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The Effects of Alcohol on the Chronic Kidney Disease Population

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There have been numerous clinical studies evaluating the consequences and possible benefits of alcohol consumption in the general population. However, few studies have focused on the chronic kidney disease (CKD) population. The relationship between heavy or abusive alcohol intake and negative health outcomes, such as elevated blood pressure, some cancers, liver cirrhosis, pancreatitis, injury from automobile accidents and violence, and death, is well documented (1). The possible health benefits of moderate alcohol consumption, such as cardiovascular protection, have been widely studied, but remain controversial.

With regards to alcohol consumption, the terms moderate and heavy are unstandardized, however physicians surveyed defined moderate alcohol intake as 2.2 drinks per day, heavy alcohol intake as 3.5 drinks per day and abusive intake as 5.4 drinks per day (2). One drink refers to either a 12-oz beer, a 5-oz serving of wine or 1.5-oz of distilled spirits. In 2003, approximately 59% of adults in the United States were current drinkers, defined as consumption of at least one alcoholic beverage during the previous month (3).

The 2005 Dietary Guidelines for Americans recommend that those who consume alcohol

should drink in moderation. Moderate consumption is defined as up to one drink per day for women and up to two drinks per day for men (4). In addition, it is recommended that some groups abstain from consuming alcohol, including children and adolescents, pregnant and lactating women, women who may become pregnant, people with conditions such as alcoholism and depression, and individuals taking certain medications which will be discussed in a later section.

Most patients undergoing dialysis take multiple medications daily to treat kidney disease and other comorbidities including diabetes, heart disease and hypertension. A patient who consumed alcohol prior to kidney failure may inquire whether alcohol consumption is contraindicated on dialysis. Due to the complicated health condition and medical treatment of those with CKD, patients should consult their physicians about alcohol intake. Once a patient makes an informed decision to drink alcohol, the renal dietitian is an appropriate source for evidence-based clinical recommendations regarding safe and moderate alcohol consumption for the CKD patient.

The following article addresses key counseling considerations for the renal dietitian and other healthcare practitioners regarding alcohol consumption in patients undergoing dialysis.

Key Counseling Considerations

Currently there is a scarcity of nutrition literature on the consumption of alcohol within the CKD population. One significant role of the renal dietitian is to educate patients regarding a modified nutrition plan that may limit

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■ FROM THE EDITOR'S DESK

Renal Nutrition Forum is published quarterly (winter, spring, summer, fall) as a publication of the Renal Dietitians Dietetic Practice Group of the American Dietetic Association.

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

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Our hearts go out to all of the people who have been affected by Hurricane Katrina, especially our members and patients. It is overwhelming to imagine all of the patients who in many cases may not only have lost their homes but who have lost their dialysis clinics too. The renal community has always been especially close. As patients relocate to new clinics around the country, they will be welcomed like family members. Clinicians will be going above and beyond to ensure that the quality of life these patients had will continue to be maintained during this tragic time. It reminds us all why we chose this profession.

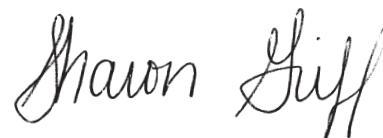
As we give thanks for all that we have this year, there will be family gatherings and holiday parties. With the holidays often comes temptation in the form of rich foods, desserts, and drinks. In our cover article, Lee-Ann Smith examines the effects of alcohol consumption in the CKD population. Ultimately, it is always the patient's decision if and when to consume alcoholic beverages. It is our role to educate on the risks involved, based on their diagnosis and comorbid conditions, which then enables the patient to make an informed decision.

Phillippa Norton Feiertag reviews the effects of ghrelin, a peptide secreted in the gastrointestinal tract, on nutritional status

in the Advances in Practice section. While currently ghrelin is not a common nutritional assessment measurement in the CKD population, this article highlights the potential benefits ghrelin may have, and makes a very interesting case for additional research to understand the impact that ghrelin replacement therapy may have in the CKD population.

Please review the area coordinator introductions and map on page 22. The area coordinators are your liaisons to RPG and the executive board. Please feel free to contact your respective area coordinator with any questions or comments.

The landscape of the renal community is constantly changing. The focus of our clinical practice has expanded to the diagnosis, treatment, and complications of CKD well before a patient reaches dialysis. Thus, it is imperative that we take the lead as practitioners in education, research, and communication in this area. Please feel free to contact me at rnfeditor@yahoo.com with comments and ideas to ensure that we continue to be on the forefront of change.



Many Thanks *Thank you to the following peer reviewers for this issue:*

Sarah Carter
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BRIEF SUMMARY OF PRESCRIBING INFORMATION

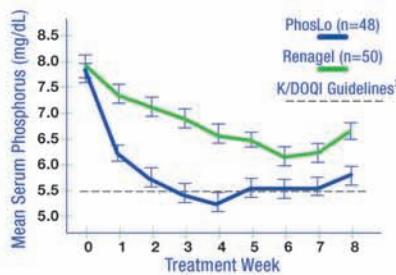
CONTRAINDICATIONS: Patients with hypercalcemia. **INDICATIONS AND USAGE:** For the control of hyperphosphatemia in end stage renal failure. **WARNINGS:** Patients with end stage renal failure may develop hypercalcemia when given calcium with meals. No other calcium supplements should be given concurrently with PhosLo. Progressive hypercalcemia due to overdose of PhosLo may be severe as to require emergency measures. Chronic hypercalcemia may lead to vascular calcification, and other soft-tissue calcification. The serum calcium level should be monitored twice weekly during the early dose adjustment period. **The serum calcium times phosphate (CaXP) product should not be allowed to exceed 66.** Radiographic evaluation of suspect anatomical region may be helpful in early detection of soft-tissue calcification. **PRECAUTIONS:** Excessive dosage induces hypercalcemia; therefore, early in the treatment during dosage adjustment serum calcium should be determined twice weekly. Should hypercalcemia develop, the dosage should be reduced or the treatment discontinued immediately depending on the severity of hypercalcemia. Do not give to patients on digitals, because hypercalcemia may precipitate cardiac arrhythmias. Always start PhosLo at low dose and do not increase without careful monitoring of serum calcium. An estimate of daily calcium intake should be made initially and the intake adjusted as needed. Serum phosphorus should also be determined periodically. **Information for the Patient:** Inform the patient about: 1) compliance with dosage, 2) adherence to diet instructions and avoidance of nonprescription antacids, and 3) symptoms of hypercalcemia. **Drug Interactions:** PhosLo may decrease the bioavailability of tetracyclines. **Carcinogenesis, Mutagenesis, Impairment of Fertility:** Long term animal studies have not been performed. **Pregnancy:** Teratogenic Effects: Category C. Animal reproduction studies have not been conducted. It is not known whether PhosLo can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Give to a pregnant woman only if clearly needed. **Pediatric Use:** Safety and effectiveness in pediatric patients have not been established. **Geriatric Use:** Of the total number of subjects in clinical studies of PhosLo (n = 91), 25 percent were 65 and over, while 7 percent were 75 and over. No overall differences in safety or effectiveness were observed between these subjects and younger subjects, 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. **ADVERSE REACTIONS:** In clinical studies, patients have occasionally experienced nausea during PhosLo therapy. Hypercalcemia may occur during treatment with PhosLo. Mild hypercalcemia (Ca>10.5 mg/dl) may be asymptomatic or manifest itself as constipation, anorexia, nausea and vomiting. More severe hypercalcemia (Ca>12 mg/dl) is associated with confusion, delirium, stupor and coma. Mild hypercalcemia is easily controlled by reducing the PhosLo dose or temporarily discontinuing therapy. Severe hypercalcemia can be treated by acute hemodialysis and discontinuing PhosLo therapy. Decreasing dialysate calcium concentration could reduce the incidence and severity of PhosLo induced hypercalcemia. The long-term effect of PhosLo on the progression of vascular or soft-tissue calcification has not been determined. Isolated cases of pruritis have been reported which may represent allergic reactions. **OVERDOSAGE:** Administration of PhosLo in excess of appropriate daily dosage can cause severe hypercalcemia (see ADVERSE REACTIONS).

REFERENCE: 1. Qunibi WY, Hootkins RE, McDowell LL, et al. Treatment of hyperphosphatemia in hemodialysis patients: The Calcium Acetate Renagel Evaluation (CARE Study). In press.
2. K/DOQI guidelines (in press).

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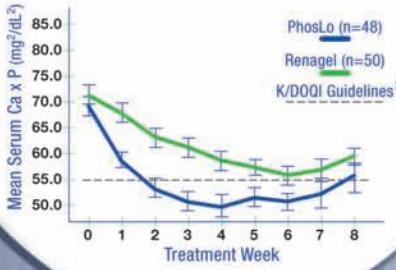
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The Effects of Alcohol *continued from page 1*

phosphorus, potassium, sodium and fluid to improve health outcomes. Patients are instructed on how to make appropriate nutrition choices to meet these restrictions while maintaining optimal nutritional status. When discussing alcohol intake with CKD patients, the renal dietitian should reinforce these nutritional limitations as well as the effects of alcohol on co-morbid conditions the patients may have.

Nutrient Restrictions

Patients should be educated to make informed decisions consistent with renal nutrition plan modifications when choosing an alcoholic beverage. While some drinks may be nearly free of phosphorus, potassium and sodium, other beverages may be a hidden source of one or more of these nutrients (Table 1).

Fluid and Portion Sizes

Alcoholic beverages provide kilocalories and fluid with minimal nutritional value. One serving of alcohol equals 12-oz beer, 5-oz wine or 1.5-oz distilled spirits. Each serving contains approximately 15 grams of alcohol. Interdialytic weight gain is a concern for patients undergoing dialysis, therefore focusing on the appropriate serving sizes of alcoholic beverages allows patients to plan fluid intake and avoid exceeding daily fluid allowances.

Multiple factors play a role in determining an acceptable alcohol intake for the individual including body weight, body composition and gender. Studies show that women and men process the same amount of alcohol per hour, but women eliminate more alcohol per unit of lean body weight compared with men (6). Since women have a higher body fat to body water ratio than do men, women reach higher blood alcohol concentrations after ingesting an equivalent number of servings of alcohol (7, 8). Thus, acceptable alcohol intake for a small woman may be significantly less than what is acceptable for a large man.

Alcohol Metabolism and Drug Interactions

There is an abundant amount of literature pertaining to alcohol metabolism and drug-alcohol interactions. The following is a synopsis of alcohol metabolism and information to consider when counseling patients to avoid drug-alcohol interactions.

Alcohol has the potential to interact adversely with numerous medications (9). The metabolism of alcohol occurs mainly in the liver. The two enzymes alcohol dehydrogenase and cytochrome P450 are responsible for the breakdown of alcohol to acetaldehyde (10). Acetaldehyde is further metabolized and eliminated from the body. Cytochrome P450, also known as microsomal ethanol oxidizing system (MEOS), is central in drug-alcohol interactions (11). The two main com-

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Table 1: Nutrient Content of Portions of Common Alcoholic Beverages (5)

Beverage	K+ (mg)	P04 (mg)	Na+ (mg)	Serving Size (fl oz)
Beer, light	74	42	14	12 oz
Beer, regular	96	50	14	12 oz
Beer, Budweiser®	118	46	11	12 oz
Beer, Bud Light®	92	39	11	12 oz
Beer, Michelob® Ultra™	60	28	11	12 oz
Crème de menthe	0	0	2	1.5 oz
Daiquiri, from recipe	13	3	3	2 oz
Daiquiri, from can	11	2	40	6.8 oz
Gin, rum, whiskey, vodka	1	2	0	1.5 oz
Liqueur, coffee	16	3	4	1.5 oz
Liqueur, coffee with cream	15	24	43	1.5 oz
Martini	5	1	1	1 oz
Piña Colada, from recipe	100	10	8	4.5 oz
Piña Colada, from can	184	80	158	6.8 oz
Sake, rice	7	2	1	1 oz
Tequila Sunrise, from can	21	21	120	6.8 oz
Whiskey Sour, from mix	19	5	65	3.5 oz
Whiskey Sour, from can	23	13	92	6.8 oz
Wine, dessert	95	9	9	3.5 oz
Wine, red	115	14	5	3.5 oz
Wine, white	82	14	5	3.5 oz
NONALCOHOLIC BEVERAGES/ MIXERS				
Club soda	7	0	75	12 oz
Cola	4	48	15	12 oz
Cranberry juice	46	5	5	8 oz
Cream, half & half	39	29	12	1 oz
Eggnog	329	209	150	8 oz
Ginger Ale	4	0	26	12 oz
Grapefruit juice	378	27	2	8 oz
Lemon-lime soda	4	0	40	12 oz
Lime juice	288	34	5	8 oz
Pineapple juice	335	20	2	8 oz
Tomato juice, no added salt	556	44	24	8 oz
Tomato juice, added salt	556	44	654	8 oz
Wine, nonalcoholic	91	14	7	3.5 oz

The Effects of Alcohol *continued from page 4*

Table 2: Interaction of Drug Classes and Alcohol (10, 12, 13)

Drug Classification	Type of Interaction
Analgesics (aspirin and acetaminophen)	Alcohol intake with aspirin may increase side effects including gastric bleeding and anticoagulation. Increased gastric emptying with aspirin may cause faster alcohol absorption. Chronic alcohol intake with acetaminophen may cause liver damage.
Anesthetics (propofol)	Chronic alcohol intake increases dose needed to induce loss of consciousness. May increase risk of liver damage.
Antibiotics (furazolidone, griseofulvin, metronidazole, erythromycin, isoniazid and quinacrine)	Acute alcohol intake may cause nausea, vomiting, headache, and convulsions. Erythromycin may increase gastric emptying causing faster alcohol absorption. Increased risk of isoniazid-related liver disease.
Anticoagulants (warfarin)	Chronic alcohol intake decreases anticoagulation. Acute alcohol intake increases anticoagulation by reduces drug's metabolism.
Anticonvulsants (phenytoin)	Acute alcohol intake increases availability and the risk of side effects. Chronic alcohol intake decreases availability, even during abstinence, decreasing drug effectiveness.
Antidepressants (tricyclics and Monoamine oxidase inhibitors including amitriptyline, clomipramine, desipramine, doxepin, imipramine, and nortriptyline)	Acute alcohol intake increases availability, increasing sedative effects with tricyclics. Tyramine, in some beers and wine, may cause a severe alteration in blood pressure with MAO inhibitors.
Antihistamines (diphenhydramine)	May cause dizziness and increase side effects on CNS causing drowsiness, sedation and decreased motor skills. The elderly may be more prone to these interactions.
Antipsychotics (chlorpromazine)	Acute alcohol intake enhances sedative side effects, may cause impaired coordination and fatal breathing complications.
Cardiovascular (nitroglycerin, reserpine, methyldopa, Isosorbide dinitrate, hydralazine, and guanethidine)	Acute alcohol intake may cause dizziness or fainting when standing up. Chronic alcohol intake decreases availability, reducing effectiveness.
Herbal remedies (chamomile, echinacea and valerian)	Alcohol may intensify the drowsiness associated with these supplements.
Histamine H₂ receptor antagonists (cimetidine, nizatidine, and ranitidine)	These drugs decrease gastric ADH and increase gastric emptying, leading to higher than expected blood alcohol levels per alcohol serving.
Narcotic pain relievers (morphine, codeine, propoxyphene, and meperidine)	Combining alcohol with opiates increases side effects of both drugs, affecting the CNS causing drowsiness, sedation and decreased motor skills; may increase risk of death by overdose.

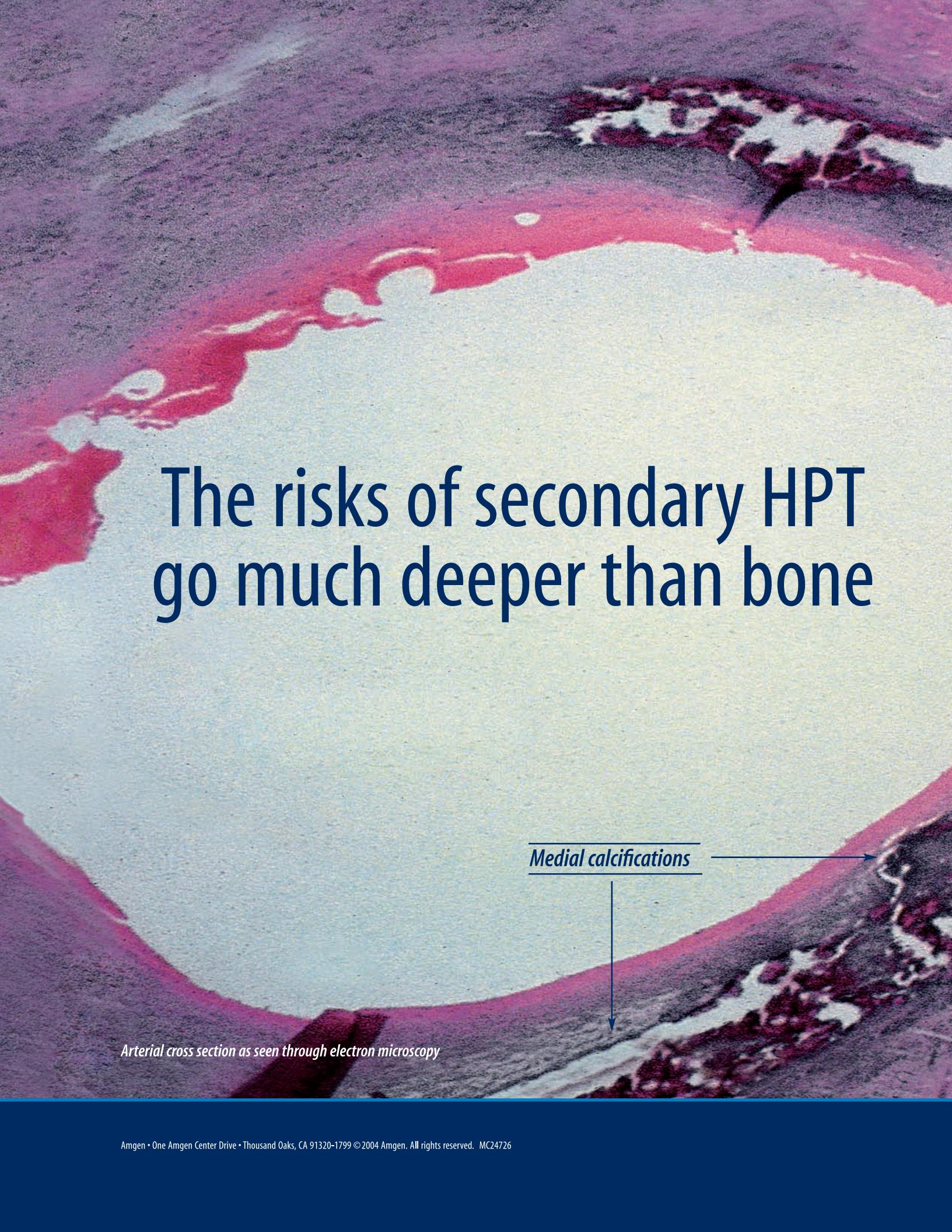
ponents of cytochrome P450 are the enzymes cytochrome P450 reductase and CYP2E1 (11). CYP2E1 metabolizes both alcohol and numerous drugs. Alcohol may alter the effectiveness of a drug by interfering with its availability or the extent to which the drug reaches the target site at a specific dose (9). The following are common mechanisms of pharmacokinetic alcohol-drug interactions:

- 1) Acute alcohol ingestion (moderate) may inhibit a drug's metabolism by competing with the drug for cytochrome

P450, which increases and prolongs the drug's availability.

- 2) Chronic heavy alcohol intake (in nonintoxicated state) enhances cytochrome P450 activity, leading to inadequate drug levels and decreased effectiveness. In addition, enzymes associated with chronic alcohol use can breakdown medications into toxins, which may accumulate and cause organ damage.

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An electron micrograph showing a cross-section of an artery. The lumen is filled with a light-colored, granular material. The tunica media is visible as a thick, pinkish-red layer containing several bright, white, irregularly shaped structures labeled 'Medial calcifications'. The tunica intima is the thin, dark layer at the top.

The risks of secondary HPT go much deeper than bone

Arterial cross section as seen through electron microscopy

Medial calcifications

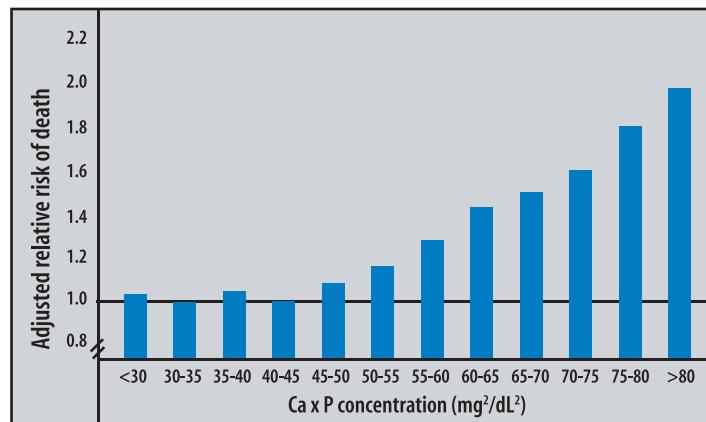
Failure to achieve NKF-K/DOQI™ bone metabolism goals* increases the risk of patient mortality¹⁻³

Uncontrolled secondary HPT can be harmful for your patients on dialysis. In addition to bone disease and parathyroid gland hyperplasia, adverse outcomes include soft-tissue and cardiovascular calcification, increased hospitalizations, cardiovascular events, and increased mortality risk.^{1,2,4} The majority of CKD patients on dialysis have metabolic parameters outside the K/DOQI[™] goals despite use of traditional therapies.⁵

Only 17% of patients meet K/DOQI™ goals for both PTH and Ca x P, and only 8% meet all 4 goals^{5,6}

A new analysis confirms that patients with PTH and Ca x P values outside the K/DOQI[™] goal range are at significantly increased risk of mortality.¹ And the risk begins at relatively lower lab parameters: one retrospective study of over 40,000 patients showed that any Ca x P above 45 mg²/dL² increased death and hospitalization rates.¹

New analyses show the adverse consequences of uncontrolled secondary HPT¹



Adapted from Block et al.¹

This significant increase in risk caused by secondary HPT can be controlled. Through optimal clinical management of bone metabolism parameters, more patients can achieve the 4 key K/DOQI[™] goals and patient outcomes can potentially be improved.⁴

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*NKF-K/DOQI[™] Guidelines for Bone Metabolism and Disease in Chronic Kidney Disease.
K/DOQI is a trademark of the National Kidney Foundation, Inc.

3) Chronic heavy alcohol intake (during intoxicated state) requires activation and primary involvement of CYP, reducing drug metabolism and excretion (11).

While pharmacokinetic interactions are of a primary concern in chronic heavy drinkers, pharmacodynamic interactions may occur during a single episode of moderate drinking (10). Pharmacodynamic drug-alcohol interactions often occur in the central nervous system when alcohol intake changes a drug's effect without altering serum drug levels. The type of interaction depends on the class of medication involved (Table 2).

Vitamin Deficiency

Vitamin deficiencies are common with excessive alcohol consumption (14). Two common vitamin deficiencies in people with alcoholism are folate and thiamine deficiencies, however multiple nutrient deficiencies may occur as alcohol interferes with the conversion and storage of vitamins in the liver (14, 15). In addition, the replacement of nutrient dense foods with alcohol further contributes to these deficiencies. Folate deficiency is likely caused by an increased need for nucleic acids to regenerate damaged liver cells (14). One study using both humans and experimental primates found an association of chronic excessive alcohol intake with intestinal malabsorption, decreased uptake by the liver and increased urinary excretion of folate, which led to low liver folate levels (16).

One study found 20% of "persistent alcohol misusers" to have abnormally low thiamine-dependent transketolase activity, suggesting an increased requirement for thiamine (15). Thiamine deficiency may lead to Wernicke-Korsakoff Syndrome, a degenerative brain disorder, which affects the heart, vascular system and nervous system. This syndrome is associated with cognitive memory deficit, impairing the retrieval of previous memories and the capacity to create new memories. In addition, vision impairment, coma and ataxia leave the patient further disabled (17). The registered dietitian plays a significant role in screening patients in the prevention and treatment of vitamin deficiencies.

Alcohol Intake and Diabetes

Currently, 43% of the CKD Stage 5 population in the United States has diabetes (18). Experimental studies have been

conducted to assess the relationship between alcohol ingestion and glycemic control in individuals with type 1 and type 2 diabetes (19, 20, 21, 22). When subjects with type 1 and type 2 diabetes consumed one gram of alcohol per kilogram body weight with food, subjects with type 2 diabetes had slightly lower blood glucose concentrations the following morning (19). Many studies have compared the effects of alcohol when fasting versus with food in subjects with type 2 diabetes (20, 21, 22). Hypoglycemia was not experienced in subjects with diabetes after consuming alcohol with food. The studies of light to moderate alcohol doses with or without food showed no short-term effect of alcohol on glycemic control. A statistically significant relationship was found between low blood glucose in persons with type 2 diabetes and alcohol infusion while fasting (22). These findings were consistent with an early study of alcohol and low blood glucose in males with type 1 diabetes (23).

The relationship between alcohol and carbohydrate metabolism is complex and varies according to the amount of alcohol consumed, timeframe of consumption, nutritional status of the individual and presence or absence of food (24). Alcoholic hypoglycemia has been attributed to the inhibition of gluconeogenesis by alcohol (25). Furthermore, alcohol may affect the utilization and production of glucose by interfering with the uptake of lactate (26).

The American Diabetes Association recommends that alcoholic beverages be planned in conjunction with a regular meal (27). In addition, to avoid the risk of hypoglycemia individuals with diabetes should not substitute food with alcohol.

Cardiovascular and Lipid Profile Benefits

Nearly 50% of the deaths of CKD patients may be attributed to cardiovascular disease (28). The "French Paradox," which first captured mainstream attention when highlighted on a *60-Minutes* broadcast in 1992, catalyzed studies of the potential benefits of light to moderate alcohol intake, especially red wine, on the progression of cardiovascular disease (29). Alcohol is known to increase the level of serum high-density lipoprotein (HDL) and lower serum lipoprotein a (30, 31). HDL is protective against atherosclerosis while high amounts of lipoprotein a promote the process (14). One study of alcohol consumption and atherosclerotic

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The Effects of Alcohol *continued from page 8*

risk in type 2 diabetes found that heavy alcohol intake was associated with both increased serum HDL levels (negative risk factor) and high serum triglyceride levels (positive risk factor). However, there was no difference in these two factors between nondrinkers and light drinkers (32). This suggests that the potential beneficial changes in serum lipid profile with light to moderate alcohol intake in nondiabetics may not be translated to the diabetic population.

There have been numerous studies of the cardioprotective value of moderate wine consumption related to antioxidant properties. Some experts are examining the role of oxidative stress, endothelial dysfunction and the intake of wine among CKD patients (33). Currently there is not conclusive evidence to recommend the intake of alcohol, a nonessential nutrient, as a cardioprotective diet modification for individuals abstaining from alcohol.

Cultural and Ethnic Considerations

Heavy drinking is most prevalent among American Indians and Alaska Natives and lowest among Asian Americans and Pacific Islanders (34). Among adolescents, Hispanics have the highest annual prevalence of heavy drinking (35). However, acculturation of ethnic groups causes individuals to assume the drinking patterns of the general population.

There appears to be genetic factors, which affect the body's ability to metabolize and eliminate alcohol (36). It is common among Asian subpopulations to have inactivity of the enzyme aldehyde dehydrogenase-2, leading to flushing of the skin, nausea and uncomfortable symptoms. The potential variation in the enzyme alcohol dehydrogenase-2 among some African Americans may affect the vulnerability of this population to alcoholic cirrhosis and alcohol-related fetal defect (36). There are ethnic differences in drinking patterns, and an understanding of the ethnic context of alcohol consumption should be utilized when working in the community.

Summary

The renal dietitian has the training and expertise to educate dialysis patients about the appropriate consumption of alcohol within the parameters of the prescribed renal nutrition plan. Patients with CKD should discuss the consumption of alcoholic beverages with attending physicians. The cur-

rent literature provides evidence that the negative affects of alcohol outweigh any potential health protective value. In addition, there is a lack of research on alcohol ingestion specific to the CKD population. By providing patients who choose to use alcohol with recommendations and encouraging moderate and safe alcohol consumption, renal healthcare professionals may empower patients to make responsible choices leading to improved quality of life and better health outcomes.

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■ ADVANCES IN PRACTICE: ■ IMPACT OF GRELIN ON NUTRITIONAL STATUS IN PATIENTS ■ WITH CHRONIC KIDNEY DISEASE

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

The high incidence of protein-energy malnutrition (PEM) is well documented in patients with chronic kidney disease (CKD). Its presence is a strong predictor of morbidity and mortality (1-3). Barriers to adequate nutrition intake in this population include accumulation of uremic toxins, co-morbid illnesses, inflammatory conditions, difficulty shopping for and preparing food and decreased palatability of high protein foods (1,2,4,5). In recent years attention has also focused on the roles of long and short-term regulators of appetite in the development of PEM in CKD patients (6).

Insulin and leptin are well known long-term regulators of appetite in persons with normal renal function, and both are secreted in proportion to the amount of stored body fat (7). In animal models, food intake decreases when increased insulin or leptin levels are detected in the brain. In CKD patients, elevated plasma leptin levels have been linked with decreased energy and protein intake, and loss of dry weight and lean body mass (8).

Peptides secreted by the gastrointestinal tract in response to meal size are short-term appetite regulators (7). These peptides include cholecystokinin, gastrin-releasing peptide, somatostatin and bombesin, all of which cause meal termination. Another peptide of interest is ghrelin. It was recently identified and found to promote food intake and weight gain in animals and humans (9). Adding to the interest in ghrelin is its ability to stimulate appetite and increase energy intake in cancer patients with anorexia (10).

This article will review the physiological effects of ghrelin, describe the impact of kidney disease on plasma ghrelin levels and examine the potential of ghrelin to alter nutritional status in CKD patients.

Structure and Physiological Effects

Prepro-ghrelin, the ghrelin precursor molecule, is synthesized mainly in epithelial cells lining the fundus of

the stomach and consists of 117 amino acids (11,13). This precursor molecule undergoes enzymatic splitting in the cytoplasm to yield ghrelin, a 28 amino acid peptide with a molecular weight of 3315. The normal range for ghrelin levels is < 2600 ng/mL. Circulating ghrelin levels are lowest shortly after eating a meal and increase prior to the next meal and during fasting (14). Ghrelin levels are decreased in obesity and elevated in cachexia (12).

Recent studies indicate that ghrelin stimulates the release of growth hormone (GH) from the anterior pituitary gland in healthy subjects and in patients with CKD (12,15). GH has previously been shown to improve serum albumin and muscle strength, promote weight gain and potentiate the effects of intradialytic parenteral nutrition (IDPN) in patients undergoing maintenance hemodialysis (16-18). Ghrelin also functions independently of GH to increase hunger through its action on the feeding center in the hypothalamus, and to suppress fat utilization in adipose tissue (12-14).

Plasma Ghrelin Levels in CKD Patients

Until recently, little was known about the metabolism and clearance of ghrelin or factors affecting circulating ghrelin levels. In a clinical study investigating the effect of kidney disease on ghrelin levels, 46 nondiabetic patients with stages 3 and 4 CKD underwent renal function measurement and radioimmunoassay of their plasma ghrelin (19). Patients with glomerular filtration rates (GFR) ≤ 30 mL/min/1.73m² had elevated ghrelin levels and there was a significant inverse relationship between plasma ghrelin level and GFR. Thus, ghrelin levels seem to increase as GFR declines.

Another study compared ghrelin levels in patients undergoing maintenance hemodialysis (HD) and healthy controls (20). Both groups were of similar age and body mass index (BMI). Ghrelin levels were significantly higher in HD patients than healthy controls (4.49 ± 0.74 ng/mL vs. 1.79 ± 0.15 ng/mL). In addition, HD patients had significantly higher levels of GH and leptin.

These findings were confirmed and extended by a study in which ghrelin levels in HD patients were tracked before and after meal ingestion, and before and after a dialysis treatment

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(21). Again, ghrelin levels were significantly higher in HD patients than in healthy controls. Meal ingestion induced a decrease in ghrelin levels in 5 out of 6 patients tested. HD treatment resulted in a significant decrease in circulating ghrelin, and ghrelin was in the normal range at the end of HD treatment in 4 out of 5 patients tested. Plasma ghrelin concentrations were also significantly higher in normal weight patients compared to overweight or obese patients.

Another investigation used a cross-sectional design to compare plasma ghrelin levels in 20 HD patients and 21 peritoneal dialysis (PD) patients with age-matched healthy controls (22). This study found higher plasma ghrelin levels in both HD and PD patients than in healthy controls, and a strong inverse correlation between ghrelin levels and age.

Collectively, these studies indicate that plasma ghrelin levels are elevated in patients with CKD (19-22). Higher ghrelin levels in patients with CKD compared with healthy controls might be caused by decreased excretion of ghrelin in renal failure. The fact that ghrelin circulates at higher levels in normal weight versus overweight or obese HD patients and decreases after a meal suggests that regulation of ghrelin does occur in CKD, but that secretion is shifted to a higher level.

Ghrelin and Nutritional Status in CKD Patients

Several high molecular weight compounds, including leptin and the proinflammatory cytokines, are elevated in CKD and impact nutritional status in this patient population (23). Findings from a number of studies suggest a relationship between ghrelin, appetite regulation and body composition in patients with CKD.

In an investigation of the relationship between long- and short-term regulators of appetite and food intake in PD patients, 42 patients were divided into three groups: those with anorexia, those with obesity and those with no eating behavior disorders (24). A visual analog scale was used to evaluate the motivation to eat. Thirty-six patients had mean plasma ghrelin levels above the normal range. Patients with anorexia had lower ghrelin levels and higher levels of cholecystokinin. The proinflammatory cytokines tumor necrosis factor alpha and interleukin-1 were also elevated compared to patients with normal appetite or obesity. A

significant positive correlation was found between circulating plasma ghrelin levels and eating motivation.

Results from a recently completed study on energy intake in PD patients with mild to moderate malnutrition indicate that subcutaneous ghrelin administration may increase food consumption (25). Nine PD patients with a mean subjective global assessment (SGA) score of 5.7 ± 1.7 showed significantly increased mean energy intake after receiving subcutaneous ghrelin (3.6 nmol/kg) when compared with placebo. A nonsignificant increase in energy intake over a 24-hour period following intervention was also noted.

The relationship between plasma ghrelin levels and body composition in maintenance dialysis patients has also been investigated (20,26). When HD patients were compared with healthy controls of similar age and BMI, the HD patients had significantly higher percentages of body fat, as well as higher ghrelin levels (20). In another study, a longitudinal evaluation of plasma ghrelin levels and body composition was performed in 52 patients undergoing maintenance dialysis therapy (26). This study found markedly elevated ghrelin levels in both HD and PD patients, and an inverse relationship between plasma ghrelin and circulating leptin and insulin levels. However, while fat mass increased and plasma ghrelin levels decreased after 12 months of dialysis in PD patients, there were no significant changes in either body composition or plasma ghrelin levels over the same time period in HD patients.

Summary

Plasma ghrelin concentrations increase in CKD, probably as a result of reduced renal excretion (13). This finding suggests that ghrelin may play a part in the metabolic abnormalities that develop as a result of uremia (27). A negative correlation between plasma ghrelin levels and nutritional markers suggests a causal relationship between reduced nutrition intake and increased ghrelin secretion in patients with CKD undergoing maintenance dialysis (22).

A number of studies show that malnutrition in CKD is characterized by reduced lean body mass and conservation of fat mass (28,29). The ghrelin imbalance that has been identified in patients with CKD could impact energy

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Sensipar® is BOUNDED to change
the way you treat secondary HPT

Only Sensipar® targets the calcium-sensing receptor to reduce PTH while simultaneously lowering Ca x P, Ca, and P¹⁻³



(Data on file.⁴)

Results from three 6-month, multicenter, randomized, double-blind, placebo-controlled clinical studies conducted in 1136 CKD patients with secondary HPT on dialysis. 30 mg to 180 mg of Sensipar® or placebo was administered once daily, either alone or in combination with vitamin D sterols and phosphate binders, to achieve a mean iPTH ≤ 250 pg/mL. Pooled median baseline iPTH concentrations were 596 pg/mL and 564 pg/mL for the Sensipar® and control groups, respectively.

- Sensipar® enables significantly more patients to achieve the 4 key NKF-K/DOQI™ secondary HPT goals, independent of vitamin D^{5-7*}
- Sensipar® is safe and well tolerated in a broad range of secondary HPT patients on dialysis⁸

FIRST-IN-CLASS Sensipar® (cinacalcet HCl) Tablets 30mg · 60mg · 90mg

*NKF-K/DOQI™ Guidelines for Bone Metabolism and Disease in Chronic Kidney Disease (Stage 5).

K/DOQI is a trademark of the National Kidney Foundation, Inc.

Sensipar® is indicated for the treatment of secondary hyperparathyroidism (HPT) in patients with chronic kidney disease on dialysis. Sensipar® lowers serum calcium. Significant reductions in calcium may lower the threshold for seizures. Secondary HPT patients, particularly those with a history of a seizure disorder, should be carefully monitored for the occurrence of low serum calcium or symptoms of hypocalcemia. The most commonly reported side effects were nausea and vomiting.

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Brief Summary

See package insert for full prescribing information

SENSIPAR® (cinacalcet HCl) 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).

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. In CKD patients with secondary HPT not on dialysis, the long-term safety and efficacy of Sensipar® have not been established. Exploratory investigation indicates that CKD patients not on dialysis have an increased risk for hypocalcemia compared to CKD patients on dialysis, which may be due to lower baseline calcium levels. In a small, short-term study, in which the median dose of cinacalcet was 30 mg at the completion of the study, 74% of cinacalcet treated patients experienced at least one serum calcium value < 8.4 mg/dL. **Adynamic Bone Disease:** Adynamic bone disease may develop if iPTH levels are suppressed below 100 pg/mL when assessed using the standard Nichols IRMA. 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)¹, 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-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 inhibitor of CYP2D6. Therefore, dose adjustments of concomitant medications that are predominantly metabolized by CYP2D6 and have a narrow therapeutic index (e.g., flecainide, vinblastine, thioridazine and most tricyclic antidepressants) may be required. 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. 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 Carcinogenesis: 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 (peri-parturient 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 and vomiting.

Table 1. Adverse Event Incidence (≥ 5%) in Patients On Dialysis

Event*	Placebo (n = 470) (%)	Sensipar® (n = 656) (%)
Nausea	19	31
Vomiting	15	27
Diarrhea	20	21
Myalgia	14	15
Dizziness	8	10
Hypertension	5	7
Asthenia	4	7
Anorexia	4	6
Pain Chest, Non-Cardiac	4	6
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. **Parathyroid Carcinoma:** The most frequent adverse events in this patient group were nausea and vomiting. **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. Overdose of Sensipar® may lead to hypocalcemia. In the event of overdose, 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 iPTH 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. 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-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, 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 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 42:S1-S201, 2003



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regulation and promote the changes in body composition observed in this population. Elevated ghrelin levels in CKD are associated with higher percentage of body fat and this action is the converse of the effects of leptin, which reduces lean tissue and fat mass (8,12). Thus, increasing levels of ghrelin in CKD may counteract leptin's effects (19). Subcutaneous ghrelin administration shows promise in improving short-term food intake in PD patients with mild to moderate malnutrition. Currently, measuring ghrelin levels and ghrelin administration is not routine in the CKD population. Future studies should be directed to determine the specific effects of elevated ghrelin levels on metabolism in CKD and the long-term effect on food intake of ghrelin administration.

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KIDNEY FRIENDLY FACTS FOR PATIENT EDUCATION - FALL 2005

By Sharon Schatz, MS, RD, CSR, CDE. *Sharon is a renal dietitian with Gambro Healthcare in Lumberton, N.J.. She can be reached at Srsmrd@aol.com or sharon.schatz@us.gambro.com.*

Fall is T Time! T = Thanksgiving = Turkey! Did you know that turkey breast has more protein and less fat per serving than other meats? Its versatility is similar to chicken, as it can be purchased whole or by parts, and it can be prepared in countless ways. Thanksgiving is an ideal time to provide your CKD patients with recipes, cooking ideas, and food safety information. Suggestions for Tasty Turkey Left-Overs are provided with this column.

Understanding how to interpret nutrition labels is an important aspect of purchasing a turkey. Many processed turkey products are often enhanced with sodium. These include pre-basted turkeys, processed turkey rolls, and smoked turkey. A serving of enhanced meat can contain up to 500 mg of sodium per serving. Refer to "Enhanced Meats – A Hidden Source of Sodium" on [iKidney.com](#) for additional information. Several of the web sites listed provide nutrient data for their products to help guide selections.

For Thanksgiving recipes, *Thanksgiving 101* by Rick Rodgers (Broadway Books, NY, 1998) is a great resource. If you'd like to learn more about the history of Thanksgiving check out [http://www.pilgrimhall.org/thanksg2.htm](#) at the Pilgrim Hall Museum or [http://www.historychannel.com/exhibits/thanksgiving/](#) at the History Channel.

To find ideas for patient hand-outs, the following web sites may be useful:

The eGG's (electronic Gourmet Guide) Perfect Turkey Handbook can be found at [http://www.globalgourmet.com/food/egg/egg1197/perfturk.html](#). This has ten chapters that range from buying a turkey to storing leftovers!

For more information regarding turkey food safety go to the University of Illinois Extension site at [http://www.urbanext.uiuc.edu/turkey/safety.html](#)

The National Turkey Federation, [http://www.eatturkey.com/](#) touts turkey as the perfect protein. It has a database of 1500 recipes that can be searched by dish type, ethnicity, meal type, occasion, preparation method, and product type.

The Thanksgiving Guide Leftover Alchemy at [http://www.foodnetwork.com](#) allows you to search for recipes using specific ingredients.

Additional web sites with recipes are: [http://www.bhg.com/home/turkey-recipes.html](#), [http://thanksgiving.allrecipes.com/](#), [http://www.bhg.com/home/turkey-recipes.html](#), [http://www.southernfood.about.com/cs/turkeyinformation/a/leftover_turkey.htm](#).

The website [http://www.butterball.com](#) provides information about their products, recipes, "plan, prep, and serve", and a sign up form for Turkey Lovers e-mail newsletter.

The Here's How section in [http://www.honeysucklewhite.com](#) tells you "everything you ever wanted to know about preparing turkey" including "whole turkey know how", preparation techniques, and "how to cook fresh turkey cuts." There is also a recipe of the month newsletter.

At [http://www.jennieoturkeystore.com/](#) find Turkey Tips for buying, preparing, cooking, carving, and gravy.

Informative "how to" tips and interesting turkey facts and statistics can be found at [http://www.norbest.com/index.cfm](#).

The use of Rock Cornish game hens could be suggested to patients in lieu of a larger turkey. They look like miniature chickens and have a similar flavor. The chicken mogul Tyson created them in 1965 by cross-breeding White Rock hens and Cornish hens. The hens can be substituted in most standard chicken recipes with little modification, but they are usually best suited to roasting. They may be easier to prepare than a whole turkey, as they are lighter to lift in and out of the oven. This type of poultry may also fit the bill for a short guest list.

All of the websites above were accessed on October 10, 2005.

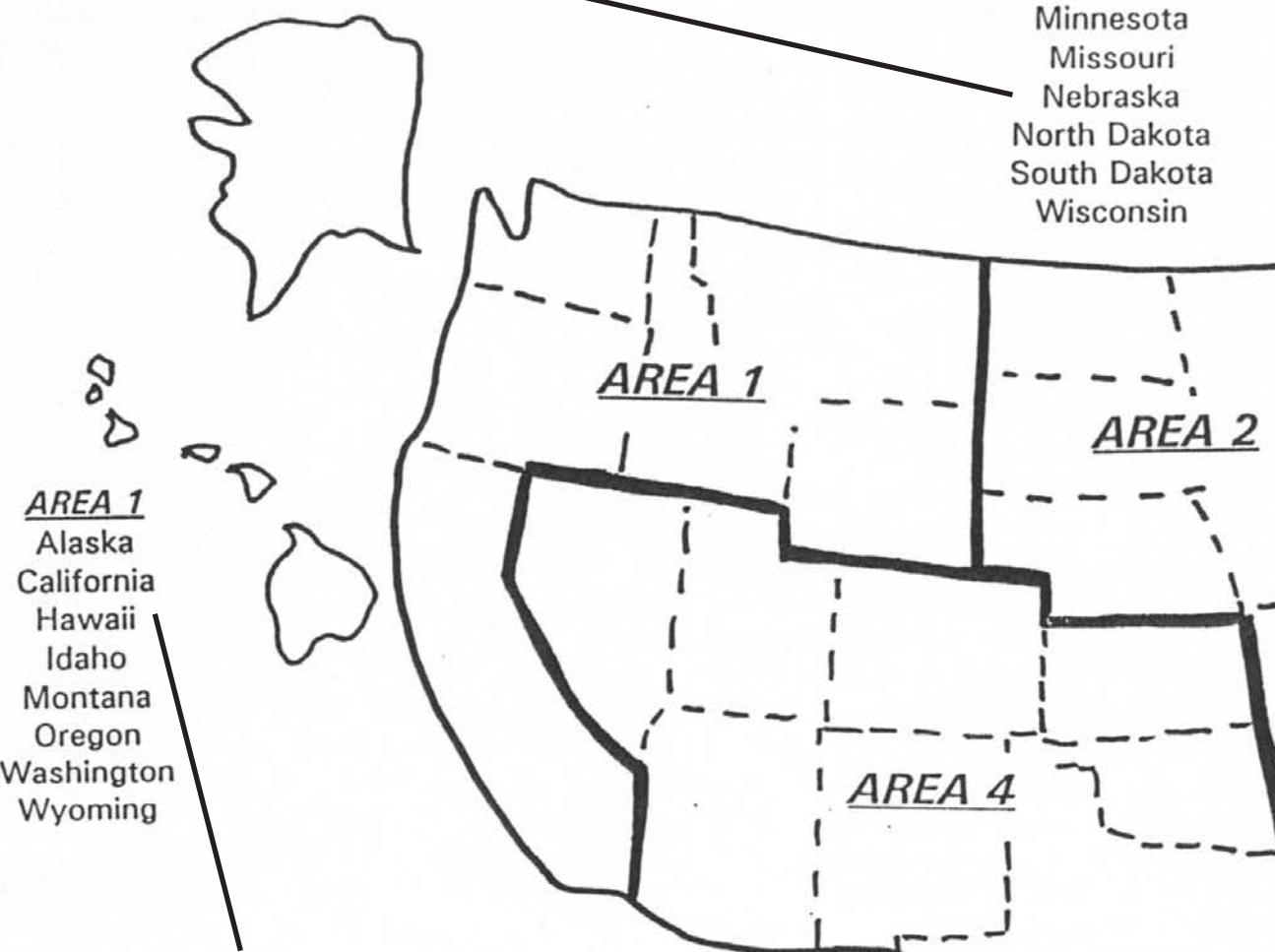


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Mary Jo is the Area II Coordinator and the Awards and Scholarship Chairperson. Mary Jo has been a renal dietitian since 1995. Currently she works for DaVita Dialysis. Mary Jo has worked with CKD patients in numerous settings including inpatient acute care and out patient clinics following patients from early stage CKD through peritoneal and hemodialysis as well as post transplant.

AREA 2

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South Dakota
Wisconsin



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Chhaya is the Area I Coordinator as well as the CQI Outcomes Chair. Chhaya has been a registered dietitian and practicing as a renal dietitian for over 25 years. Currently, she is working for the DaVita Corporation as a Divisional and Area dietitian for the Sierra Pacific Division. She has volunteered for CRN/NKF and RPG/ADA for over 20 years on both the local and national levels. In addition, Chhaya has contributed over 20 articles for publication, lectures and also consults for various organizations and corporations.

AREA 4
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Colorado
Kansas
Nevada
New Mexico
Oklahoma
Texas
Utah



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Lisa is the Area V Coordinator and manages the RPG Lending Library for the Eastern U.S. (Areas 3, 5, 6, 7). Lisa has been a registered dietitian for almost 20 years and has been a renal dietitian for 18 years working with both in-patient and outpatient CKD patients. Lisa is active with NKF and CRN on both the local and national level.



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Patricia is the Area VII Coordinator and the Historian Chairperson. Patti was instrumental in the coordination and affiliation of the first CRN of NE. She has been involved with ADA-RPG for many years and is one of the past Chairpersons. In her role as historian, she is interested in obtaining pertinent historical information from members and officers (past and present) for the RPG archives. Patti is currently employed as a renal dietitian with Gambo Healthcare.



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Karen is the Area VI Coordinator and Chair of the Legislative/Reimbursement Committee. She is very involved with legislative issues pertaining to chronic kidney disease. Her most recent project is the CMS Guidelines for the requirements of the dietitian. Karen has been a Renal Dietitian since 1981. She is a member of both RPG and CRN. In addition to her work with CKD, she lectures, consults and provides outpatient MNT.



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Patricia is the Area III coordinator and the Education Chairperson. Patricia has been a renal dietitian for eight years and currently works with Renal Care Group. In addition to being affiliated with ADA and RPG, Patricia is a member of CRN both on a local and national level. She is a member of the 2006 NKF-CRN Program Committee.

Tasty Turkey Left Overs

Turkey and Pepper Fajitas:

Sauté red onion, green and red bell pepper strips, and minced garlic in hot vegetable oil until very soft. Add turkey strips and cook until heated through. Stir in chili powder to taste. Roll up in warm flour tortillas. Add small amounts of salsa and sour cream.

Sweet and Zesty Turkey Sandwich:

Mix cranberry sauce with Dijon mustard (use more, if you like it spicier) to make a sweet and zesty sandwich spread. Smear the cranberry mustard on a split Kaiser roll or hamburger bun and top with turkey slices and lettuce.

Turkey Wrap:

Spread softened cream cheese onto a flour tortilla. For more zip, stir a small amount of cream-style horseradish into cream cheese before spreading onto tortilla. Then spread jellied cranberry sauce over the cream cheese. Top with strips or thin slices of turkey and roll the tortilla into a wrap.

Tropical Turkey Salad:

Toss together chopped turkey, 1 small can drain mandarin orange sections, 1 cup halved grapes, and 1 small can drained pineapple chunks. Combine ½ cup low fat mayonnaise with 2 TBSP honey and 1/8 tsp ground ginger. Blend dressing into turkey mixture. Serve on lettuce leaves or shredded green cabbage.

Curried Turkey Salad:

Mix diced turkey with mayonnaise, chopped apples, dried cranberries, and curry powder (to taste). Serve on lettuce leaf or stuff a pita pocket.

Turkey Pasta Salad:

Combine chunks of left-over turkey with cooked corkscrew pasta and lower potassium chopped vegetables such as cucumbers, green and red peppers, red onion, and cooked green beans. Sprinkle with salt free Italian seasoning blend and toss with olive oil and red wine vinegar.

Turkey Casserole:

Combine left-over turkey and stuffing with cooked sliced carrots and green peas in a casserole dish. Heat until warm. Can be served with low sodium gravy or cranberry sauce, if desired.

Pineapple-Cherry Turkey:

In small saucepan combine ½ cup crushed pineapple in its own juice with 3 Tablespoons cherry preserves (those with diabetes could use low sugar version) and 1 teaspoon ground ginger. Bring to a boil over medium heat while stirring and then simmer for a few minutes. In a small cup mix 1 teaspoon cornstarch with 1 Tablespoon water, and then pour this gradually into simmering pineapple-cherry sauce; stir until thickened. Pour sauce over warmed sliced turkey. Serve with rice.

Proven ReZults

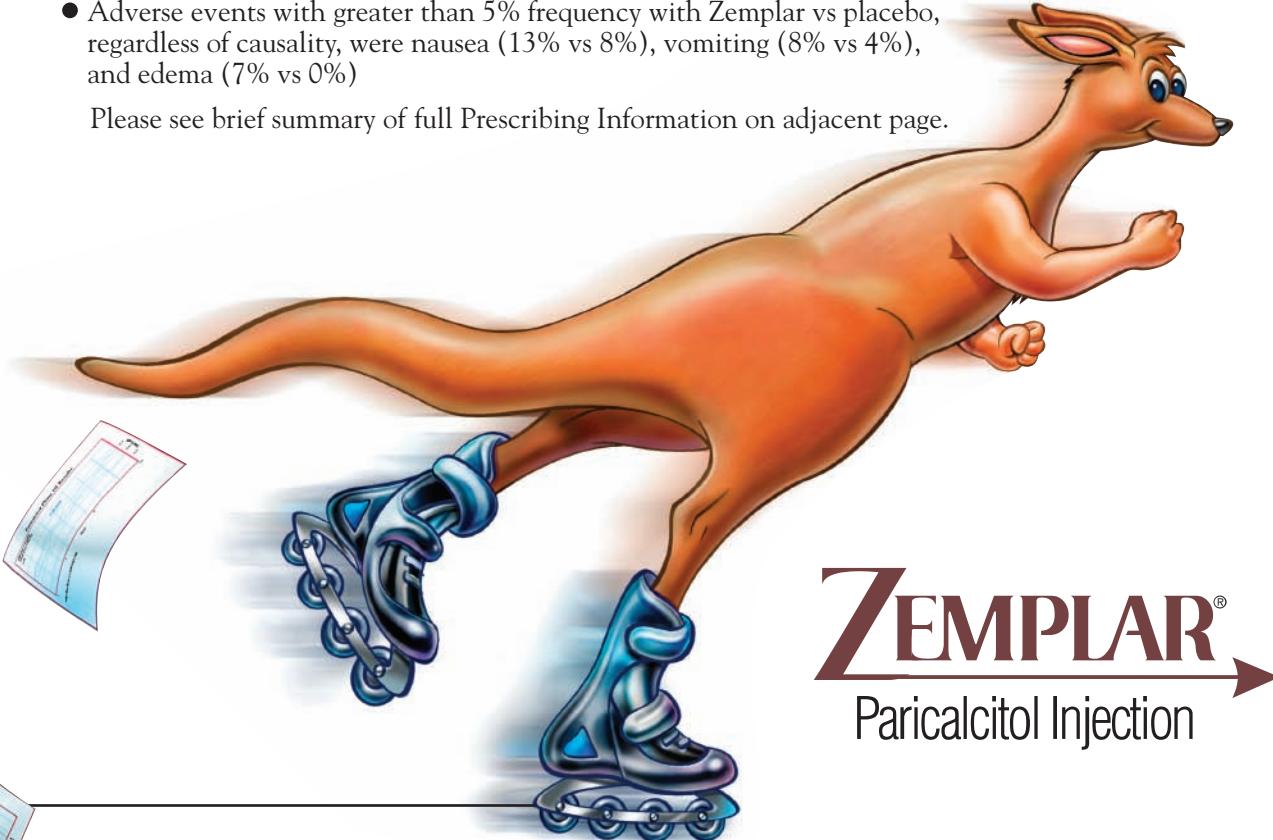
There is more to know about Zemplar...

- Clinically studied in over 700 patients¹⁻⁶
- PTH outcomes: proven to safely and rapidly reduce PTH levels with no significant difference in the incidence of hypercalcemia or hyperphosphatemia when compared to placebo²
- Zemplar is indicated for both the treatment and prevention of secondary hyperparathyroidism associated with chronic renal failure

Important Safety Considerations

- Zemplar is contraindicated in patients with vitamin D toxicity, hypercalcemia, or hypersensitivity to product ingredients
- Administration may place patients at risk for hypercalcemia, elevated Ca × P product, and metastatic calcification
- Adverse events with greater than 5% frequency with Zemplar vs placebo, regardless of causality, were nausea (13% vs 8%), vomiting (8% vs 4%), and edema (7% vs 0%)

Please see brief summary of full Prescribing Information on adjacent page.



ZEMPLAR®
Paricalcitol Injection

 Abbott Laboratories
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REFERENCES: 1. Clinical data on file, 2003. 2. Martin KJ, et al. *J Am Soc Nephrol*. 1998;9:1427-1432. 3. Martin KJ, et al. *Am J Kidney Dis*. 1998;32:s61-s66. 4. Lindberg J, *Clin Nephrol*. 2001;56(4):315-23. 5. Martin KJ, et al. *Am J Kidney Dis*. 2001;38(5):s57-s63. 6. Sprague SM, et al. *Kidney Int*. 2003;63:1483-1490.

BRIEF SUMMARY
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Zemplar®

(paricalcitol injection, USP)

Fliptop Vial

Rx only

INDICATIONS AND USAGE

Zemplar® is indicated for the prevention and treatment of secondary hyperparathyroidism associated with chronic renal failure. Studies in patients with chronic renal failure show that Zemplar® suppresses PTH levels with no significant difference in the incidence of hypercalcemia or hyperphosphatemia when compared to placebo. However, the serum phosphorus, calcium and calcium x phosphorus product (Ca x P) may increase when Zemplar® is administered.

CONTRAINDICATIONS

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

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 for hypercalcemia, elevated Ca x P product, and metastatic calcification. 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.

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 renal failure (CRF), but excessive use of aluminum containing compounds should be avoided. Patients should also be carefully informed about the symptoms of elevated calcium.

Essential 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 CRF. During dose adjustment of Zemplar®, laboratory tests may be required more frequently.

Drug Interactions

Specific interaction studies were not performed. 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 to 10 mcg/kg (< 1 to 3 times the maximum recommended human weekly dose of 0.72 mcg/kg, based on body surface area, mg/m²). 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 of rats, there was an increased incidence of benign adrenal pheochromocytoma at subcutaneous doses of 0.15 to 1.5 mcg/kg (≤ 1 times the maximum recommended human weekly dose of 0.72 mcg/kg, based on body surface area, mg/m²). The increased incidence of pheochromocytomas in rats may be related to the alteration of calcium homeostasis by paricalcitol. In carcinogenicity studies in rats and mice, paricalcitol did not affect the incidences of tumors apart from benign rodent-specific lesions related to the effects of chronic hypercalcemia.

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/m²].

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/m²) 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 justifies the potential risk to the fetus.

Nursing Mothers

It is not known whether paricalcitol is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when Zemplar® is administered to a nursing woman.

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 15-19 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 – 9.5 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 40/5 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 75 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 CRF 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 CRF 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.

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

OVERDOSAGE

Overdosage of Zemplar® may lead to hypercalcemia (see **WARNINGS**).

Revised: October, 2004

Ref: EN-0423 (10/04)

04L-130-F901-1 **MASTER**

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I would like to welcome all new members to RPG as well as all returning members! We have a busy year ahead of us and much to look forward to. RPG had quite a presence at FNCE. This year's meeting was held in St. Louis, Oct 22-25th. Dr. Kevin Martin from St. Louis University and Catherine Goeddeke-Merickel, MS, RD spoke on the role of Vitamin D in Chronic Kidney Disease. The session was held on Tuesday, October 25, 2005 and was entitled "No Bones about it: The role of Vitamin D in Chronic Kidney Disease". In addition, I hope those of you who attended the RPG Member Breakfast met the Executive Board and enjoyed a great breakfast and networking opportunity. The DPG Showcase offered many opportunities to network and see new materials, including the debut of the Spanish simplified renal diet.

Looking ahead, a workgroup has been formed to draft Standards of Practice and Standards of Professional Performance for Chronic Kidney Disease. This is a joint

project with the Council on Renal Nutrition of the National Kidney Foundation. The workgroup members include myself, Deborah Brommage, MS, RD, Jenny Smothers, RD, LD, Jessie Pavlinac, MS, RD, CSR, Maria Karalis, MBA, RD, Debbie Benner, MA, RD, Karen Wiesen, MS, RD, Linda McCann, RD, CSR, Jennie House, RD, LD, Maureen McCarthy, MPH, RD, CSR, and Laura Byham-Gray, PhD, RD, CNSD. The project is expected to be accomplished within a year and will be published in the Journal of the American Dietetic Association upon completion.

RPG is seeking interested and enthusiastic members that would like to be involved at the Executive Committee level. I would like to personally encourage you to consider running on the ballot for Chair-Elect, Secretary or Nominating Committee. If you are interested, please send an email to Susan Knapp, MS, RD, Chair of the Nominating Committee at sknapp@intcon.net.

CRN CHAIR MESSAGE

The Power of Alliancing

Deborah Brommage

Attending meetings and other networking opportunities have their merit, but according to Carol Gallagher, Ph.D., forming meaningful alliances can be far more rewarding. In her book *Going to the Top*, Dr. Gallagher shares the insights of 200 of America's highest level female executives on career advancement. In a chapter titled "Create Alliances: Networking Is Not a Requirement for Success", the author dispels the myth that networking is used by successful executive women to get ahead. Rather, she coins the term alliancing to describe the activity of developing meaningful relationships that advanced these female executives forward in their careers.

Alliancing vs. Networking

Networking functions are charged with generating relationships that are usually too superficial to be significant. Dr. Gallagher reports that more than 90% of female executives

said that their professional relationships came from collaborative work on projects rather than from networking. So, the difference between networking and alliancing is that knowing people is not enough, it's how well you know them that matters. In other words, having a Rolodex full of contacts does not have meaning unless a connection or common bond is formed with each person.

Female executives reveal that working on a project in a group setting with colleagues provides a way to exhibit personal character and ability. Collaborative activities result in producing something together while building trust, and learning about each others' values and beliefs. Trust in particular is deemed as an important attribute in order to get

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ahead. Working with a deadline can also prove your ability to work under pressure. The author also reminds us that teamwork is the best way to get things done effectively.

Make Alliancing Work for You

The two main areas to form alliances are at work (internal alliances) and outside of work (external alliances). In order to get things done, good relationships within the organization you work for are important. Relationships with executives, immediate bosses, cohorts, peers, and assistants are included as internal alliances that can influence your career. Forming alliances with people outside of your department is considered as important as the relationships built within your appointed area. Interaction with people in other departments allows sharing of information thereby giving insight into vital company issues, while building team pride.

External alliances allow us to learn from people outside of our organization. Dr. Gallagher relates that this knowledge gives us the ability to think outside the box and improve the contributions we make to our organization. One female executive shared that her company considers her a valuable resource because she knows how things are being done elsewhere. While most of us are probably comfortable with co-workers to develop internal relationships, alliances outside of work may be more of a challenge. Fortunately for

renal dietitians NKF and ADA exude with opportunities to build meaningful external alliances. Having been a member of the CRN Executive Committee for the past 5 years I can tell you first hand that I have had the privilege of developing long lasting relationships with renal dietitians from across the country.

Local CRN chapters and ADA affiliates are good places to start. These groups hold professional meetings which are great for networking. Use meetings to develop and build alliances by running for Executive Committee positions and volunteering for committee projects. In particular, sitting on an executive board at the local level offers the opportunity to work with colleagues to build professional relationships outside of your company. Volunteering for activities with local NKF Affiliates is another way to form external alliances. Many NKF Affiliates have Program Planning Committees, Professional Advisory Boards and other multidisciplinary activities to contribute your talent. These activities provide the opportunity to help people with kidney disease while developing meaningful external alliances.

Reference

Gallagher C. *Going to the Top*. New York, NY: Penguin Books; 2000.

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DON'T FORGET TO VOTE!

Participate in the ballot process by voting for RPG 2006-2007 elected officers:

- **Chair-Elect**
- **Secretary**
- **Nominating Committee Member**

- **Online voting goes live January 15, 2006**
- **The last day to vote is February 28, 2006**
- **Paper ballots are available upon request only at practice@eatright.org**
- **Paper ballots must be postmarked or faxed by February 24, 2006**

Visit www.renalnutrition.org and cast your vote

Please cast your vote and make a difference!!!

2005-2006 RPG Executive Committee

RPG Mission: The RPG is the advocate of the nutrition profession serving the public through the promotion of optimal renal nutrition, health and well-being.
RPG Vision: RPG members will be leaders in providing scientifically sound renal nutrition care and education for patients, the profession and the public.

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