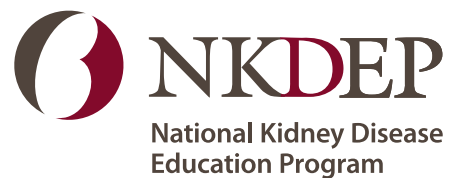# Chronic Kidney Disease (CKD) and Diet:
Assessment, Management, and Treatment

Treating CKD Patients Who Are Not on Dialysis

An Overview Guide for Dietitians

Revised September 2011

NKDEP
National Kidney Disease
Education Program

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This document, developed by the National Kidney Disease Education Program (NKDEP), is intended to help registered dietitians (RDs) provide effective medical nutrition therapy (MNT) to CKD patients who are not on dialysis.

I. About CKD

The kidneys regulate the composition and volume of blood, remove metabolic wastes in the urine, and help control the acid/base balance in the body. They activate vitamin D needed for calcium absorption and produce erythropoietin needed for red-blood-cell synthesis.

CKD is typically a progressive disease. It is defined as:

- Reduction of kidney function—defined as an estimated glomerular filtration rate (eGFR) < 60 mL/min/1.73 m2 and/or
- Evidence of kidney damage, including persistent albuminuria—defined as > 30 mg of urine albumin per gram of urine creatinine

Kidney failure is typically defined as an eGFR < 15 mL/min/1.73 m2.

CKD is detected and monitored by two tests:

- Estimated glomerular filtration rate (eGFR) and
- Urine albumin-to-creatinine ratio (UACR)

The purpose of diet therapy for CKD is to maintain good nutritional status, slow progression, and to treat complications.

The key diet components to slowing progression of CKD are:

- Controlling blood pressure by reducing sodium intake
- Reducing protein intake, if excessive
- Managing diabetes

CKD RISK FACTORS

Diabetes
Hypertension
Family history of kidney failure
Cardiovascular disease
Recurrent urinary tract infections
HIV infection
Immunological diseases

As eGFR declines, complications occur more commonly and are more severe. These may include:

- Malnutrition
- Metabolic acidosis due to reduced acid (hydrogen ion) excretion
- Hyperkalemia
- Mineral imbalance and bone disorder (calcium, phosphorus, and vitamin D)
- Anemia due to impaired erythropoiesis and low iron stores
- Cardiovascular disease (CVD) (dyslipidemia)

II. Assess Kidney Function and Damage

Test and Its Relevance	Results	Dietary Intervention
Estimated Glomerular Filtration Rate (eGFR) eGFR estimates kidney function. As eGFR declines, complications are more likely and more severe.	eGFR (mL/min/1.73m2) Not diagnostic of CKD ≥ 60 CKD 15–59 Kidney failure < 15	• Evaluate eGFR to assess kidney function; track over time to monitor effectiveness of diet therapy. • Stable eGFR may indicate therapy is working. • Decline of eGFR reflects progression of CKD. **Additional Information** Each filtering unit of the kidney, or nephron, filters a tiny amount of plasma each minute. eGFR reflects the total filtration of all two million nephrons. As nephrons are damaged or destroyed, eGFR declines. The quantity or volume of urine may not change significantly as eGFR declines. However, what is excreted into the urine does change. Rapidly declining eGFR may warrant appropriate discussion of renal replacement therapies. In adults, the best equation for estimating eGFR from serum creatinine is the Modification of Diet in Renal Disease (MDRD) Study equation (Levey at al, 1999). NKDEP offers calculators online and as downloadable applications for estimating GFR. Serum creatinine level, age, gender, and race are needed. Many laboratories routinely report eGFR with all serum creatinine determinations.
Urine Albumin-to-Creatinine Ratio (UACR) UACR is the preferred measure for screening, assessing, and monitoring kidney damage. UACR estimates 24-hour urine albumin excretion. Unlike a dipstick test for urine albumin, UACR is unaffected by variation in urine concentration.	UACR (mg/g) Normal 0–30 Albuminuria > 30	• Evaluate UACR over time to assess response to therapy and monitor progression of CKD. • Change in albuminuria may reflect response to therapy and risk for progression. • A decrease in urine albumin may be associated with improved renal and cardiovascular outcomes. **Additional Information** Each filtering unit of the kidney, or nephron, filters a tiny amount of plasma each minute. eGFR reflects the total filtration of all two million nephrons. As nephrons are damaged or destroyed, eGFR declines. The quantity or volume of urine may not change significantly as eGFR declines. However, what is excreted into the urine does change. Rapidly declining eGFR may warrant appropriate discussion of renal replacement therapies.

III. Slow Progression

Therapeutic Goal and Its Relevance	Ranges/Goals	Dietary Intervention
Control Blood Pressure Blood pressure control slows progression of CKD and lowers CVD risk. Sodium plays a large role in blood pressure control in CKD as a result of alterations in sodium excretion by the kidneys.	**Individualize**	• Limit sodium intake to 1,500 mg a day or less (USDA & US DHHS, 2010). • Weight reduction may be beneficial. • Monitor serum potassium in patients on renin angiotensin aldosterone system (RAAS) antagonists; limit dietary potassium intake when serum potassium > 5 mEq/L. **Additional Information** For patients with hypertension, reduction of dietary sodium has been associated with improved blood pressure control in clinical trials and epidemiological studies. Multiple medications may be required to control blood pressure. RAAS antagonists, such as angiotensin-converting enzyme inhibitors (ACEi) or angiotensin receptor blockers (ARBs), are often used to control blood pressure, delay progression, reduce albuminuria, and protect against heart disease. These medications increase the risk of hyperkalemia. Diuretics are prescribed to treat fluid overload and high blood pressure, and may help control serum potassium levels.
Reduce Albuminuria Decreased albuminuria is associated with slower progression of CKD, particularly in diabetics. Limiting dietary protein may reduce albuminuria and improve blood glucose control, hyperlipidemia, blood pressure, renal bone disease, and metabolic acidosis. (de Zeeuw et al, 2004).	Reduce or stabilize the amount of albumin lost in the urine (see UACR on page 2).	Limit excessive dietary protein as follows: • Nondiabetic: 0.8 g protein/kg/day. • Diabetic: 0.8-1.0 g protein/kg/day. Evidence suggests that further lowering to 0.6 g protein/kg/day in nondiabetic patients may be beneficial, but adherence is difficult. Some patients may be able to achieve this level with intensive counseling. **Additional Information** Limiting excessive protein may activate adaptive responses that decrease albuminuria and increase serum albumin, without increasing risk for protein malnutrition. Reducing excessive protein also decreases nitrogen, phosphorus, and potassium intake.

Therapeutic Goal and Its Relevance	Ranges/Goals	Ranges/Goals
Manage Diabetes Blood glucose control may help slow progression of CKD (DCCT,1993; UKPDS,1998) Good control of newly diagnosed diabetes may slow progression of CKD. Tight control in diabetes of long duration may not be indicated.	Individualize A1C/eAG goal eAG = estimated average glucose	• Consider less-stringent control for patients with histories of hypoglycemia, the elderly, and patients with multiple co-morbid conditions. • Instruct patients to treat hypoglycemia with cranberry juice cocktail, grape or apple juice, glucose tablets, or 10 jelly beans to prevent hyperkalemia. **Additional Information** As eGFR declines, renal metabolism of insulin and certain oral diabetes medications is reduced, potentially causing hypoglycemia in diabetes (Snyder and Berns, 2004). Unexplained improvement in glucose control may reflect progression of CKD. Low-protein diets have been associated with improved insulin sensitivity and fasting serum insulin levels, lower insulin requirements and blood glucose levels, and a decrease in endogenous glucose production in patients with diabetes.

IV. Prevent, Monitor, and Treat Complications

Data is limited for CKD. Many of the recommendations for CKD are extrapolated from renal replacement therapies literature.

Complication and Its Relevance	Ranges/Goals*	Dietary Intervention
Malnutrition Malnutrition is common in CKD; as eGFR declines, so may appetite. Malnutrition in CKD patients is associated with increased morbidity and mortality.	Albumin > 4.0 g/dL Normal range: 3.4–5.0 g/dL Serum albumin < 4.0 g/dL, prior to initiation of dialysis, may predict morbidity and mortality (Kaysen et al, 2008). Blood urea nitrogen (BUN) < 20 mg/dL	• Manage with adequate calories and nutrients. • A low protein diet (0.6 g/kg) may require supplemental riboflavin and cyanocobalamin. Supplemental pyridoxine may be beneficial due to medication-vitamin interactions. It may be prudent to provide adequate amounts of thiamin and folic acid. High doses of vitamin C are not recommended. Data do not support routine supplementation with niacin and vitamins A, E, and K. In advanced CKD, certain vitamins may accumulate due to reduced excretion (Steiber & Kopple, 2011). **Additional Information** Serum albumin is used to monitor nutritional status. Hypoalbuminemia may result from reduced protein and/or calorie intake, uremia, metabolic acidosis, albuminuria, inflammation, or infection. Although not used to indicate nutritional status, elevated BUN may be associated with aversion to certain high-biological-value protein foods. Appetite may improve in renal failure with adequate renal replacement therapy (i.e., dialysis treatment or kidney transplantation).
Metabolic Acidosis Patients with CKD are at risk for metabolic acidosis as a result of reduced excretion of acid load.	Bicarbonate (CO_2) > 22 mEq/L Normal range: 21–28 mEq/L	• Dietary protein is a source of metabolic acid. Serum bicarbonate levels may increase with dietary protein restriction. (Gennari et al, 2006) • Sodium bicarbonate supplementation may be prescribed to improve nutritional parameters and slow rate of CKD progression (de Brito-Ashurst et al, 2009). Monitor blood pressure closely when this medication is used, as some patients may experience elevated blood pressure associated with increased sodium load. **Additional Information** Metabolic acidosis is thought to result in loss of bone and muscle mass, negative nitrogen balance, increased protein catabolism, and decreased protein synthesis (ibid).

IV. Prevent, Monitor, and Treat Complications *(continued)*

Complication and Its Relevance	Ranges/Goals*	Dietary Intervention
Hyperkalemia Patients with CKD are at risk for hyperkalemia as a result of reduced potassium excretion, intake of high-potassium foods, metabolic acidosis, and medications that inhibit potassium excretion, such as RAAS antagonists for blood pressure control.	Potassium: 3.5–5.0 mEq/L Hyperkalemia is usually not seen until CKD is advanced, but may be seen at higher eGFRs in people with diabetes.	• Counsel patients to restrict dietary potassium when serum level is 5.0 mEq/L or higher. • Caution patients to avoid potassium-containing salt substitutes. • Instruct patients with diabetes to treat hypoglycemia with cranberry juice cocktail, grape or apple juice, glucose tablets, or 10 jelly beans to prevent hyperkalemia. • Counsel patients to adhere to sodium bicarbonate therapy, if prescribed. Correction of acidosis may lower potassiums. **Additional Information** The potassium content of most vegetables can be decreased through a process of leaching. Leaching entails slicing and soaking the vegetable overnight in water, then draining and boiling the vegetable in new water. A recent study, however, shows that white potatoes do not need to be soaked overnight (Bethke & Jansky, 2008). The potassium content of other tuberous root vegetables commonly eaten in the Caribbean and South America has been shown to be reduced somewhat by double-cooking, however, most still remained higher than 200 mg per serving (Burrowes & Ramer, 2006).

IV. Prevent, Monitor, and Treat Complications *(continued)*

Complication and Its Relevance	Ranges*/Goals	Dietary Intervention
CKD Mineral and Bone Mineral Disorder (CKD-MBD) CKD-MBD is renal bone disease that occurs when the kidneys fail to maintain serum calcium and phosphorus levels.	See sections on calcium, phosphorus, parathyroid hormone (PTH), and vitamin D.	Existing guidelines on management of CKD-MBD reflect consensus rather than high-grade evidence. Early intervention may help prevent vascular calcification and secondary hyperparathyroidism. The kidneys maintain calcium and phosphorus levels and activate vitamin D. As kidney function declines, complex interactions occur that affect calcium, phosphorus, vitamin D, and the parathyroid gland. Abnormal levels of PTH (measured as intact or iPTH) may be seen. Mineral and bone disorders may result from these interactions. See the specific sections that follow.
		Additional Information Depending on the type of renal bone disease, calcium, phosphorus, and iPTH may be normal, decreased, or elevated (Martin & Gonzales, 2007). • **Secondary hyperparathyroidism** is associated with high bone turnover, and elevated levels of calcium, phosphorus, iPTH, and alkaline phosphatase. • **Osteomalacia** results in low bone turnover with elevated serum calcium levels and normal-to-decreased serum phosphorus, iPTH, and alkaline phosphatase. • **Adynamic bone disease** results in low bone turnover and may be characterized by normal-to-low iPTH and alkaline phosphatase. Serum calcium and phosphorus may be normal to elevated. • **Mixed bone disease**, as the name implies, has features of both low and high bone turnover.
Calcium Control of calcium and phosphorus levels helps control PTH.	Calcium: 8.5–10.2 mg/dL Maintain within normal range.	• Dietary calcium recommendations for CKD have yet to be established. • Calcium-based phosphate-binding medications can increase total daily intake and elevate calcium. • Supplementation with active vitamin D increases the risk for hypercalcemia. • Use formula to correct calcium with hypoalbuminemia: Corrected calcium (mg/dL) = serum calcium (mg/dL) + 0.8 (4.0 - serum albumin g/dL)

Complication and Its Relevance	Ranges*/Goals	Dietary Intervention
Phosphorus Control of phosphorus and calcium levels helps control PTH.	Phosphorus: 2.7–4.6 mg/dL Maintain within normal range. Serum phosphorus levels may be "normal" until CKD is advanced	• If serum phosphorus is elevated, dietary phosphorus restriction may be indicated. The recommended level of restriction has yet to be determined in CKD. • Dietary protein restriction decreases phosphorus intake. If further restriction is needed, counsel patients to reduce intake of foods with added phosphorus. (Uribarri, 2007). • Counsel patients to read ingredient lists for "phos" to identify foods with phosphate additives, as these additives may be absorbed more efficiently than food sources. • Limiting whole grains may help if further reduction is needed. • Phosphorus binders may be prescribed to lower phosphorus levels. Counsel patients to take binders with meals to help limit absorption of phosphorus from food and beverages.
		Additional Information Calcium acetate and calcium carbonate are common calcium-containing phosphate binders. Calcium citrate is not recommended as a phosphate binder for CKD patients because it may increase aluminum absorption. Other binders, used more often in renal replacement therapy, are typically composed of resins (sevelamer carbonate) and earth metals (lanthanum carbonate).
Parathyroid Hormone (PTH) Secondary hyperparathyroidism (elevated PTH) is associated with the most common cause of bone disease in CKD.	Normal PTH < 65 pg/mL Measured as iPTH PTH varies by level of kidney function and type of bone disease	Dietary phosphorus restriction and use of active vitamin D or its analogs may help control PTH levels in CKD. Calcium supplementation may help as well.
		Additional Information PTH is the hormone that regulates serum calcium levels. Low levels of 1,25(OH)2D, hypocalcemia, and hyperphosphatemia stimulate PTH secretion. Its metabolic actions include mobilizing calcium and phosphorus from bone, increasing intestinal absorption and renal tubular reabsorption of calcium, and decreasing renal tubular reabsorption of phosphorus. PTH enhances conversion of 25(OH)D to 1,25(OH)2D.

IV. Prevent, Monitor, and Treat Complications *(continued)*

Complication and Its Relevance	Ranges*/Goals	Dietary Intervention
Vitamin D The kidneys activate 25(OH)D (calcidiol) to 1,25(OH)2D (calcitriol or active vitamin D). Reduction of kidney function results in decreased production and conversion of calcidiol to calcitriol. There may be corresponding imbalances of calcium, phosphorus, and PTH.	Vitamin D ≥ 20 ng/mL Measured as 25(OH)D Maintain within normal range (IOM, 2011).	• Supplementation may be indicated. Specific requirements in CKD have yet to be determined. • Ergocalciferol (vitamin D2) or cholecalciferol (vitamin D3) may be used in early CKD to replete vitamin D. • Active vitamin D (calcitriol) or its analogs (doxercalciferol, paricalcitol, or alfacalcidol) may be used as eGFR declines (ibid). Monitor for hypercalcemia and/or hyperphosphatemia when using supplements. Active vitamin D increases calcium and phosphorus absorption.
Anemia Anemia may develop early during the course of CKD due to inadequate synthesis of erythropoietin by the kidneys.	Hemoglobin 12–17 g/dL Transferrin saturation (TSAT) > 16% may indicate iron deficiency Ferritin < 12ng/dL may indicate iron depletion (IOM, 2001)	Both iron supplementation and injectable erythropoiesis-stimulating agents (ESAs) have been used to correct anemia. The risks and benefits of these treatments in CKD are not yet defined. (Besarab & Coyne, 2010). **Additional Information** Hemoglobin is used to assess anemia in CKD. Uncomplicated anemia of CKD is usually normocytic and normochromic. TSAT is a measure of iron saturation. Transferrin transports iron absorbed by the intestines. Ferritin levels reflect iron stores. Existing guidelines regarding TSAT and ferritin levels in CKD reflect consensus rather than high grade evidence.

IV. Prevent, Monitor, and Treat Complications *(continued)*

Complication and Its Relevance	Ranges/Goals*	Dietary Intervention
Cardiovascular Disease (CVD) **Patients with CKD are at high risk for developing CVD; the risk increases as eGFR declines.** **CVD is the leading cause of mortality in CKD.**	Total cholesterol < 200 mg/dL LDL cholesterol < 100 mg/dL HDL cholesterol > 40 mg/dL Triglycerides < 150 mg/dL	Decreasing intake of saturated and trans fats (substituting for monounsaturated and poly-unsaturated fats), along with physical activity, can help control hyperlipidemia and reduce inflammation. **Additional Information** Controlling dyslipidemia may reduce the rate of decline in eGFR. To further decrease risk of developing CVD, pharmacological therapy may be necessary (Fried et al, 2001). Nontraditional risk factors for CVD include anemia, albuminuria, and abnormal calcium and phosphorus metabolism (Kendrick & Choncol, 2008).

V. Patient Education Materials

NKDEP offers a suite of materials to support RDs in providing MNT to patients with CKD. These free materials—designed to distill key information about CKD and diet for RDs and patients—are available to download from the NKDEP website at **www.nkdep.nih.gov/ckd_nutrition**.

- **Eating Right for Kidney Health: Tips for People with CKD**—a handout on the basics of nutrition and CKD

- **Nutrition Tips for People with CKD**—individual nutrient handouts on:
  - Protein
  - Phosphorus
  - Potassium
  - Sodium
  - Food-label reading

- **Your Kidney Test Results**—a tool for assessment and education of test results with patients

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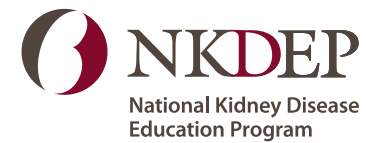
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