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NON-DAIRY BEVERAGE

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Join us at the RPG breakfast at FNCE next October, sponsored by Diehl, Inc., makers of Vitamite" 100 non-dairy beverage. Contest winners will be announced at the breakfast. Look for more information on the breakfast in the fall issue of this newsletter

Contest rules and information: To enter, create an original recipe appropriate for renal patients which uses at least 1/2 cup of Vitamite" 100, liquid or 1/4 cup of the powder form. Recipes must fit into one of these categories: entrée, soup, side dish or dessert. Winners will be announced at the RPG breakfast at FNCE in San Antonio, TX and by mail/phone if not present at the breakfast. First place winner will receive a check for $200, second place will receive $100 and third place will receive $75. Two runners-up will receive non-cash prizes. Send typewritten entries, along with your name, address and phone number to: Vitamite"100 Recipe Contest, c/o K. Broihier, 42 Stanley St., S. Portland, ME 04106. **All entries must be received by September 1st, 2003.** All decisions are final. All recipes become the property of Diehl, Inc. and may be reproduced in printed materials or electronically without permission from the recipe's creator.

Nutritional Comparison For Vitamite 100 vs. Vitamite Ultra

	Vitamite 100	Vitamite Ultra
Serving Size	240 mls - 8 oz	240 mls - 8 oz
Per Serving Analysis		
Calories	100	110
Calories from Fat	45	15
Total Fat	5g	1.5g
Saturated Fat	0g	1.5g
Polyunsaturated Fat	0g	1g
Monounsaturated Fat	3.5g	0g
Cholesterol	0g	0g
Sodium	120mg	230mg
Potassium	140mg	290mg
Total Carbohydrates	14g	16g
Dietary Fiber	0g	3g
Soluble Fiber	0g	0g
Sugars	4g	7g
% Daily Values are based on a 2,000 calorie diet		
Protein	3g	6.25g
Vitamin A	10%	10%
Vitamin C	0%	8%
Calcium	30%	30%
Iron	2%	6%
Vitamin D	25%	25%
Vitamin E	0%	25%
Thiamin	10%	20%
Riboflavin	15%	20%
Niacin	0%	2%
Vitamin B6	10%	15%
Vitamin B12	30%	30%
Pantothenic Acid	10%	15%
Phosphorus	15%	20%

10/11/02

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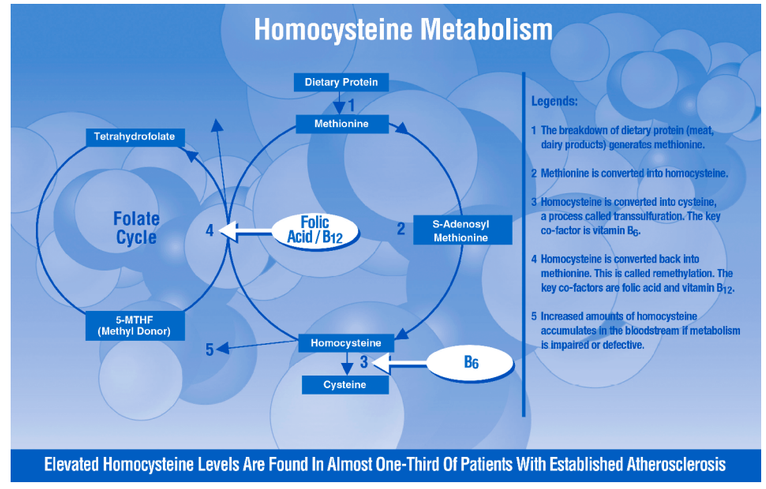
VOLUME 22
NUMBER 3
SUMMER 2003

Homocysteine: The Newest Uremic Toxin?

By Louise Clement, MS, RD, CSR, LD
Louise has worked in an out-patient dialysis unit for 17 years. She has served on the Medical Review Board for Network 14 in Texas, and teaches undergraduate nutrition courses at Texas Tech University. She can be reached at 806-799-2992.

Cardiovascular Disease



Elevated Homocysteine Levels Are Found In Almost One-Third Of Patients With Established Atherosclerosis

Homocysteine is gaining more notoriety as research reveals its role in the pathogenesis of cardiovascular disease (CVD). Recent studies have demonstrated that homocysteine plays a role in other disease states, including chronic kidney disease (CKD) and Alzheimer's. The focus of this article is to review the physiology of homocysteine, its role in cardiac disease and CKD, treatment options, and the results of a quality improvement evaluation.

Physiology

Homocysteine is a sulfur-containing intermediate of the essential amino acid methionine. It plays a key role in the synthesis of nucleic acids that affects all new cell production, including red blood cells. Homocysteine was first described in 1932, but the biochemical finding of homocysteinuria occurred in 1962.

In human physiology, methionine is converted to homocysteine by a process called demethylation and can then be metabolized by one of two pathways. In the trans-sulfuration pathway, which is dependent on vitamin B6, it is converted into cysteine and inorganic sulfates. In the remethylation pathway, homocysteine is converted back to methionine with the aid of folate and vitamin B12-dependent enzymes. **Figure 1.**

Elevated levels of homocysteine can result from either nutritional inadequacies of folate, vitamin B6, vitamin B12, or from inborn errors of metabolism. The latter are rare genetic defects of one of the metabolic pathways in the conversion of homocysteine. One of these, a metabolic defect of cystathionine beta synthase, an enzyme containing vitamin B6, was described in 1964. Historically, children with these defects have suffered from accelerated atherosclerosis and thrombotic events. This was the first indication that homocysteine was involved in the etiology of cardiovascular disease (1).

Excess homocysteine directly injures vascular endothelial cells, and causes lesion formation. Affected cells become pro-thrombotic by releasing excess tissue factor. Surplus homocysteine also promotes platelet aggregation. The initiation of procoagulant reactions can produce acute events such as myocardial infarction, deep vein thrombosis and thrombotic stroke. Lastly, in the presence of homocysteine, oxidized low density lipoproteins penetrate endothelial cells with less difficulty and are deposited to form foam cells. This constellation of abnormalities helps explain the relationship seen between homocysteine levels and peripheral artery disease, coronary artery disease, and the aforementioned venous thrombosis and stroke (2).

Elevated homocysteine levels can also predict future cardiovascular events, including death. In a Norwegian study

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(3), 587 adults with diagnosed coronary artery disease were followed for a mean of 4.6 years. Cause of death was compared to homocysteine levels that were drawn at the beginning of the study. A marked association was demonstrated between mortality rates and four classifications of homocysteine: <9.0, 9.0–14.9, 15.0–19.9, and >20.0 mmol/L. While the mortality rate of persons with homocysteine levels below 9.0 was 3.8%, it was 24.7% for those with levels of >15.0.

After adjustment for gender and age, the factors with the strongest predictive power to homocysteine were serum folate, creatinine, uric acid, vitamin B12, and left ventricular ejection fraction.

Homocysteine and Alzheimer's

Elevated homocysteine levels have been implicated in additional disease states including Alzheimer's and other forms of dementia. In a study by Seshadri et al (4), 1092 persons from the Framingham Study, without dementia, were followed for a median of 8 years. The incidence of dementia was compared with homocysteine levels drawn before and after the study period. The authors concluded that an elevated homocysteine was a strong, independent risk factor for developing Alzheimer's and other forms of dementia. A 5-point increase in the homocysteine level raised the risk of Alzheimer's by 40%. The risk of Alzheimer's was nearly doubled when homocysteine levels were above 14 mmol/L.

Role of Folic Acid

The role of folic acid in homocysteine metabolism is well known. Folic acid serves as a methyl donor in the remethylation pathway, being "consumed" by that chemical process. In the dialysis population, supplemental folic acid helps, but does not normalize elevated homocysteine levels. Bostom, et al described results from an eight week trial providing 15 mg of folic acid, 100 mg of vitamin B6 and 1 mg of vitamin B12 for a group of 27 persons on dialysis, who were already consuming a standard renal vitamin (1 mg folic acid, 10 mg of B6, and 12 mcg of B12). After 8 weeks, the subjects experienced a 25.8% drop in homocysteine levels, from 29.5 to 21.9 mmol/L (5). The lower value is still more than twice the recommended level established by the American Heart Association of <10.0 mmol/L. Ten of the 15 subjects ended the study with homocysteine values above 15 mmol/L (5). In another study by Moustapha (6), plasma folate levels in 130 persons on hemodialysis were negatively correlated with the homocysteine levels, and were significantly lower than the folate levels of the 46 study participants on peritoneal dialysis.

Role of Vitamin B6

Vitamin B6 (pyridoxine) is involved in the trans-sulfuration pathway of homocysteine metabolism. By itself, it does not seem to decrease the elevated homocysteine levels that are seen in End Stage Renal Disease (ESRD). Arnadottir et al (7) provided 300 mg of pyridoxine daily for four months to a group of 12 persons on hemodialysis and six persons on peritoneal dialysis, who received no other vitamin supplementation. Homocysteine levels rose from a mean of 26.7 to 32.9 mmol/L during the four months. It was postulated that the increase was due to a concurrent decline in blood folate levels, which dropped from 780 to 423 mmol/L during the same time period. A previous folic acid supplement had been discontinued four months prior to the pyridoxine trial.

Another study analyzed the effects of pyridoxine in non-dialyzed white males with creatinine clearances between 10 and 80 ml/min. The provision of 70 mg of vitamin B6 without other vitamin supplementation did not significantly decrease homocysteine levels (8).

Although pyridoxine supplementation does not independently decrease homocysteine levels in CKD, its use should not be ignored. Suboptimal levels of vitamin B6 in both pre-dialysis and dialysis patients are well documented, related to uremic toxin interference, dialysis losses, insufficient dietary intake, and potential drug interference (9). Tremblay et al (10) reported vitamin B6 deficiency in nearly 40% of a group of 65 persons on hemodialysis, who had previously received no vitamin supplementation.

Role of Vitamin B12

Vitamin B12 (cobalamin) is primarily absorbed by active transport in the terminal ileum, mediated by intrinsic factor in gastric acid. Various plasma proteins, plus two transport proteins, transcobalamin I and II, carry cobalamin to peripheral tissues. Vitamin B12 status can be compromised by abnormalities such as atopic gastritis, gastrectomy, use of Omeprazole, Crohn's disease, and/or genetic deficiencies or antibodies to intrinsic factor or transcobalamin I or II.

Interestingly, one percent of large oral doses is absorbed by passive diffusion. Kuzminski et al concluded, in a randomized study, that a high oral cobalamin dose of 2 mg was as effective as intramuscular injections for vitamin B12 repletion (11).

Vitamin B12 is needed in the remethylation pathway of homocysteine to methionine. While folic acid acts as a substrate by providing the methyl group, vitamin B12 acts as a cofactor in this reaction.

A negative correlation has been shown to exist between homocysteine and vitamin B12 levels in persons on hemodialysis (6). Vitamin B12 is also removed during dialysis. Chandra et al (12) demonstrated a significant decrease in serum B12 levels after 12 months of hemodialysis that could lead to a functional vitamin B12 deficiency. This further exacerbates rising homocysteine levels, according to Herrmann et al (13). These authors also noted that methylmalonic acid detects intracellular vitamin B12 deficiency with more sensitivity than serum vitamin B12 levels (13).

Homocysteine in Chronic Kidney Disease (CKD)

The kidney itself plays a key role in homocysteine metabolism, but not in its excretory capacity, as less than 1% of homocysteine is actually excreted in the urine (14). Animal studies suggest that kidney tissue contributes substantially as a site of homocysteine metabolism (15). Rising creatinine

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Rehab Corner

By Maureen McCarthy, MPH, RD, CSR *Maureen is a Renal Dietitian with Renal Care Group– Pacific Northwest Renal Services, Portland, OR and can be reached at mmccarthy@renalcaregroup.com.*

This column features an article submitted to the web page of the Life Options Rehabilitation Advisory Council (LORAC) by Jo Reeder, PT, MCSP, of UVA Health Systems. For other articles filled with helpful ideas, go to LORAC's home page (www.lifeoptions.org), select the "For Renal Professionals" link, and then select "Showcase of Ideas".

Getting PT Services for Your Patients *By Jo Reeder, PT, MCSP*
Physical therapy (PT) can be a valuable resource for the dialysis unit team as it treats patients' physical decline. A variety of payers will cover PT services, but certain procedures must be followed.

How Physical Therapy Services Are Prescribed In most states, a physician must make the actual PT referral. However, any health care professional (i.e., RN, RD, SW, PCT) can recommend physical therapy to a patient's doctor. PT referrals are for functional decline, decreased muscle strength, decreased joint range, gait problems, new onset of stroke or head injury, and/or a wound affecting function.

After the referral and a patient evaluation, the physical therapist develops a written plan of therapy. This plan includes the diagnosis, treatment plan, goals of therapy, and frequency and duration of the intervention. The physician reviews and approves the plan of care; there is an expectation that the condition will improve in a reasonable length of time.

Most insurance providers allow a set number of visits, and this varies from company to company. If your patient has commercial insurance, check with the patient's insurance carrier about coverage for physical therapy. There are also limitations on Medicare coverage of physical therapy. If your patient has Medicare, a physical therapy provider or the Center for Medicare and Medicaid Services (CMS) regional office can give you information about Medicare coverage for physical therapy services. You can find the phone number for your CMS (formerly HCFA) regional office at www.medicare.gov/Contacts/Home.asp.

The American Physical Therapy Association (APTA) has posted information about reimbursement for physical therapy services on its website at www.apta.org/reimbursement. You can find a chart of state regulations pertaining to direct access to physical therapy evaluation, examination, and intervention at www.apta.org/pdfs/gov_affairs/directalaws.pdf.

Where Physical Therapy Services Are Provided
Dialysis unit staff can help patients access PT services in different settings. Patients can go to a free-standing, outpatient physical therapy clinic; go to an inpatient rehabilitation center; receive home health physical therapy if they qualify as "homebound" (for definition, see www.hcfa.gov/pubforms/transmit/a0121.pdf); or they can go to the physical therapy department of a hospital.

At University of Virginia Health Systems, for example, physical therapy is provided within the dialysis center so the patients can get their physical treatment before, during, or after dialysis. This on-site PT has been very effective for both patients and staff. It is easier to facilitate on-site physical therapy in a hospital-based unit, but with a little extra planning it can be achieved in a free-standing unit.

Physical therapy intervention can break the spiral of debilitation and decompensation frequently observed in the ESRD population. In addition, a more physically active, higher functioning end-stage renal disease population may have significantly better long-term survival rates.

Jo Reeder, PT, MCSP is willing to reply to readers' questions and can be reached at jr3f@virginia.edu.

Do you have a rehab success story highlighting the role of renal dietitians in the process? Contact Maureen McCarthy for an interview that may lead to an article in this column (mmccarthy@renalcaregroup.com; or phone 503-250-5011).

Kidney Friendly Food Facts

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tablespoon honey and ½ cup mayonnaise. Chinese mustard – mix ¼ cup mustard powder with 2 tablespoons cold water. For yellow color add a little turmeric. This is good with cold shrimp.

Hot dogs go with mustard. Most hot dog recipes combine meat, meat fat, and a cereal filler with herbs and seasoning. The ground ingredients are stuffed into a casing. Although we often counsel patients to avoid hot dogs due to sodium content, we may also need to look at the potassium and phosphorus contents. Per ESHA the average hot dog (57 grams) has 6.4 grams protein, 650 mg sodium, 89 mg potassium, and 91 mg phosphorus. However, not all hot dogs fall into this range as I discovered web surfing (refer to Table 1). Data isn't always available for potassium and phosphorus, but what I found made me rethink the issue. The phosphorus and potassium content could be higher depending upon the filler used or whether potassium phosphates have been added. Referring to carbohydrate content might help indicate this. Some brands are much lower in sodium. Realistically patients are probably eating hot dogs, so perhaps we need to advise them how to do it.

Table 1 – Hot Dog Nutrients

Brand	Wt-gm	Prot-gm	Fat-gm	Carb-gm	Sodium-mg	K+ -mg
Boars Head	N/A	N/A	N/A	N/A	N/A	N/A
Beef Frankfurters, natural casing	57	7	14	1	440	N/A
Beef Frankfurters, skinless	45	6	11	0	350	N/A
Lite Beef Frankfurters	45	7	6	0	270	N/A
Miller's	N/A	N/A	N/A	N/A	N/A	N/A
Colossal Beef Frankfurters, skinless	56	7	11	3	530	N/A
Farmer John	N/A	N/A	N/A	N/A	N/A	N/A
Wieners, pork & beef	46	6	11	N/A	470	N/A
Beef franks	46	5	12	N/A	470	N/A
Dodger Dogs, extra long	76	8	21	N/A	780	n/a
Berks	N/A	N/A	N/A	N/A	N/A	N/A
Grill Meat Franks	57	7	16	0	590	N/A
Beef Franks	57	7	14	0	590	N/A
Ball Park	N/A	N/A	N/A	N/A	N/A	N/A
Beef Franks	56	6	16	3	620	460
Franks, beef & pork	56	6	16	3	610	450
Kosher Beef Franks	42	6	12	2	440	N/A
Beef Singles	45	5	13	2	500	370
Fat Free Franks, beef & pork	50	6	0	6	490	520
Beef Fat Free Franks	50	7	0	7	490	530
Lite Franks	50	6	7	3	540	410
Beef Lite	50	6	7	3	510	440
Hebrew National	N/A	N/A	N/A	N/A	N/A	N/A
Reduced Fat Beef Franks	49	6	10	0	360	N/A
97% Fat Free Beef Franks	49	6	1.5	3	400	N/A
Beef Franks	49	6	14	1	370	N/A
Healthy Choice	N/A	N/A	N/A	N/A	N/A	N/A
Meat Franks	50	6	2.5	6	440	N/A
Beef Franks	50	6	2.5	7	440	N/A
Sabrett	N/A	N/A	N/A	N/A	N/A	N/A
Skinless Beef Frankfurters	57	7	15	1	530	N/A

Debbie Alexandrowicz, MA, RD, CSR, shared a hand-out, Thirst Quencher Guide for Hemodialysis Patients. This highlighted cold beverages as being better choices or not recommended based on phosphorus, potassium, sodium, and sugar content. Each area of the country has beverages that may be more commonly used, so you may want to do a similar one for your patients. Reformulation of some beverage recipes has increased the amounts of potassium and/or phosphorus. Although we generically advise patients regarding sodas or other drinks, specifying brand names may be more helpful to patients in making soda and soft drink selections from supermarket shelves.

I found information on the Pepsi web site for potassium content of the following items, all per 8 oz serving: Fruitworks: Apple Raspberry, 25 mg; Peach Papaya, 40 mg; Pink Lemonade, 55 mg; Strawberry Melon, 70 mg; and Tangerine Citrus, 25 mg. Although Mountain Dew regular doesn't have potassium, the diet version has 45 mg. Slice: Regular Orange, 70 mg; Diet Orange, 60 mg; Fruit Punch, 28 mg; and Strawberry, 27 mg. Remember, most soft drink cans are 12 ounces which would be 1.5 servings.

Try these quick flavor tricks for beverages. Add a small amount of non-dairy creamer to cream soda for a mock ice cream soda. Combine ginger ale with cranberry juice cocktail products. Experiment with cranberry or raspberry flavored ginger ales as well as cranraspberry or cran-grape drinks. Persons with diabetes could use fat free or low sugar versions.

Check out these informative web sites on produce, courtesy of Y. Jeffries, MS,RD,LD: http://www.specialtyproduce.com/,http://www.nre.vic.gov.au/trade/asiaveg/thes-00.htm

Homocysteine: The Newest Uremic Toxin?

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levels are a well-known risk factor in hyperhomocysteinemia (8). This rise in homocysteine has also been evident after dialysis has been initiated (6), reflecting progressive decline in renal function.

In the dialysis population, elevated homocysteine levels are commonplace. One study documented that 91% of persons on hemodialysis and 67% of those on peritoneal dialysis experienced an elevated homocysteine level (6). The means were significantly different from each other, however. Mean homocysteine levels in those on hemodialysis averaged 29.8 mmol/L, compared with 19.9 for peritoneal dialysis. These differences were attributed to the significantly lower plasma folate levels observed in persons on hemodialysis.

Homocysteine and ESRD Mortality

Are cardiovascular risk factors affected in ESRD as they are in the general population? This topic was examined by Mallamaci et al who followed 175 persons on hemodialysis for an average of 29 months, and monitored cardiovascular outcomes. Their results showed a clear association between atherothrombotic events, such as thrombotic stroke, myocardial infarction, mesenteric infarction, and pulmonary embolism, and homocysteine. Even more significant was the finding that homocysteine levels independently predicted cardiovascular mortality. The risk of fatal and non-fatal atherothrombotic events was 8.2 times higher in persons in the third homocysteine tertile compared to the first tertile (16). With the small sample size, however, further research is needed to corroborate the potential benefits on cardiovascular outcomes in patients with ESRD.

Quality Control Project

A review of the literature on homocysteine prompted the decision to begin checking homocysteine levels in our out-patient dialysis unit. A quality improvement project was undertaken to analyze demographics and risk factors in our population, plus the effectiveness of a renal multi-vitamin designed to treat hyperhomocysteinemia, which contains 5 mg of folic acid, 1 mg of cobalamin, 50 mg of vitamin B6, plus standard amounts of thiamine, riboflavin, niacin, pantothenic acid, biotin, and vitamin C. Pan American Laboratories, LLC provided all the vitamins used during the 6 month study period. No grant or research funds were requested or provided. Laboratory evaluation of homocysteine was covered by the Centers for Medicare and Medicaid Services.

Methods

Sixty-five persons on dialysis consented to use of the study vitamin, while 24 remained on standard renal multivitamin therapy. Homocysteine levels were measured quarterly. Mean homocysteine values at baseline were not statistically different, with 26.2 mmol/L for the intervention group, and 27.8 for the control group. See Table 2 for demographics.

Results and Discussion

After 3 months, the intervention group's mean homocysteine values dropped 16% below the control group, and after 6 months, the drop was 25%. Both of these were statistically significant, using Pearson Correlation Coefficients ($p < .01$).

Age was not related to homocysteine levels at baseline or after 3 months. No differences were seen between males and females and their homocysteine levels at baseline, but males were significantly higher after 3 months using MANOVA ($p \leq .05$). The cause of renal failure or primary diagnosis did not predict homocysteine levels. This may be because non-functioning kidney tissue, regardless of the cause, results in elevation in homocysteine levels.

A trend was observed between baseline homocysteine levels and the duration of dialysis. When persons new to dialysis (less than 1 year) were compared to those on dialysis for 3-8 years, or more than 8 years, significant differences were noted using Post Hoc comparisons ($p \leq .03$). This is probably related to the decline in kidney function commonly seen as the disease process continues over time, even after dialysis is initiated. Persons on hemodialysis had significantly higher homocysteine values than those on peritoneal dialysis at baseline using ANOVA ($p \leq .013$), but not after 3 months of therapy with the study vitamin.

It is well known that the correction of anemia is highly correlated with quality of life and fewer medical complications. Our results demonstrated that, at baseline, the more anemic patients had higher homocysteine levels ($p < .03$), and higher requirements of epoetin alfa ($p \leq .001$), both of which were statistically significant using Pearson Correlation. It is postulated that, with similar vitamin requirements for red blood cell production and homocysteine control, a patient's vitamin B6, B12, and folic acid status is relevant to the management of both anemia and elevated homocysteine.

The inverse relationship between homocysteine and hemoglobin levels continued to be clinically significant by the sixth month, although statistical differences were not shown. Epoetin alfa dosing declined 15% in the intervention group over the 6 months, with a decline of 19% compared to the control group. The potential for cost savings in dialysis-related medications is obvious.

The differences in homocysteine levels with regard to race were demonstrated. Blacks had significantly higher homocysteine levels at 3 months compared to whites and Hispanics ($p < .0005$). Their values at 6 months showed an overall decline when compared to their baseline values. However, this decline was noticeably lower than the decline in the white and the Hispanic groups, approaching significance ($p < .07$), both based on the ANOVA Procedure Duncan's Multiple Range Test. Blacks may need closer, earlier monitoring of their homocysteine levels both in the pre-dialysis period and while on dialysis.

Summary

An elevated homocysteine level is an independent risk factor for CVD in the general population and in ESRD, and for Alzheimer's and other forms of dementia. Folic acid, vitamin B6, and vitamin B12 status is compromised in the dialysis population, based on current standards of practice.

Our data demonstrated that a renal mul-

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Homocysteine: The Newest Uremic Toxin?

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tivitamin with 5 mg of folic acid, 1 mg of cobalamin, 50 mg of vitamin B6, plus standard amounts of thiamine, riboflavin, niacin, pantothenic acid, biotin, and vitamin C lowered homocysteine levels compared to standard renal vitamin therapy in a group of 89 out-patient chronic dialysis patients.

Continuing declines in homocysteine were seen during the 6 month period of study in the intervention group. These drops in homocysteine levels produced a corresponding drop in epoetin alfa requirements and suggest that folic acid is a potential ally in the management of anemia. Compliance with vitamin therapy can be monitored by regularly measuring homocysteine levels.

Males tend to have higher homocysteine levels than females, and persons on hemodialysis have higher levels on standard vitamin therapy compared to those on peritoneal dialysis. Blacks have higher homocysteine levels, compared to whites and Hispanics, and are more resistant to therapy. Higher homocysteine levels were observed as the duration of dialysis progressed for patients prior to intervention.

Recommendations

Monitoring homocysteine levels is indicated in chronic kidney disease, both before dialysis begins and afterward. Standard vitamin therapy needs to be re-evaluated for most persons on dialysis. Black patients need closer, earlier monitoring of homocysteine and appropriate vitamin therapy. Efforts to lower homocysteine levels could lead to potential cost savings for epoetin alfa use.

Further research is needed to determine if a reduction in homocysteine levels has a beneficial effect on cardiovascular outcomes in patients with ESRD.

Table 2. Demographic Characteristics of Study Participants (n=89)

Demographic Characteristics		N	%	
Gender	Male	46	52	
	Female	43	48	
Mean Age	60			
Race	Black	13	15	
	Hispanic	40	45	
	White	36	40	
Control Group		24	27	
Treatment Group		65	73	

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Kidney Friendly Food Facts

By Sharon Schatz, MS, RD, CSR, CDE
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I'm continually amazed by the fount of informational newsletters that are available by e-mail. FoodNavigator.com originates from Europe and provides updates on the food industry. This is not always directly applicable to the U.S. market, but it increases awareness of food developments that may not have been publicized elsewhere. You might want to check this site.

In one of the recent FoodNavigator mailings, I learned that McCormick is forecasting lemongrass, turmeric, and mustard as some of the "hot" flavors for 2003. Additional items to look for are bay leaf, chili peppers, cinnamon, coriander, vanilla, and even pepper. "Americans are on a quest to experience bolder, more exciting flavor combinations at restaurants and at home," said Laurie Harrsen, director of public relations at McCormick. "Our research shows that these essential flavors are influencing the foods we eat – whether spicy, sweet, worldly, or comforting – each and every day." According to the report, American taste buds in 2003 will call for plenty of flavor, with new pairings, continued use of ingredients that heat and cool the palate, and a growing interest in authentic, regional ethnic cuisines. I'm unsure whether these trends are an effort to merchandise products or if they respond to what is wanted. Either way it may influence our patients' choices.

Mustard seems more likely than lemongrass or turmeric to be used. I like trying different prepared mustards and use them in cooking, but I never gave much thought to their origins. Use of prepared mustard dates back to ancient Egyptian times. Although mustard is prepared from the herb seed, it is generally considered a spice. Mustard seeds can be black, white (yellow), or brown. The seeds themselves do not have an odor, but when ground they release an acrid, earthy aroma. Mustard powder, also known as mustard flour, is obtained when the whole mustard seeds are crushed, ground, and sifted. The pungent taste is determined by myrosinase, an enzyme that is activated by water. Preparations with the sharpest, hottest taste use only water for the liquid; but this is the most unstable form, as it doesn't stop the enzymatic activity. The acidic liquid that is used provides most of the resulting flavor. Vinegar gives a mild tang. Wine adds a spicier pungency. Beer yields a real heat. Do not add acid by itself or hot water as these will kill the enzyme and produce a more bitter flavor.

Prepared mustards can be smooth or whole grained and are often identified by country of origin. English mustard utilizes white seeds, is often hot or sharp, and goes well with bland foods. French mustard is milder and more delicately flavored; and usually we think of Dijon, a blend of husked black seeds, wine, and salt. Provençal mustard is influenced by garlic, red pepper, and wine. American ball-park style is made from white seeds, sugar, and vinegar with a runnier consistency. It is probably the mildest in flavor with lots of turmeric responsible for the bright yellow color. German mustards from black seeds and vinegar are often dark and bold and need foods with strong flavors to combat the robustness. Dutch mustards are strong and sour. Swedish mustard is tangy. Common herbal flavors include horseradish, marjoram, tarragon, and thyme. Sweet mustards contain additional honey or brown sugar and are good for glazing chicken or pork. Wasabi blends have additional potency from Japanese horseradish.

Commercial mustards are mostly used as a condiment but can be added to cold dressings and sauces. A simple sauce can be made by stirring prepared mustard such as Dijon or stone ground into sour cream to compliment prime rib or into mayonnaise for sandwich spread. I mix it with white wine to form a paste that I brush onto fish prior to baking. As the fish cooks a crust layer forms from the mustard and keeps the fish moist while adding flavor. Mustards should be added towards the end of cooking with heated sauces. They can also be mixed with bread crumbs to make a coating for chicken and lamb.

Sodium content of mustard can vary depending upon its ingredients. Salt free or low sodium products are available but may not be readily available in every market. A sampling of mustard blends showed a range of 12 to 120 milligrams per 1 teaspoon (ESHA Food Processor and jar labels). Honey mustard and cranberry mustard had the lowest amount. As a rule of thumb I would estimate 60 milligrams per teaspoon. The taste value it adds seems worth this amount of sodium.

Supermarket shelves have a multitude of products with different tastes and consistencies. Or you can expand a plain mustard's flavor at home by the following: Lemon mustard (good on chicken) – stir in 1 teaspoon grated lemon rind and 1 tablespoon fresh lemon juice into ½ cup Dijon mustard. You could also use lime. Green peppercorn mustard (use on beef) – mix 1 tablespoon freshly ground green peppercorns and 1 minced shallot into ½ cup Dijon mustard Horseradish mustard dip – combine 1 tablespoon grated horseradish root, a dash of Tabasco, and 2 tablespoons nonfat mayonnaise into ½ cup Dijon mustard.

You can easily fix these from mustard powder:
Honey mustard - mix 4 tablespoons powdered mustard with 2 tablespoons cold water and 1 teaspoon vinegar to form a stiff paste. Stir in 1 tablespoon vegetable oil until the mixture is smooth, and then add 2 tablespoons honey until combined. Honey mustard sauce – mix 4 teaspoons mustard powder with 1

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