold for seizures is lowered by significant reductions in serum calcium levels. Therefore, serum calcium levels should be closely monitored in patients receiving Sensipar®, particularly in patients with a history of a seizure disorder (see PRECAUTIONS, Hypocalcemia).
Hypotension and/or Worsening Heart Failure: In postmarketing safety surveillance, isolated, idiosyncratic cases of hypotension, worsening heart failure, and/or arrhythmia have been reported in patients with impaired cardiac function, in which a causal relationship to Sensipar® could not be completely excluded and which may be mediated by reductions in serum calcium levels. Clinical trial data showed hypotension occurred in 7% of Sensipar®-treated patients and 12% of placebo-treated patients, heart failure occurred in 2% of both Sensipar®- and placebo-treated patients.
PRECAUTIONS
General
Hypocalcemia: Sensipar® lowers serum calcium, and therefore patients should be carefully monitored for the occurrence of hypocalcemia. Potential manifestations of hypocalcemia include paresthesias, myalgias, cramping, tetany, and convulsions. Sensipar® treatment should not be initiated if serum calcium is less than the lower limit of the normal range (8.4 mg/dL). Serum calcium should be measured within 1 week after initiation or dose adjustment of Sensipar®. Once the maintenance dose has been established, serum calcium should be measured approximately monthly (see DOSAGE AND ADMINISTRATION). If serum calcium falls below 8.4 mg/dL but remains above 7.5 mg/dL, or if symptoms of hypocalcemia occur, calcium-containing phosphate binders and/or vitamin D sterols can be used to raise serum calcium. If serum calcium falls below 7.5 mg/dL, or if symptoms of hypocalcemia persist and the dose of vitamin D cannot be increased, withhold administration of Sensipar® until serum calcium levels reach 8.0 mg/dL, and/or symptoms of hypocalcemia have resolved. Treatment should be re-initiated using the next lowest dose of Sensipar® (see DOSAGE AND ADMINISTRATION). In the 26-week studies of patients with CKD on dialysis, 66% of patients receiving Sensipar® compared with 25% of patients receiving placebo developed at least one serum calcium value < 8.4 mg/dL. Less than 1% of patients in each group permanently discontinued study drug due to hypocalcemia. Sensipar® is not indicated for CKD patients not on dialysis. In CKD patients with secondary HPT not on dialysis, the long-term safety and efficacy of Sensipar® have not been established. Clinical studies indicate that Sensipar®-treated CKD patients not on dialysis have an increased risk for hypocalcemia compared to Sensipar®-treated CKD patients on dialysis, which may be due to lower baseline calcium levels. In a phase 3 study of 32 weeks duration and including 404 subjects (302 cinacalcet, 102 placebo), in which the median dose for cinacalcet was 60 mg at the completion of the study, 80% of Sensipar®-treated patients experienced at least one serum calcium value < 8.4 mg/dL compared to 5% of patients receiving placebo. **Adynamic Bone Disease:** Adynamic bone disease may develop if iPTH levels are suppressed below 100 pg/mL. One clinical study evaluated bone histomorphometry in patients treated with Sensipar® for one year. Three patients with mild hyperparathyroid bone disease at the beginning of the study developed adynamic bone disease during treatment with Sensipar®. Two of these patients had iPTH levels below 100 pg/mL at multiple time points during the study. In the three 6-month, phase 3 studies conducted in CKD patients on dialysis, 11% of patients treated with Sensipar® had mean iPTH values below 100 pg/mL during the efficacy-assessment phase. If iPTH levels decrease below the NKF-K/DOQI recommended target range (150-300 pg/mL)' in patients treated with Sensipar®, the dose of Sensipar® and/or vitamin D sterols should be reduced or therapy discontinued. **Hepatic Insufficiency:** Cinacalcet exposure as assessed by $AUC_{(0\text{-}inf)}$ in patients with moderate and severe hepatic impairment (as indicated by the Child-Pugh method) were 2.4 and 4.2 times higher, respectively, than that in normals. Patients with moderate and severe hepatic impairment should be monitored throughout treatment with Sensipar® (see CLINICAL PHARMACOLOGY, Pharmacokinetics and DOSAGE AND ADMINISTRATION). **Information for Patients:** It is recommended that Sensipar® be taken with food or shortly after a meal. Tablets should be taken whole and should not be divided. **Laboratory Tests: *Patients with CKD on Dialysis with Secondary Hyperparathyroidism:*** Serum calcium and serum phosphorus should be measured within 1 week and iPTH should be measured 1 to 4 weeks after initiation or dose adjustment of Sensipar®. Once the maintenance dose has been established, serum calcium and serum phosphorus should be measured approximately monthly, and PTH every 1 to 3 months (see DOSAGE AND ADMINISTRATION). All iPTH measurements during the Sensipar® trials were obtained using the Nichols IRMA. In patients with end-stage renal disease, testosterone levels are often below the normal range. In a placebo-controlled trial in patients with CKD on dialysis, there were reductions in total and free testosterone in male patients following six months of treatment with Sensipar®. Levels of total testosterone decreased by a median of 15.8% in the Sensipar®-treated patients and by 0.6% in the placebo-treated patients. Levels of free testosterone decreased by a median of 31.3% in the Sensipar®-treated patients and by 16.3% in the placebo-treated patients. The clinical significance of these reductions in serum testosterone is unknown. **Drug Interactions and/or Drug/Laboratory Test Interactions:** See CLINICAL PHARMACOLOGY, Pharmacokinetics and Drug Interactions. Effect of Sensipar® on other drugs: Drugs metabolized by cytochrome P450 2D6 (CYP2D6): Sensipar® is a strong in vitro, as well as in vivo, inhibitor of CYP2D6. Therefore, dose adjustments of concomitant medications that are predominantly metabolized by CYP2D6 (e.g., metoprolol and carvedilol) and particularly those with a narrow therapeutic index (e.g., flecainide, vinblastine, thioridazine and most tricyclic antidepressants) may be required. Desipramine: Concurrent administration of cinacalcet (90 mg) with desipramine (50 mg) increased the exposure of desipramine by 3.6 fold in CYP2D6 extensive metabolizers. Amitriptyline: Concurrent administration of 25 mg or 100 mg cinacalcet with 50 mg amitriptyline increased amitriptyline exposure and nortriptyline (active metabolite) exposure by approximately 20% in CYP2D6 extensive metabolizers. Midazolam: There were no significant differences in the pharmacokinetics of midazolam, a CYP3A4 and CYP3A5 substrate, in subjects receiving 90 mg cinacalcet once daily for 5 days and a single dose of 2 mg midazolam on day 5 as compared to those of subjects receiving 2 mg midazolam alone. This suggests that cinacalcet would not affect the pharmacokinetics of drugs predominantly metabolized by CYP3A4 and CYP3A5. Effect of other drugs on Sensipar®: Sensipar® is metabolized by multiple cytochrome P450 enzymes, primarily CYP3A4, CYP2D6, and CYP1A2. Ketoconazole: Sensipar® is metabolized in part by CYP3A4. Co-administration of ketoconazole, a strong inhibitor of CYP3A4, increased cinacalcet exposure following a single 90 mg dose of Sensipar® by 2.3 fold. Dose adjustment of Sensipar® may be required and PTH and serum calcium concentrations should be closely monitored if a patient initiates or discontinues therapy with a strong CYP3A4 inhibitor (e.g., ketoconazole, erythromycin, itraconazole; see DOSAGE AND ADMINISTRATION). **Carcinogenesis, Mutagenesis, and Impairment of Fertility: Carcinogenicity:** Standard lifetime dietary carcinogenicity bioassays were conducted in mice and rats. Mice were given dietary doses of 15, 50, 125 mg/kg/day in males and 30, 70, 200 mg/kg/day in females (exposures up to 2 times those resulting with a human oral dose of 180 mg/day based on AUC comparison). Rats were given dietary doses of 5, 15, 35 mg/kg/day in males and 5, 20, 35 mg/kg/day in females (exposures up to 2 times those resulting with a human oral dose of 180 mg/day based on AUC comparison). No increased incidence of tumors was observed following treatment with cinacalcet. **Mutagenicity:** Cinacalcet was not genotoxic in the Ames bacterial mutagenicity assay or in the Chinese Hamster Ovary (CHO) cell HGPRT forward mutation assay and CHO cell chromosomal aberration assay, with and without metabolic activation or in the in vivo mouse micronucleus assay. **Impairment of Fertility:** Female rats were given oral gavage doses of 5, 25, 75 mg/kg/day beginning 2 weeks before mating and continuing through gestation day 7. Male rats were given oral doses 4 weeks prior to mating, during mating (3 weeks) and 2 weeks post-mating. No effects were observed in male or female fertility at 5 and 25 mg/kg/day (exposures up to 3 times those resulting with a human oral dose of 180 mg/day based on AUC comparison). At 75 mg/kg/day, there were slight adverse effects (slight decreases in body weight and food consumption) in males and females. **Pregnancy Category C:** In pregnant female rats given oral gavage doses of 2, 25, 50 mg/kg/day during gestation no teratogenicity was observed at doses up to 50 mg/kg/day (exposure 4 times those resulting with a human oral dose of 180 mg/day based on AUC comparison). Decreased fetal body weights were observed at all doses (less than 1 to 4 times a human oral dose of 180 mg/day based on AUC comparison) in conjunction with maternal toxicity (decreased food consumption and body weight gain). In pregnant female rabbits given oral gavage doses of 2, 12, 25 mg/kg/day during gestation no adverse fetal effects were observed (exposures less than with a human oral dose of 180 mg/day based on AUC comparisons). Reductions in maternal food consumption and body weight gain were seen at doses of 12 and 25 mg/kg/day. In pregnant rats given oral gavage doses of 5, 15, 25 mg/kg/day during gestation through lactation no adverse fetal or pup (post-weaning) effects were observed at 5 mg/kg/day (exposures less than with a human therapeutic dose of 180 mg/day based on AUC comparisons). Higher doses of 15 and 25 mg/kg/day (exposures 2-3 times a human oral dose of 180 mg/day based on AUC comparisons) were accompanied by maternal signs of hypocalcemia (periparturient mortality and early postnatal pup loss), and reductions in postnatal maternal and pup body-weight gain. Sensipar® has been shown to cross the placental barrier in rabbits. There are no adequate and well-controlled studies in pregnant women. Sensipar® should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. **Lactating Women:** Studies in rats have shown that Sensipar® is excreted in the milk with a high milk-to-plasma ratio. It is not known whether this drug is excreted in human milk. Considering these data in rats and because many drugs are excreted in human milk and because of the potential for clinically significant adverse reactions in infants from Sensipar®, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the lactating woman. **Pediatric Use:** The safety and efficacy of Sensipar® in pediatric patients have not been established. **Geriatric Use:** Of the 1136 patients enrolled in the Sensipar® phase 3 clinical program, 26% were ≥ 65 years old, and 9% were ≥ 75 years old. No differences in the safety and efficacy of Sensipar® were observed in patients greater or less than 65 years of age (see DOSAGE AND ADMINISTRATION, Geriatric Patients).
ADVERSE EVENTS
Secondary Hyperparathyroidism in Patients with Chronic Kidney Disease on Dialysis: In 3 double-blind placebo-controlled clinical trials, 1126 CKD patients on dialysis received study drug (656 Sensipar®, 470 placebo) for up to 6 months. The most frequently reported adverse events (incidence of at least 5% in the Sensipar® group and greater than placebo) are provided in Table 1. The most frequently reported events in the Sensipar® group were nausea, vomiting, and diarrhea.
Table 1. Adverse Event Incidence (≥ 5%) in Patients On Dialysis

Event*:	Placebo n=470 (%)	Sensipar® n=656 (%)	Event*:	Placebo n=470 (%)	Sensipar® n=656 (%)
Nausea	19	31	Hypertension	5	7
Vomiting	15	27	Asthenia	4	7
Diarrhea	20	21	Anorexia	4	6
Myalgia	14	15	Pain Chest, Non-Cardiac	4	6
Dizziness	8	10	Access Infection	4	5

*Included are events that were reported at a greater incidence in the Sensipar® group than in the placebo group.

The incidence of serious adverse events (29% vs. 31%) was similar in the Sensipar® and placebo groups, respectively. **12-Month Experience with Sensipar®:** Two hundred and sixty-six patients from 2 phase 3 studies continued to receive Sensipar® or placebo treatment in a 6-month double-blind extension study (12-month total treatment duration). The incidence and nature of adverse events in this study were similar in the two treatment groups, and comparable to those observed in the phase 3 studies. **Postmarketing Experience with Sensipar®:** Rash, hypersensitivity, diarrhea and myalgia have been identified as adverse reactions during post-approval use of Sensipar®. Isolated, idiosyncratic cases of hypotension, worsening heart failure, and/or arrhythmia have been reported in Sensipar®-treated patients with impaired cardiac function in postmarketing safety surveillance. Because these reactions are reported voluntarily from a population of uncertain size, it is not always possible to reliably estimate their frequency or establish a causal relationship to drug exposure. **Laboratory values**: Serum calcium levels should be closely monitored in patients receiving Sensipar® (see PRECAUTIONS and DOSAGE AND ADMINISTRATION).
OVERDOSAGE
Doses titrated up to 300 mg once daily have been safely administered to patients on dialysis. Overdosage of Sensipar® may lead to hypocalcemia. In the event of overdosage, patients should be monitored for signs and symptoms of hypocalcemia and appropriate measures taken to correct serum calcium levels (see PRECAUTIONS). Since Sensipar® is highly protein bound, hemodialysis is not an effective treatment for overdosage of Sensipar®.
DOSAGE AND ADMINISTRATION
Sensipar® tablets should be taken whole and should not be divided. Sensipar® should be taken with food or shortly after a meal. Dosage must be individualized. ***Secondary Hyperparathyroidism in Patients with Chronic Kidney Disease on Dialysis:*** The recommended starting oral dose of Sensipar® is 30 mg once daily. Serum calcium and serum phosphorus should be measured within 1 week and PTH should be measured 1 to 4 weeks after initiation or dose adjustment of Sensipar®. Sensipar® should be titrated no more frequently than every 2 to 4 weeks through sequential doses of 60, 90, 120, and 180 mg once daily to target iPTH consistent with the NKF-K/DOQI recommendation for CKD patients on dialysis of 150-300 pg/mL. PTH levels should be assessed no earlier than 12 hours after dosing with Sensipar®. Sensipar® can be used alone or in combination with vitamin D sterols and/or phosphate binders. During dose titration, serum calcium levels should be monitored frequently and if levels decrease below the normal range, appropriate steps should be taken to increase serum calcium levels, such as by providing supplemental calcium, initiating or increasing the dose of calcium-based phosphate binder, initiating or increasing the dose of vitamin D sterols, or temporarily withholding treatment with Sensipar® (see PRECAUTIONS). **Special Populations: Geriatric patients:** Age does not alter the pharmacokinetics of Sensipar®; no dosage adjustment is required for geriatric patients. **Patients with renal impairment:** Renal impairment does not alter the pharmacokinetics of Sensipar®; no dosage adjustment is necessary for renal impairment. **Patients with hepatic impairment**: Cinacalcet exposures, as assessed by $AUC_{(0\text{-}inf)}$, in patients with moderate and severe hepatic impairment (as indicated by the Child-Pugh method) were 2.4 and 4.2 times higher, respectively, than in normals. In patients with moderate and severe hepatic impairment, PTH and serum calcium concentrations should be closely monitored throughout treatment with Sensipar® (see CLINICAL PHARMACOLOGY, Pharmacokinetics and PRECAUTIONS). **Drug Interactions:** Sensipar® is metabolized in part by the enzyme CYP3A4. Co-administration of ketoconazole, a strong inhibitor of CYP3A4, caused an approximate 2-fold increase in cinacalcet exposure. Dose adjustment of Sensipar® may be required and PTH and serum calcium concentrations should be closely monitored if a patient initiates or discontinues therapy with a strong CYP3A4 inhibitor (e.g., ketoconazole, erythromycin, itraconazole; see CLINICAL PHARMACOLOGY, Pharmacokinetics and PRECAUTIONS).

Storage: Store at 25°C (77°F); excursions permitted to 15-30°C (59-86°F). [See USP controlled room temperature]. Rx Only: This product, or its use, may be covered by one or more US Patents including US Patent Nos. 6313146, 6211244, 6031003 and 6011068, in addition to others, including patents pending.

References: 1. National Kidney Foundation: K/DOQI clinical practice guidelines: bone metabolism and disease in chronic kidney disease. American Journal of Kidney Disease 4 2:S1-S201, 2003

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A Renal Nutrition Forum Series with Practice-Based Examples of the Nutrition Care Process (NCP): Why Should Renal Dietitians Use Standardized Terminology?

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This article has been approved for 1 CPE unit. 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 August 20, 2010.

Members without internet access can request a copy of the quiz and certificate of completion from Megan Sliwa, RD, LDN, Address: 425 North Front Street, Apartment 424, Columbus, OH 43215. Please provide your name, ADA number, and phone number.

Introduction

Recent columns in this series have presented case studies to illustrate the use of the standardized terminology that has been developed by the American Dietetic Association (ADA) to support the Nutrition Care Process (NCP) Model (1). The purpose of standardized language (SL) is to allow registered dietitians across many different clinical settings to speak in a common language when they describe the unique care that they provide as nutrition experts. Among other things, this will communicate our role in patient care concisely and clearly; and it will support practice-based research to demonstrate the impact of nutrition care.

The NCP includes 4 steps: assessment, diagnosis, intervention, and monitoring and evaluation. Starting with the first manual in Fall 2006, ADA has developed three lists of SL:

- Terms to describe nutrition diagnosis, step 2 in the NCP (2)
- Terms to describe nutrition intervention, step 3 in the NCP (3) and
- Terms to describe nutrition monitoring and evaluation, as well as assessment, steps 4 and 1 in the NCP (4)

The most recent revised terminology lists are contained in the second edition of the International Dietetics and Nutrition Terminology (IDNT) Reference Manual and will continue to be updated regularly just like other major classification systems in healthcare (4).

End-stage renal disease (ESRD) care providers across the United States are working hard to implement policies that comply with the new Conditions for Coverage (CfCs) which became effective on October 14, 2008 (5). This seems like the ideal opportunity to incorporate SL into documentation of nutrition care. But from conversations with many of our colleagues, we know that this is difficult, due to the urgency of implementing the CfCs at this time when renal dietitians are still uncertain how to use the new SL.

With the following "David Letterman"-type list of important reasons to use SL (Table 1), the authors hope to convince readers that it is essential to include SL in policies, and in forms and documentation templates that are developed for nutrition care of all chronic kidney disease (CKD) patients, including the large subgroup with ESRD.

Top 10 Reasons To Use Standardized Language

10. Using SL will allow renal dietitians to clearly identify the most common diagnoses/interventions in their population.

If renal dietitians in all 50 states are using the SL to describe the nutrition problems or diagnoses they encounter, this will produce a large number of observations, presenting a powerful picture of the state of nutritional well-being in the CKD caseload across the nation.

9. SL will allow center-specific/regional/national data to determine most frequently reported nutrition diagnoses.

It is true that there is an abundance of center-specific, regional and national data about CKD patients; but we have no idea of the connection between certain altered biochemistries, for example, and nutrition diagnoses. Using one variable, serum phosphorus, imagine how interesting it will be to analyze the most prevalent nutrition diagnoses in patients with elevated serum phosphorus. It is conceivable that we will discover that in some centers with less frequent patient/dietitian contact there is a higher incidence of the diagnosis "food and nutrition-related knowledge deficit" (4). In other centers/regions there may be more "self-monitoring deficit" (4). The first diagnosis suggests more dietitian contact time for nutrition education would be beneficial. The second could also suggest that; or it may also suggest that different tools are needed to help patients with self-monitoring of dietary phosphorus and phosphorus binders depending on the etiology that is linked with the diagnosis.

8. Nutrition diagnoses (and/or nutrition interventions) may have potential as a basis for a reimbursement framework. SL is essential if that is to happen.

ADA has been successful at building a foundation for reimbursement for nutrition care in the last decade. There may be potential to add nutrition diagnostic codes to the current procedural terminology (CPT) codes that already exist for medical nutrition therapy, as International Classification for Disease codes are combined with CPT codes in medicine (7).

7. Nutrition diagnosis provides the critical link between assessment and intervention.

The nutrition diagnosis identifies, clearly and concisely, a problem(s) that is the responsibility of the registered dietitian among the full interdisciplinary team (IDT). SL is a tool for communication among the IDT members. Other clinicians, such as physicians, nurses and physical therapists have developed SL to define the major problems they diagnose. Naming a diagnosis and identifying its etiology from assessment data directs the dietitian to an appropriate intervention. And it informs other IDT members clearly about nutrition problems of each patient.

6. SL terms are defined in the IDNT Reference Manual.

The strength of the terminology is that each term is clearly defined on the worksheets (see example in Table 2) in the IDNT Reference Manual (4). For example, for each nutrition diagnosis term, the pertinent worksheet starts with a full definition. This is followed by a list of possible etiologies; and then by a list of possible signs and symptoms. In other words, the elements of the problem-etiology-signs and symptoms statement (PES statement) for a given diagnosis are well delineated. As dietitians begin to implement SL in practice, referring to the manual to be sure terms are being used accurately is very important.

5. SL may help to identify regional differences in nutrition status of CKD patients.

As we all know, electronic health records (EHRs) provide very powerful databases of information about our patients. Through data mining, nutrition managers can develop reports to better understand what types of nutrition problems are being identified; how they are being treated (what services or interventions are being used); and what outcomes are being observed. It may be possible to link certain diagnoses and interventions with successful outcomes. Understanding what interventions are not effective is also valuable. When assessments, diagnoses, interventions and outcomes are described in standard terms, a robust analysis of what works and what does not work becomes much more likely. Understanding regional differences in nutrition diagnoses may help in allocation of materials and resources for intervention, such as education materials, nutrition supplements and renal dietitian hours.

4. SL facilitates electronic charting, coding, and outcomes reports.

Dietitians who are working with EHRs are learning that data entered in free text fields is much more difficult, if not impossible, to retrieve in reports. However, data that is entered by selecting from pre-determined entries in the EHR is easily retrieved and organized into useful reports. SL provides ready-made choices for the checklists that are more easily data-mined for reports of nutrition diagnoses and nutrition interventions, as just two

Table 1
Top 10 Reasons to Use Standardized Language (SL)

10	Using SL will allow renal dietitians to clearly identify the most common diagnoses/interventions in their population.
9	SL will allow center-specific/regional/national data to determine most frequently reported nutrition diagnoses.
8	Nutrition diagnoses (and/or nutrition interventions) may have potential as a basis for a reimbursement framework. SL is essential if that is to happen.
7	Nutrition diagnosis provides the critical link between assessment and intervention.
6	SL terms are defined in the International Dietetics and Nutrition Terminology Reference Manual.
5	SL may help to identify regional differences in nutrition status of chronic kidney disease (CKD) patients.
4	SL facilitates electronic charting, coding, and outcomes reports.
3	SL may allow some analysis of diagnoses and interventions to develop a database of patient acuity.
2	SL presents nutrition care in clinically relevant terms, better quantifying nutrition contributions in interdisciplinary team care.
1	SL supports multi-center, practice-based research in nephrology nutrition outcomes.

Table 2
Excessive Fluid Intake (NI-3.2)

Definition
Higher intake of fluid compared to established reference standards or recommendations based on physiological needs.

Etiology (Cause/Contributing Risk Factors)
Factors gathered during the nutrition assessment process that contribute to the existence or the maintenance of pathophysiological, psychosocial, situational, developmental, cultural, and/or environmental problems:

- Physiological causes, e.g., kidney, liver, cardiac, endocrine, neurological, and/or pulmonary dysfunction; diminished water and sodium losses due to changes in exercise or climate, syndrome of inappropriate antidiuretic hormone (SIADH)
- Food- and nutrition-related knowledge deficit concerning appropriate fluid intake
- Psychological causes such as depression and disordered eating

Signs/Symptoms (Defining Characteristics)
A typical cluster of subjective and objective signs and symptoms gathered during the nutrition assessment process that provide evidence that a problem exists; quantify the problem and describe its severity.

Nutrition Assessment Category	**Potential Indicators of this Nutrition Diagnosis** (one or more must be present)
Biochemical Data, Medical Tests and Procedures	• Lowered plasma osmolarity (270-280 mOsm/kg), only if positive fluid balance is in excess of positive sodium balance • Decreased serum sodium in SIADH
Anthropometric Measurements	• Weight gain
Nutrition-Focused Physical Findings	• Edema in the skin of the legs, sacral area, or diffusely; weeping of fluids from lower legs • Ascites • Pulmonary edema as evidenced by shortness of breath; orthopnea; crackles or rales • Nausea, vomiting, anorexia, headache, muscle spasms, convulsions • Shortness of breath or dyspnea with exertion or at rest • Providing medications in large amounts of fluids • Use of drugs that impair fluid excretion
Food/Nutrition-Related History	Reports or observations of: • Estimated intake of fluid more than requirements (e.g., per body surface area for pediatrics) • Estimated salt intake in excess of recommendations
Client History	• Conditions associated with a diagnosis or treatment, e.g., end-stage renal disease, nephrotic syndrome, heart failure, or liver disease • Coma (SIADH)

examples (6).

Those of us who utilize NCP's SL understand that the code that precedes each term is not meaningful in daily entries into the medical record. Today's codes are very likely to change when ADA identifies a vendor to manage the terminology of the NCP. However, codes for our SL can be a very helpful shorthand in research and for other purposes such as coding nutrition interventions and outcomes.

3. **SL may allow some analysis of diagnoses and interventions to develop a database of patient acuity.**

If renal dietitians in a variety of work settings apply SL in documenting nutrition assessment, diagnosis, intervention, and monitoring and evaluation, it is conceivable that analysis of this database could allow some conclusions about patient acuity. When a plan of care is written according to the CfCs, it will indicate a time frame for specific outcomes. This creates as a metric for evaluating the success of a particular intervention or service. An analysis of diagnoses, interventions and outcomes (including the time frame) will provide very useful information to describe the acuity of specific caseloads in the practice setting.

2. **SL presents nutrition care in clinically relevant terms, better quantifying nutrition contributions in IDT care.**

SL describes clearly and succinctly the unique care that registered dietitians provide. The terms have been developed and defined in detail to document what dietitians do in the NCP. They create a language that is specific and unique to the expertise of clinical nutrition practice.

1. **SL supports multi-center, practice-based research in nephrology nutrition outcomes.**

In 2001, ADA's House of Delegates presented a call for practice-based research that continues to resound in practice settings across the nation (8). Such research is critical to answer basic questions about the impact of nutrition interventions. And these answers are, of course, critical to the future of our profession. SL promises to be a very strong tool in practice-based research for renal dietitians because it means that we will describe every part of the NCP using terminology that is common throughout the United States—quite a powerful foundation. This means that multi-center research projects become more feasible which, in turn, means that researchers are more likely to achieve population sizes that produce meaningful results. Practice groups, such as our own Renal Practice Group, can bring members across the country to study standard nutrition care and evaluate its impact on outcomes.

Conclusions

Implementation of SL in documenting care of CKD patients offers a strong opportunity to develop a sizable number of observations about the four steps of the NCP in this population. This will support essential research to demonstrate that certain nutrition interventions lead to positive outcomes in a given time frame. And this evidence, in turn, will be critical to the future of nutrition as a central component of care of patients with CKD. Dietitians and others with input into patient care policies should strongly encourage the use of SL as defined in the IDNT Reference Manual. Finally, nephrology nutrition leaders who are developing and revising forms that are used in patient care should include SL at every opportunity. ◆

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WANT TO GET INVOLVED?
Let us know!

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The Change to Estimated Average Glucose

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What is Estimated Average Glucose?

Estimated Average Glucose (eAG) is a new measure of blood glucose for the educator's toolbox. The eAG provides a value that correlates with the A1c but is expressed in the same measurements as your patients' blood glucose meters (mg/dL).

Why the Change?

The International Association of Clinical Chemists announced a new A1c assay to standardize measurements around the world. This new assay would have been reported 1.5-2 points lower then the current A1c normal. Diabetes organizations were very concerned that a change in the normal reporting value of A1c would cause considerable confusion for both patients and health care providers.

In an effort to find a solution to this issue, the American Diabetes Association (ADA), European Association for the Study of Diabetes (EASD), and International Diabetes Federation (IDF) joined together to sponsor a study to define the mathematical relationship between A1c and eAG and determine if A1c could be reliably reported as eAG in the same units as what patients see on their own blood glucose meters.

What Did the Study Show?

The A1c-Derived Average Glucose Study established what has long been assumed but never demonstrated … that A1c does represent average glucose over time. With that relationship demonstrated and defined, health care providers can now report A1c results to patients in the same units that they are using for self-monitoring (i.e., mg/dL) which should benefit clinical care. See Table 1 for a comparison of A1c (%) and average glucose levels (mg/dL).

The Pros for Diabetes Education

Reporting glucose control as 'average glucose' will assist health care providers and their patients in being able to better interpret the A1c value in units similar to those seen regularly with blood glucose monitoring.

The pro for diabetes education is that patients will only need to learn one set of numbers. The same numbers they see with their own meters.

Part of the logic for choosing the term "eAG" is that the medical community recently adopted another new term, eGFR, for estimated glomerular filtration rate, which was introduced as an easier to understand measure of kidney function than the established method of measuring creatinine levels to assess kidney function. The hope is that the growing acceptance of eGFR will help spur the adoption of the similar eAG.

Similarly, average glucose will still fit the message of the ABCs of diabetes care that is promoted by ADA and the National Diabetes Education Program as well as many other organizations.

ADA, EASD, and IDF will be working together to conduct educational efforts to make both patients and providers aware of this new terminology, and help to understand the relationship between A1c and eAG. ADA will use both A1c and eAG in our educational materials. ◆

Table 1
Comparison of A1c and eAG Levels

A1c %	eAG (mg/dL)	eAG (mmol/L)
6%	126	7.0
6.5%	140	7.8
7%	154	8.6
7.5%	169	9.4
8%	183	10.1
8.5%	197	10.9
9%	212	11.8
9.5%	226	12.6
10%	240	13.4

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Type 1 and Type 2 Diabetes Evidenced-Based Nutrition Practice Guideline for Adults

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The Type 1 and Type 2 Diabetes Evidence-Based Nutrition Practice Guideline (EBNPG) for Adults have recently been published by the American Dietetic Association (ADA) and are available in the ADA Evidence Analysis Library (EAL) (1). The objective is to provide Medical Nutrition Therapy (MNT) guidelines for type 1 and type 2 diabetes that assist in the normalization and maintenance of glycemia, lipid profiles, blood pressure, and improved quality of life for the person with diabetes.

Evidence-Based Dietetics Practice is the use of systematically reviewed scientific evidence in making food and nutrition practice decisions by integrating the best available evidence with professional expertise and client values to improve outcomes (2). Professional expertise is important to ensure that the guidelines can reasonably be expected to be implemented in the "real world." Client values remind the practitioner that in practice, guidelines need to be individualized, taking into account the client's personal preferences, cultural background, goals, and willingness to change.

Diabetes EBNPG and Toolkit

Guidelines published in the EAL begin with an executive summary of the recommendations followed by an introduction to the process used by the ADA to develop the guidelines. This is followed by lists of major diabetes recommendations, adult weight management, disorders of lipid metabolism, and hypertension recommendations that also apply to diabetes. The recommendations state a course of action for the practitioner and are followed by a summary of the supporting evidence. Evidence is rated as strong, fair, weak, consensus or insufficient. Conditional evidence applies to a specific situation or sub-population group. Imperative evidence is broadly applicable to the target population. Also included with each guideline recommendation is a list of potential risks/harms of implementation, a brief narrative reviewing the supporting evidence, rationale for the recommendation rating, and a link to supporting evidence. The user can use this link to view the conclusion statements and grades, summaries and tables of the articles analyzed, and criteria for the literature search.

A Diabetes Toolkit will be available in 2009. It is a set of companion documents for application of the practice guideline. The toolkit incorporates the Nutrition Care Process and standardized language of the ADA. The toolkit includes documentation forms, outcome monitoring sheets, client education resources, and case studies. It will be available for purchase and downloaded electronically.

Prioritizing MNT for Diabetes

Type 1 diabetes is primarily a disease of insulin deficiency. The first priority of MNT is to integrate insulin therapy into the

Table 1
Questions Analyzed for the Diabetes EBNPG

1. How effective is MNT implemented by RDs in diabetes management?
2. What is the relationship of carbohydrate intake, fiber, glycemic index and metabolic outcomes?
3. What is the relationship of protein and metabolic outcomes?
4. What is the effect of weight management on metabolic outcomes?
5. What is the effect of physical activity combined with MNT on metabolic outcomes?
6. What is the relationship between self-monitoring of glucose and continuous glucose monitoring and metabolic outcomes?
7. What evidence supports nutrition interventions in the prevention and treatment of cardiovascular disease?

individual patient's usual eating and physical activity patterns. Individuals are then taught how to adjust mealtime insulin doses.

Priorities of MNT progress from lifestyle interventions for prevention of diabetes to MNT alone or in combination with glucose-lowering medications or insulin for glucose management. MNT is based on achievement of metabolic goals, using proven strategies and lifestyle changes that the adult with diabetes is willing and able to make.

Questions Analyzed

Table 1 lists the seven questions that were analyzed for the diabetes EBNPG.

MNT Recommendations

The guidelines recommend that MNT provided by RDs for individuals with type 1 and type 2 diabetes should include an initial series of three to four encounters, each lasting 45-90 minutes. This series, beginning at the time of diagnosis of diabetes or at first referral to an RD for MNT, should be completed within three to six months. The RD should determine if additional MNT encounters are needed after the initial series based on the nutrition assessment of learning needs and progress toward desired outcomes. The recommendations are based on studies that differed in the number of lessons (one to five individual sessions or a series of six to twelve group sessions) and length (45-90 minutes). Sustained positive outcomes at one year and longer are reported. Studies that implement a variety of nutrition interventions report reduction in hemoglobin A1c levels, as well as improved lipid profiles, improved weight management, adjustments in medications, and reduction in the risk for onset and progression of co-morbidities. This recommendation was rated strong and imperative.

The guidelines also state that at least one follow-up encounter is recommended annually to reinforce lifestyle changes and to evaluate and monitor outcomes that impact the need for changes in MNT or medications. The RD should determine if additional MNT encounters are needed. Studies involving regular lifestyle intervention sessions (up to one per month) report sustained positive outcomes at one year and longer. This recommendation is rated strong and imperative.

Summary

In summary, evidence clearly supports the role of MNT provided by RDs for the management of diabetes. In providing MNT, the RD:

- Assesses food and nutrition factors, glycemic control, and relative importance of weight management.
- Implements appropriate patient interventions.
- Coordinates care with an interdisciplinary team.
- Monitors outcomes of MNT interventions to determine whether adjustments in foods and meals will be sufficient to achieve goals, or if medication additions or adjustments need to be combined with MNT. ◆

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The Renal Dietitians Practice Group would like to recognize our RPG Members of 50 or more years. Thanks to all RPG 50+ Year Members for your continued support and membership!

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Renal Dietitians Chair Message

Patricia Williams, RD, CSR, LDN
RPG Chair

Wow! Can you believe it? We are beginning another year in the Renal Dietitians Dietetic Practice Group (RPG). I want to welcome all of you and thank you for the opportunity to serve as RPG chair for 2009 – 2010.

An unknown author said, "Footprints on the sands of time are not made by sitting down." RPG has been making lots of footprints on the sand. The faithful people who serve as RPG officers, editorial board members, the ADA practice manager, area coordinators and on RPG committees continually work to meet the needs of RPG members. I look forward to working with those who are continuing in their positions and those who are joining the RPG team this year.

Times are challenging for all of us with the recession and the new Conditions of Coverage from the Centers for Medicare and Medicaid Services. RPG is aware of the need to provide quality information and resources to our members, and to this end, RPG has plans for expanding our ability to communicate with members through the Forum, e-blasts, web site upgrades, and the use of webinars. This will allow members to participate in educational sessions without the cost of travel or housing. RPG also offers members the opportunity to borrow literature from the lending library for studying for the Board Certification as a Specialist in Renal Nutrition exam or other projects. RPG's Legislative/Reimbursement Chair continues to represent members and present RPG's views on Capitol Hill. Members also have the opportunity to apply for awards and scholarships.

At FNCE 2009, October 17 to 20 in Denver, Jessie Pavlinac, MS, RD, CSR, LD, a leader in renal nutrition and a member of RPG, will be President of ADA. RPG will join with the Oregon Dietetic Association and the Clinical Nutrition Managers DPG to honor Jessie with a reception. The new Standards of Practice/ Standards of Professional Performance in Nephrology Nutrition should be completed in fall 2009.

Karen Wiesen, MS, RD, LD, is the new chair for the Council of Renal Nutrition (CRN). I would like to welcome her and pledge our continued partnership as CRN and RPG work together to promote excellence in renal nutrition. We currently have a joint project of revising *A Clinical Guide to Nutrition Care in Kidney Disease* and it is slated to be completed in 2011.

I see my role as that of service to the membership of RPG and I want you to feel free to email me any time with your thoughts, needs, suggestions, or requests. May the RPG of ADA always leave footprints on the sands of time. ◆

2009 Board Certification for a Specialist in Renal Nutrition (CSR)

www.cdrnet.org/certifications/spec/sdates.htm

Next Specialty Examination Dates	Application Deadlines (postmarked)
November 2-20, 2009	September 4, 2009
May 3-20, 2010	March 1, 2010

The requirements to be eligible to take the Board Certification as a Specialist in Renal Nutrition are as follows:

- Current Registered Dietitian (RD) status by CDR.
- Maintenance of RD status for a minimum of two years from original examination date (by specialty examination date).
- Documentation of 2,000 hours practice experience as an RD in the specialty area within the past five years (by the date the application is due). Related experience can include direct and indirect activities.

Are you preparing for the CSR exam? RPG members have access to the Lending Library, which offers many of the recommended study materials, including:

- A Clinical Guide to Nutrition Care and Kidney Disease, 2004
- Nutritional Management of Renal Disease, 2nd edition
- Guidelines for Nutrition Care of Renal Patients, 3rd edition

CRN Chairperson Message

Karen Wiesen, MS, RD, LD
CRN Chair

"When you learn, teach. When you get, give. Maya Angelou taught me that." - Oprah Winfrey

At the National Kidney Foundation Spring Clinical Meeting this past March, I had the opportunity to speak with a new Council on Renal Nutrition (CRN) member from Beirut, Lebanon. She had come to the meeting to present a poster on nutrition intervention and education techniques to improve patient compliance in a developing country. She told me that in Lebanon she was the only 'trained' renal dietitian and that general dietitians were currently managing dialysis patients. She had experienced the opportunity to spend time with an American renal dietitian who helped her expand her nutrition knowledge base in renal. She now wanted to share that knowledge and specifically mentor dietitians in the area of renal nutrition so that there would be more renal dietitians in her country. Her enthusiasm to share what she had been taught was exciting, refreshing and a reminder of the importance of mentoring.

The history of mentoring can be traced back to the character Mentor in Homer's epic poem *The Odyssey*. When King Odysseus leaves to fight in the Trojan War, he entrusted the care and teaching of his son to Mentor. Merriam-Webster Dictionary defines a mentor as a "trusted counselor or guide" but through the years other definitions have developed such as an advisor or coach who helps and guides another person's development, helps them improve their performance, develop their leadership qualities or realize their vision. We mentor not to show off our knowledge but to show we care. If we reflect back through our own individual career paths, can we think about a person or persons who we would consider a mentor? Perhaps, someone we looked to as a role model, someone who inspired us, someone who gave us advice or encouraged us to ask any question without fear of being thought naive?

Professional mentoring can be formal such as being a dietetic intern preceptor. With time constraints and patient loads, you may think, "I can't do that!" Remember that mentoring can be informal too, in groups or one on one. With Conditions of Coverage now requiring that a renal dietitian have at least one year's clinical experience after registration, it is more important than ever that we look at how we can mentor other dietitians—whether new to the area of renal or just in need of help with a renal question. Mentoring can also be applied to informally teaching other allied health professionals such as nurses, dialysis technicians or social workers in our own unit. Do you have a nurse new to renal with whom you should be interacting or dialysis technicians who you notice listening while you talk to a patient? Would they like you to help them better understand what you were talking about? Mentoring not only helps the mentored but the mentor. Teachable moments can occur throughout the day and don't take a lot of time.

Share your knowledge. Look around your unit, your office, your clinic, your local dietetic association or CRN group. Find your teachable moment. ◆

ERRATUM

Please note a misprint in the Spring 2009 Renal Nutrition Forum Feature Article **Reducing Inflammation through Micronutrient Therapy for Chronic Kidney Disease**. The following text replaces the last two sentences on page 6, paragraph 2 and the last row of Table 1 (page 5):

> According to the NKF KDOQI, initial dosing recommendations for injectable VD is dependent upon type of VD sterol administered and the severity of secondary hyperparathyroidism and serum PTH levels. VD injection therapy should commence when PTH levels are greater than 300 pg/mL and should cease when PTH levels are less than 150 pg/mL (NKF KDOQI).

Table 1 - Summary of Micronutrient Recommendations

Micronutrient	Therapeutic Dosage	Sources	Comments
Injectable Vitamin D	Individualized based on PTH, per NKF KDOQI recommendations	Calcitriol Doxicalciferol Paricalcitol	Follow NKF KDOQI goals to maintain PTH at 150-300 pg/mL

NKF KDOQI Clinical Practice Guidelines for Bone Metabolism and Disease in Chronic Kidney Disease. *Am J Kidney Dis.* 2003;42:1-201.

These changes will be updated on the web site and CPE quiz.

2009-2010 RPG Executive Committee

Mission: Renal dietitians dietetic 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.

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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 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.

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