

Kidney Disease

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For many years, the Centers for Disease Control (CDC) has listed kidney disease as one of the top 10 causes of death by disease in the United States. Kidney disease also plays a significant role in hypertension and diabetes, two other diseases that are also included on the CDC's list of top ten causes of death each year. End-stage renal (kidney) disease (ESRD) is growing at a rate of 4-8% each year in the United States. Someone with advanced ESRD may require either therapeutic or regular dialysis, or both, and may eventually require a kidney transplant to save his or her life. When kidney function is reduced to 10-15% or less, dialysis is started in ESRD patients. Sometimes ESRD patients are placed on a waiting list for a kidney transplant.

According to statistics compiled by the National Institute of Diabetes & Digestive & Kidney Diseases (NIDDK)(2001), kidney conditions such as inflammation, kidney stones, and cancer affected some 2.553 million persons; ESRD affected 424,179 people; polycystic kidney diseases affected 600,000 people; and other urinary conditions such as kidney infections, bladder infections, and cystitis affected millions more, costing billions of dollars of medical care funded by the public and by private individuals (NCHS 1999; Grantham et al. 2000; USRDS 2001).

Due to the limited scope of this protocol, we will briefly describe some of the more common kidney disorders and treatments. However, two conditions will be described in greater detail: *autosomal dominant polycystic kidney disease* (ADPKD) and *kidney stones*. ADPKD is a common human genetic disease, resulting in many cases of ESRD and eventually the need for kidney transplantation. Kidney stones affect approximately 10% of the U.S. population at some point in their lives (LaPorte et al. 1990). Unfortunately, about 60% of persons who have a kidney stone will develop another stone. In statistics reported by the NIDDK, urinary stones accounted for 1.325 million visits to physicians in 1997 (NIDDK 2001b).

Attention to overall kidney health is essential. If you have healthy kidneys, take care of them. Educate yourself about how to do this. We will provide information in the paragraphs that follow to assist you in being proactive in maintaining healthy kidneys. If you have a health condition such as diabetes or hypertension that poses a threat to your kidneys, seek a qualified medical professional to treat and control these conditions. Then carefully follow monitoring and treatment advice. Information will also be provided to assist you in supporting kidneys that have already sustained damage.

KIDNEY FUNCTION

The kidneys are bean-shaped organs that act as sophisticated filters to remove organic waste products from the blood and then excrete these waste products, along with excess salt and water, from the body through the urine. We are normally born with two kidneys located on either side of the lower back just below the rib cage. The kidneys are such incredibly well-functioning organs that only one normal, healthy kidney is required for good health. Each kidney is 4-5 inches long, weighs about 6 ounces, and contains about 1 million nephrons. Nephrons are the working units of the kidney that are responsible for waste removal (NIDDK 1998). As part of our normal aging process, kidney function diminishes as the number of functional nephrons is reduced.

The kidneys play a role in controlling the acid-base balance in the body as well as helping to control blood pressure. Another function of the kidneys is to produce hormones such as erythropoietin, which regulates the production and release of red blood cells from the bone marrow.

Each day, the kidneys filter approximately 200 quarts of blood, producing about 2 quarts of waste products and water (NIDDK 1998). These waste products and excess water pass from the kidneys through the ureters (tubes that connect the kidneys to the bladder) and into the bladder where they are briefly stored before being eliminated as liquid waste via the urine. Filtered waste products include the normal organic material from the breakdown of cells, proteins, excess food by-products, and various minerals, as well as the individual waste excretions from cells of the body. Alcohol, drugs, excess protein, minerals, and ingested toxins are also filtered by the kidneys. These toxic agents can have a dramatic, destructive effect on the health and function of the kidneys.

The rate of blood flow through the kidneys is about 20% of the total blood pumped by the heart each minute. (Anatomical Chart Company 2002®, Lippincott Williams & Wilkins)

Kidney function is often measured by using routine blood and urine tests to indicate gross problems. These tests measure creatine levels, possible blood in the urine, blood urea nitrogen (BUN), proteinuria (protein in the urine), and mineral content, including calcium, magnesium, phosphorus, sodium, potassium, oxalic acid, and other elements. If blood or urine tests indicate improper kidney function, additional testing is indicated using conventional x-rays, needle biopsy, ultrasound, a computed tomography scan (CT scan), or magnetic resonance imaging (MRI) (NORD 2002).

KIDNEY DISORDERS

- Kidney Stones
- Medical Intervention
- Prevention

Kidney disease is any disorder that affects how the kidneys function. A list of all of the diseases and conditions that can affect kidney function and the possible causes are beyond the scope of this protocol. However, some of these disorders include analgesic nephropathy, chronic nephritis, diabetes, ESRD, hypertension, infection, injury, stones, lupus erythematosus, and ADPKD (NORD 2002).

Symptoms of renal disease can include frequent headaches and urination, itching, poor appetite, fatigue, burning bladder, anemia, baggy eyes, nausea and vomiting, swollen or numb hands or feet, poor concentration, darkened skin, and muscle cramps (NORD 2002).

Kidney Stones (Calculi)

Kidney stones (or calculi) are a common condition and also an incredibly painful one. It is estimated that in the United States, 10% of us will pass a kidney stone at some time in our lives. Men have more kidney stones than women, and white people are more prone to kidney stone formation than black people. The incidence of kidney stones is higher in the summer. This may be because we perspire more in the summer and our urine becomes more concentrated.

A kidney stone is a solid, rock-like type of material that has formed or is present in the kidneys, ureters, or bladder. A kidney stone is formed from mineral substances that precipitate from the urine. Kidney stones can stay in the kidney or travel down the urinary tract. Small stones are sometimes passed from the body with either a small or large degree of pain. Larger stones may lodge in the ureter, bladder, or urethra, blocking urine flow and causing extreme pain (NIDDK 1998).

Most kidney stones contain calcium combined with either oxalate or phosphate. Calcium stones are formed when extra calcium is not eliminated in the urine. Another type of kidney stone is a *struvite stone*. A struvite stone can form following a urinary infection. *Uric acid stones* form when there is too much acid in the urine. A rare type of kidney stone is made up of cystine. Evidence shows that cystine-based stones tend to run in families (the result of a genetic disease) (NIDDK 1998).

Kidney stones vary widely in size: from a grain of sand, to the size of a pearl, or to the size of a golf ball. However, most kidney stones are quite small. Kidney stones can grow to a size that is life threatening or that requires surgical removal. Some large kidney stones cannot be surgically removed because of the age of the patient or because of the danger of associated trauma to a vital organ.

Kidney stones are usually yellow or brown in color. Their structure and texture can be smooth or jagged. Another common visual characteristic is a crystalline appearance with different mineral striations appearing throughout the structure of the stone. Examination and testing of a kidney stone by a specialist in urology can determine significant information about the possible cause of the kidney stone and perhaps suggest a remedy for people who have the potential to form additional kidney stones (NIDDK 1998).

As noted earlier, kidney stones tend to run in families. They can also be associated with geographic factors as well. Therefore, people who live in tropical climates may be at greater risk for kidney stone formation because of the way the body manages water in a tropical setting. As a percentage, perspiration often becomes the prevalent method of how the body excretes water in tropical or very hot conditions, and urination may decline slightly because urine is stored longer in the urinary tract. Although it seems obvious, the fact is that most people do not drink enough water every day, and in tropical areas this is even more significant. Excessive perspiration becomes even more significant when performing hard physical labor or engaging in strenuous sports activities in very hot conditions. The body loses large amounts of water during excessive perspiration. For example, a NFL lineman can lose as much as a gallon of water or as much as 10 lbs of water weight during a 4-hour game. Therefore, sufficient water intake is both a preventive and a therapeutic measure.

The symptoms of a kidney stone attack include sudden extreme pain in the lower back, side, or groin; blood in the urine; fever and chills; vomiting; a bad odor or cloudy appearance to the urine; and a burning sensation during urination. Any of these symptoms require evaluation by a physician. Pain in the lower back, side, or groin can also be indicative that a kidney stone is moving or that there is a serious urinary tract blockage that requires immediate medical intervention. Kidney stone episodes frequently include urinary tract infections (UTIs). Recurrent, untreated UTIs can eventually cause permanent kidney damage and reduced kidney function.

Passing a kidney stone can be as simple as drinking large amounts of liquid and running up and down stairs or jumping up and down vigorously to dislodge the stone! This practice uses the basic physics of gravity to get the stone moving so that it can be passed normally. If you know you are passing a kidney stone, try to catch it in a strainer or retrieve it so it can be examined by a nephrologist or urologist (NIDDK 1998).

Medical Intervention for Kidney Stones

Many kidney stones pass from the body on their own with no medical help. However, more complex procedures are required to assist stones that cannot be passed or to remove stones that are growing larger (NIDDK 1998). Either lithotripsy or surgical removal of the stone is used when a kidney stone is firmly lodged in the ureters, bladder, or urethra. In the past, problem kidney stones represented a significant health concern because the only way to remove them was invasive surgery with a high risk of postoperative infection. It is now possible for urologists to avoid surgery except as a last resort or when there is no other alternative. Newer methods to remove kidney stones include using ureteroscopy, tunnel surgery, extracorporeal shock wave lithotripsy (ESWL), and percutaneous lithotripsy. All of these methods break the stone into smaller pieces so that the stone can be removed or passed through the urinary tract (NIDDK 1998).

Preventing Kidney Stones

Research into the prevention of recurrent kidney stones has produced many helpful dietary guidelines, nutritional protocols, and lifestyle changes that can reduce or eliminate the potential for future kidney stones. Using these effective protocols can significantly reduce the chance of recurring kidney stones after a first episode. They may also help pass a recurrent stone faster and with less difficulty.

In 1997, a research division of a healthcare provider conducted a double-blind study with a group of 64 patients who had a history of renal calculi to determine if potassium/magnesium citrate would prevent the recurrent formation of calcium oxalate kidney stones (Ettinger et al. 1997). The patients were given 42 mEq (milliequivalent) potassium, 21 mEq magnesium, and 63 mEq citrate or a placebo daily for 3 years. New renal calculi formed in 63.6% of patients receiving the placebo. However, patients receiving the potassium/magnesium citrate protocol presented with 12.9% recurrent renal calculi. Ettinger et al. (1997) concluded that "potassium/magnesium citrate effectively prevents recurrent calcium oxalate stones, and this treatment given for up to 3 years reduces risk of recurrence by 85%."

Contrary to what was considered to be "common sense" thinking in the past, two major studies have shown that calcium should not be reduced for patients with a history of kidney stones (Takei 1998; Williams 2001). It was originally postulated that patients with a history of renal calculi should limit their intake of calcium. In fact, current recommendations from the National Institutes of Health published on their Web site continue to call for calcium-restricted diets. Such dietary changes also affect the alkali and pH of the body by calling for the restriction of foods such as apples, beets, parsley, broccoli, spinach, and pineapples. However, newer findings contradict these dietary restrictions and offer scientific evidence that uncombined intestinal oxalic acid is the real culprit for calcium oxalate kidney stones (Ohgitani 2000).

Harvard researchers studied nearly 92,000 nurses over a period of 12 years to determine the relationship between calcium intake and the occurrence of renal calculi (the well-known Harvard Nurses' Health Study). The conclusion of this massive study was that those nurses who consumed diets that were higher in calcium were at lower risk for kidney stones!

The reason that this type of dietary modification reduced the chance of kidney stones was relatively simple. A high percentage of kidney stones are comprised of calcium and oxalic acid which form calcium oxalate *inside the kidneys*. Oxalic acid is able to pass through the intestinal wall into the blood and enter the kidneys where it has a chance to combine with calcium. Calcium oxalate, when normally combined *inside the digestive tract*, does not pass through the intestinal wall and into the blood, but is eliminated with other waste products. Therefore, when oxalic acid combines with dietary calcium or supplemental calcium inside the intestinal tract, oxalic acid will never reach the kidneys and therefore calcium oxalate kidney stones cannot be formed.

The *Harvard Nurses' Health Study* presented the following important findings: dietary calcium intake from food or supplements reduced the risk for renal calculi; calcium supplementation must be taken with food and in small dosages (< 400 mg); plant foods high in calcium, fiber, vitamins, minerals, antioxidants, and some protein were an excellent source for dietary phytochemicals.

Another study conducted in South Africa found that "mineral water containing calcium and magnesium deserves to be considered

as a possible therapeutic or prophylactic agent in calcium oxalate kidney stone disease" (Rodgers 1997). A French mineral water containing calcium (202 ppm) and magnesium (36 ppm) was selected as the delivery method. Twenty subjects of each sex who had previously formed calcium oxalate renal calculi and 20 healthy volunteers of each sex participated in the study. Each subject provided 24-hour urine collection samples each day during the study. The mineral water was ingested over a 3-day period. Then the participants switched to tap water. The cycle was repeated at least twice by each subject. The male stone formers received the most benefit, showing nine risk factors that were favorably affected by the mineral water containing calcium and magnesium (Rogers 1997).

Recommendations from the National Kidney and Urologic Diseases Information Clearinghouse (1998) include a few simple things to do to avoid kidney stones:

- Drink more water. Try to drink at least 12 full glasses of water each day. Drinking extra water helps to flush substances that form stones from the kidneys.
- It is not necessary to eliminate coffee, tea, and colas from your diet, but limit caffeine because it can increase fluid loss. Consider drinking ginger ale, lemon-lime soda, and fruit juices.
- Follow your physician's recommendations about dietary limitations. If you form uric acid stones, your physician will probably ask you to eat less meat because meat breaks down to form uric acid.
- Follow your physician's recommendations about taking medicines to prevent stone formation.

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Kidney Disease

FACTORS AFFECTING KIDNEY FUNCTION

- Analgesics
- Autoimmune
- Congenital and Genetic
- Drug Reactions
- Homocysteine
- Hypertension
- Impaired Blood Supply
- Infection
- Inflammatory Cytokines
- Metabolic
- Tumors
- Other

Adults lose renal function and capacity with normal aging. A number of factors, including drug reactions and degenerative disease not endemic to the kidneys, may bring added stress.

Analgesics

An analgesic is any medicine that is intended to kill pain. Analgesics that contain narcotics are for more severe pain and require a prescription from a physician. However, many analgesics can be purchased as over-the-counter (OTC) products (aspirin, ibuprofen, acetaminophen, and naproxen). OTC products require no prescription from a physician. OTC analgesics rarely present a problem for most people if they are taken according to the recommended dosage. However, some conditions such as chronic kidney disease or taking OTC analgesics for a long time or in combination with other analgesics make OTC analgesics dangerous. According to the NIDDK (1998), analgesics such as aspirin, ibuprofen, acetaminophen, and naproxen have been attributed to incidence of acute kidney failure in persons with lupus erythematosus or chronic renal conditions; persons of advanced age; or persons who have had a recent binge of alcohol consumption.

Some cases involved a single dose or no more than 10 days of analgesic use. Painkillers that combine two or more analgesics (e.g., aspirin and acetaminophen together) with caffeine or codeine are more likely to cause kidney damage. These mixtures are often sold in powder form. Single analgesics (e.g., aspirin alone) have been found to be less likely to cause kidney damage (NIDDK 1998). According to Fored et al. (2001), more research is required to determine whether the use of aspirin or acetaminophen contributes to kidney failure or whether people who have ailments that predispose them to kidney failure are more likely to use painkillers. Fored et al. (2001) recommended that each patient be considered individually with respect to their risk of kidney failure, length of time the painkiller will be taken, and other existing illnesses, particularly in the elderly and persons with chronic conditions. If possible, avoid acetaminophen-based analgesics, as these may be most toxic to the kidneys.

Autoimmune

Glomeruli, the tiny blood vessels in the nephrons where blood is filtered in the kidneys, can become inflamed by autoimmune disorders. When an autoimmune disorder occurs, the body attacks itself with its own immune system. Examples of an autoimmune disorder are Goodpasture syndrome and lupus erythematosus. In the kidneys, this type of inflammation is called glomerulonephritis (Glanze 1996; NIDDK 1999). While glomerulonephritis is usually caused by an autoimmune disorder, it can also be caused by infection (e.g., by streptococcal bacterial).

Congenital and Genetic

Congenital abnormalities of the kidneys are not uncommon. Sometimes the two kidneys are joined together at their base. Some people are born with only one kidney, both kidneys on the same side of the body, or with underdeveloped kidneys that are barely functional. Polycystic kidney disease is a genetic condition that may manifest at birth, but often appears in young adulthood or even middle age.

Drug Reactions

Acute kidney damage can result from an allergic reaction to a drug; taking large quantities of a drug for a long period of time; taking out-dated tetracyclines; taking long-term or large amounts of pain killers; taking potent antibiotics; accidental ingestion of poisons;

toluene inhalation (e.g., industrial exposure and glue sniffing); or combining prescription drugs, over-the-counter drugs (aspirin, acetaminophen, ibuprofen, naproxen sodium), and alcohol (NIDDK 1998). Regular blood tests to assess kidney function are recommended for anyone who takes medicine known to damage the kidneys or who has a condition that puts them at risk for developing kidney disease.

Homocysteine

Discovered in 1932, homocysteine is a sulfur-containing amino acid normally found in small amounts in the blood of healthy persons. Homocysteine is derived from dietary protein (meat, milk, eggs) and is metabolized in the liver using vitamins B6 and B12. High levels of homocysteine can result from genetic disease (homocystinuria); kidney disease; hyperthyroidism; psoriasis; systemic lupus erythematosus; drug treatment for chronic diseases; and dietary vitamin deficiencies (folic acid, B6, B12) (Welch et al. 1998).

Homocysteine levels tend to increase with age and are higher in men than in women. High levels of homocysteine can be very damaging to the kidneys and the vascular system (Dierkes et al. 1999; Marangon et al. 1999; Levin et al. 2002). Accumulation of toxic homocysteine has been associated with the development of cardiovascular disease (atherosclerosis, stroke, heart attack); pulmonary embolism and deep venous thrombosis; dementias (Alzheimer's disease, multi-infarct dementia); and kidney disease ESRD (Joosten et al. 1997; McCaddon et al. 1998; Welch et al. 1998; Dierkes et al. 1999; Levin et al. 2002; Seshadri et al. 2002). Cardiovascular disease (CVD) is common in patients with chronic kidney disease (CKD) and is responsible for the majority of morbidity and mortality in patients (Levin et al. 2002).

As early as 1969, researchers began to make clinical observations linking elevated homocysteine to vascular diseases (McCully 1969). Subsequent investigations confirmed these observations (Clarke et al. 1991; Ueland et al. 1992; Stampfer et al. 1992; 1995; Selhub et al. 1995; Welch et al. 1998). In CVD, there is evidence that elevated levels of homocysteine are related to arterial wall damage, but the mechanism is unclear (Welch et al. 1998). It may be that homocysteine has a toxic effect on the endothelial (cellular) lining of blood vessels. Data from a study on healthy U.S. physicians (14,916) with no prior history of heart disease demonstrated that highly elevated homocysteine levels are associated with a more than threefold increase in the risk of heart attack over a 5-year period. This finding was published in 1992 in the *Journal of the American Medical Association (JAMA)* as part of the Physicians' Health Study (Stampfer et al. 1992). The Framingham Heart Study (1041 elderly subjects) (Selhub et al. 1995) and other studies have also confirmed that elevated homocysteine is an independent risk factor for heart disease (Chaveau et al. 1993; van Guldener et al. 2000; Hoffer et al. 2001; Suliman et al. 2001).

In kidney disease, homocysteine levels in the blood increase because the kidneys do not properly filter homocysteine. Elevated levels of homocysteine are commonly seen in renal patients, sometimes three or four times higher than normal levels (van Guldener et al. 2000; Friedman et al. 2001; Herrmann et al. 2001; Suliman et al. 2001). Homocysteine is consistently elevated to very high levels in patients who require dialysis (Levin et al. 2002). Plasma homocysteine concentrations often decrease after dialysis (Welch et al. 1998). Therefore, to further help lower homocysteine levels, dialysis patients often require high levels of nutrients, including folic acid, vitamin B12, TMG (also known as betaine or trimethylglycine), and vitamin B6 (Bostom et al. 1996; Chauveau et al. 1996; Robinson et al. 1996; Sadava et al. 1996; Tucker et al. 1996; Welch et al. 1998; van Guldener et al. 2000; Herrmann et al. 2001; Levin et al. 2002).

Folic acid was used in a study conducted in 82 patients undergoing dialysis 3 times a week for 4 weeks (hemodialysis, 70 patients; peritoneal dialysis, 12 patients) (Dierkes et al. 1999). The results demonstrated that in both groups, homocysteine concentration was reduced by 35% after taking 2.5-5 mg of folic acid after each dialysis treatment.

As noted earlier, although dialysis has the effect of lowering homocysteine levels, folic acid further reduced homocysteine levels and, more importantly, had long-term effects even after supplementation was withdrawn (Dierkes et al. 1999).

Although the relationship between CVD and CKD is convincing, therapeutic strategies appear to be underused in the care of patients with kidney disease. CVD and CKD have similar traditional risk factors (diabetes, hypertension, dyslipidemia, obesity) as well as nontraditional risk factors (hyperhomocysteinemia, anemia, disturbed mineral metabolism, parathyroid excess). Because these risk factors are also specific to kidney disease and are modifiable, they should be identified and treated in persons with CKD (Levin et al. 2002). Patients with mild hyperhomocysteinemia have no clinical signs and are typically asymptomatic until the third or fourth decade of life (Welch et al. 1998).

For some time, physicians have recognized the danger of homocysteine and they recommend use of vitamin supplements to lower homocysteine levels (Tucker et al. 1996; Welch et al. 1998). The "normal range" used by commercial laboratories is 5-15 micromoles/L of blood. However, epidemiological data reveal that homocysteine levels above 6.3 result in a steep, progressive risk of heart attack, with each three-unit increase equaling a 35% increase in risk for heart attack (Verhoef et al. 1996; Robinson et al. 1996). There may be no safe "normal range" for homocysteine. A survey in *Cardiologia* reported that the average American's level of homocysteine is 10 (Andreotti et al. 1999).

For many persons, daily intake of TMG (500 mg), folic acid (800 mcg), vitamin B12 (1000 mcg), vitamin B6 (100 mg), choline (250 mg), inositol (250 mg), and zinc (30 mg) will keep homocysteine levels in a safe range. Unfortunately, without a homocysteine blood test, it is impossible to know if the proper amounts of nutrients are being taken. Therefore, the only way to be certain is to have a blood test to ascertain that your homocysteine level is below 7. Sometimes treatment must be individualized for complicated conditions. High levels of homocysteine can require up to 6 grams of TMG or vitamin B6 (in cystathione-B synthase deficiencies).

Hypertension

High blood pressure (or hypertension) creates a significant risk factor for kidney failure. This risk factor is amplified for persons who have ADPKD. Li Kam Wa et al. (1997) investigated the 24-hour blood pressure profile of ambulatory patients, particularly to measure nocturnal fall of blood pressure. The researchers found that in ADPKD patients, the reduction in nocturnal blood pressure was attenuated (lessened), indicating increased risk for kidney damage. Further studies are needed to evaluate the contribution that nocturnal hypertension makes on the overall progression of renal failure. However, in another related study of untreated children, it was found that nocturnal hypertension was a major risk factor for renal deterioration (Ligens et al. 1997).

Impaired Blood Supply

Any condition that impairs blood flow to the kidneys can damage or cause obstruction in the small blood vessels in the kidneys (e.g., diabetes mellitus, hemolytic uremic syndrome, physiological shock, lupus erythematosus).

Infection

A kidney may become infected when the flow of urine is restricted in the urinary tract (NIDDK 1998). An obstruction may lead to stagnation of urine in the kidney that allows infection to spread into the bladder. Possible causes of an obstruction are a congenital defect, a kidney stone, a bladder tumor, or enlargement of the prostate gland. Tuberculosis of the kidney occurs when infection is carried by the blood to the kidney from somewhere else in the body (usually the lungs).

Inflammatory Cytokines

Destructive cell-signaling chemicals called inflammatory cytokines contribute to degenerative, inflammatory, and autoimmune diseases (Van der Meide et al. 1996; Licinio et al. 1999). Degenerative diseases appear to be factors in or possible underlying causes of kidney failure and disease (congestive heart failure, anemia, rheumatoid arthritis, fibrinogen formation, fibrosis, diabetes, asthma, lupus, psoriasis). People who have multiple degenerative disorders often exhibit excess levels of pro-inflammatory markers in their blood. Therefore, seemingly unrelated inflammatory or autoimmune diseases can have a common link to kidney disease: inflammatory cytokines. In kidney failure, inflammatory cytokines restrict circulation and damage nephrons (the filtering units of the kidneys).

For those who have degenerative diseases, particularly multiple ones, cytokine profile and C-reactive protein blood tests are highly recommended (available through your own physician or Life Extension Foundation). If your cytokine test reveals excess levels of cytokines--tumor necrosis factor-alpha (TNF-alpha), interleukin-1b (IL-1b)--nutritional supplementation, dietary modifications, and low-cost prescription medications (pentoxifylline or PTX) are advised (*see the Inflammation: Chronic protocol for a discussion of systemic inflammation and recommendations for reducing inflammatory conditions*).

Metabolic

Kidney stones are more common in middle age and are usually caused by excessive concentrations of substances such as calcium, uric acid, or cystine in the urine (NIDDK 1998). Hyperparathyroidism, cystinuria, and hyperoxaluria are rare, inherited metabolic disorders that can cause kidney stones. In cystinuria, too much of the amino acid cystine can lead to the formation of stones made of cystine. In patients with hyperoxaluria, the body produces too much of the salt oxalate. Excessive oxalate in the urine cannot be dissolved, crystals settle out, and stones form. Absorptive hypercalciuria occurs when the body absorbs too much calcium from food. The extra calcium ends up in the urine and the high levels cause calcium oxalate or calcium phosphate crystals to form in the kidneys or urinary tract. Other causes are hyperuricosuria (a disorder of uric acid metabolism), gout, excess intake of vitamin D, and blockage of the urinary tract. Certain diuretics ("water" pills) or calcium-based antacids can increase the risk of kidney stone formation by increasing the amount of calcium in the urine (NIDDK 1998).

Tumors

Tumors in the kidneys, either benign or malignant, are rare. When malignant, the most common type is renal cell carcinoma, particularly in adults over 40.

Other

Urinary tract infections (UTIs) are frequently occurring health conditions that are caused by various urinary systemic infections, sexual contact, bacteria entering the kidneys via the bloodstream or the urethra, kidney stone blockages, and kidney damage (Glanze 1996). Infection can lead to impaired kidney function. Therefore, a kidney infection should be treated immediately to prevent more serious disease. A direct blow to the kidneys can also cause extensive damage (e.g., a car accident, industrial accident, sports injury, or accidental fall) (NORD 2002).

AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE (ADPKD)

ADPKD is one of the most common genetic diseases in humans. It is a systemic disease that is caused by at least three different genes: PKD1, PKD2, and PKD3. However, most of the mutations are found in the PKD1 gene (Merta et al. 1997; Sessa et al. 1997). ADPKD is a very serious disease. Worldwide, it is responsible for 8-10% of all cases of ESRD. Patients with ADPKD develop cysts in both kidneys. These cysts continue to grow over the lifetime of the patient and ultimately lead to hypertension, reduced kidney function, and eventually renal failure. Poor kidney function in ADPKD patients accounts for many kidney transplants each year. According to the PKD Foundation (Kansas City, www.pkdcure.org), 60% of individuals with ADPKD develop kidney failure or ESRD. The only treatment is dialysis or transplant. Interestingly, because ADPKD is genetic in origin, persons who receive kidney transplants do not reacquire their genetic mutation with transplanted kidneys. Common symptoms are frequent infections, blood in the urine, and back pain.

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Kidney Disease

Polycystic kidney disease may occur at birth, during childhood, or in adults. Congenital polycystic disease can be detected at birth and may affect all or only small parts of one or both kidneys. Childhood polycystic kidney disease can cause death after a few years because of liver and kidney failure. In some adults, the disease may actually be present at birth, but not manifest any symptoms until young adulthood or middle age. In adults, it can affect either one or both kidneys (Glanze 1996). Polycystic kidney disease is characterized by autonomous cellular proliferation, pockets of fluid accumulation within the cysts, and intraparenchymal fibrosis of the kidney. Other clinical observations include renal failure, liver cysts, and cardiac valve abnormalities (Bacallao et al. 1997).

The traditional method of detecting ADPKD has been by using ultrasound, computed tomography (CT), or magnetic resonance imaging (MRI) of the kidneys to look for the presence of renal cysts. However, the challenge is to detect ADPKD in people who carry the defective gene, but who may not have any symptoms or show any developed cysts and therefore be undiagnosed as having ADPKD. Newer methods of DNA testing can now identify individuals who carry the defective gene, but are not symptomatic. For example, every member of four Chinese families with a known history of ADPKD showed unique DNA patterns (Yuan 1997). DNA diagnostic testing methods have value for patients with existing ADPKD as well as for presymptomatic patients.

ADPKD progresses to end-stage renal insufficiency before the age of 73 in about 50% of affected patients (Grantham 1997). Some patients are affected by numerous cysts that form inside the proximal and distal tubules, while other patients are spared. Why this is the case remains a mystery. The formation of cysts begins in early childhood, affecting less than 1% of tubules as a consequence of mutated DNA. The risk factors associated with polycystic kidney disease include gender (males progress more quickly than females), race (black patients progress more rapidly than whites), and other contributing factors such as hypertension and proteinuria. These factors can aggravate and accelerate polycystic kidney disease through to end term (Grantham 1997).

Because hypertension is a common and serious factor of ADPKD that usually occurs early in the disease before renal function begins to decrease, Doppler ultrasonography has been used to assess renal vascular resistance (RVR) by measuring resistive and pulsatility indices. In a study of 42 patients with ADPKD and 65 control subjects, Brkljacic et al. (1997) found that Doppler indices do reflect increased RVR in those patients with ADPKD and that renal function disturbance did manifest systemic arterial hypertension. The abnormality of the kidneys in these patients was easily observed using ultrasound. However, this method did not show ADPKD potential for patients if renal cysts were not present. DNA testing is required to determine whether a patient carries the PDK1 and PDK2 chromosomes.

The occurrence of cardiovascular complications is a very common cause of death for persons with ADPKD. Chapman et al. (1997) examined the relationship of known cardiovascular risk factors, hypertension, and ADPKD. According to the researchers, left ventricular hypertrophy (LVH) is an important risk factor for premature cardiovascular death in persons with essential hypertension. Hypertension occurs frequently and early in ADPKD patients. In 116 adult ADPKD patients and 77 healthy controls, Chapman et al. (1997) found a higher frequency of LVH in ADPKD men (46% versus 20%) and women (37% versus 12%) compared to the control subjects. LVH in ADPKD patients was associated with higher systolic and diastolic blood pressure. According to the researchers, the role of blood pressure as a contributing factor to LVH in ADPKD patients may be partly due to early onset and inadequate treatment.

The possibility is being explored that ADPKD may have an emerging infectious disease component as well. Research has shown fungal DNA in kidney tissue and cyst fluids of ADPKD patients, but not in healthy kidneys of persons without ADPKD (Miller-Hjelle et al. 1997). In a differential activation protocol assay, the researchers showed bacterial endotoxin and fungal beta-D-glucans in cyst fluids from human kidneys with PKD. Tissue and cyst fluids were examined for fungal components, and the serological tests showed *Fusarium*, *Aspergillus*, and *Candida* antigens. Miller-Hjelle et al. (1977) concluded that "endotoxin and fungal components, sphingolipid biology in PKD, the structure of PKD gene products, infection, and integrity of gut function [will establish a mechanism] for microbial provocation of human cystic disease."

TREATMENT FOR KIDNEY DISEASES

- Conventional
- Natural and Adjuvant

Treatment of kidney disease is a complex issue and depends on the type of disease, the underlying cause, and the duration of the disease. Treatment usually starts with addressing the original cause such as inflammation. Inflammation from infection is treated with antibiotics. Inflammation caused by an immune reaction is more difficult to treat. In this case, immunosuppressant drugs (corticosteroids) are used in an attempt to control the immune reaction.

In the case of acute kidney failure, treating the underlying cause may return the kidneys to normal function. Sometimes dietary

restrictions (less salt and protein) are required until the kidneys are better able to handle these substances. Diuretic medicines help the body to excrete more water and salt. However, with chronic kidney failure, medicines are used to stop progression of the disease so it does not reach ESRD.

When kidney disease does not respond to treatment with dietary restrictions and medicines, dialysis or kidney transplantation are the next treatments to consider (Glanze 1996). Dialysis is a technique used to remove waste products from the blood and excess fluid from the body in the case of renal failure. Kidney transplantation is a surgical procedure in which the diseased kidney (sometimes both kidneys) is removed and replaced with a healthy kidney from a donor (NIDDK 1999).

Conventional Medical Treatment

- Medicine and Drugs
- Kidney Dialysis
- Transplantation

Medicine and Drugs

In the United States, 4% of the population is at risk for kidney disease. As part of an annual physical checkup, we should have three important tests: blood levels of creatinine, blood urea nitrogen, and urine levels of protein. Small elevations of creatinine can be an early sign of kidney disease. According to the National Kidney Foundation (2001a), 11 million Americans have elevated blood levels of creatinine. Healthy kidneys remove creatinine, but when kidney function diminishes, creatinine levels in the blood go up. Early detection leads to early treatment, which can occur at a stage when treatment can help prevent kidney disease from advancing to a more serious stage. Diabetes is the leading cause of chronic kidney disease, followed by hypertension. See your physician regularly and follow prescribed dietary and drug treatment to control blood sugar levels and blood pressure (National Kidney Foundation 2001a). Treatments for conditions which can lead to kidney disease include numerous prescription drugs and treatment protocols. (*See the Life Extension protocols on Diabetes, Immune Enhancement, Cardiovascular Disease [sections on Homocysteine and Hypertension], Thyroid Deficiency, and Urinary Tract Infection for additional information on specific conditions and treatment.*)

Kidney Dialysis

Kidney dialysis is a medical treatment used to filter out waste products from the blood. Dialysis has been proven to be an effective technique for removing wastes and extra fluid from the body. According to the annual report of the U.S. Renal Data System (2001), over 243,000 persons in the United States were using dialysis treatment in 1999. Dialysis treatment permits these people to live relatively normal lives within the limitations of their disease.

There are two types of dialysis methods: hemodialysis and peritoneal dialysis. The most common technique is hemodialysis, accounting for slightly over 85% of dialysis treatment. The remaining 15% of patients use peritoneal dialysis (NIDDK 2001b). Neither hemodialysis nor peritoneal dialysis is uncomfortable and both are equally effective in removing wastes and extra fluids from the body. The choice is usually one of preference or level of convenience desired by the patient in consultation with appropriate medical professionals.

Even for patients who use dialysis, kidney failure can cause other health-related problems over time, including high blood pressure (including a latent nocturnal factor), bone disease, anemia, and nerve damage. As kidney function declines past the minimum threshold, kidney transplant becomes the only hope for patients with advanced ESRD.

Studies on human dialysis patients indicate that a high number of free radicals are formed in response to dialysis and that antioxidant dietary supplements can protect against this damage (Saionji 1999; Wratten 1999; Clermont et al. 2000).

Transplantation

Statistical surveys are made of medical facilities yearly. The most recent statistics available indicate that kidney transplantation accounted for 13,483 transplant operations in 1999 (NIDDK 2001b). In the United States, many people live with a functioning kidney as a result of transplantation. However, it is very difficult to obtain accurate statistics on the number of persons who live with a functioning kidney transplant at any given time. Unfortunately, each year patients die while awaiting a matching donor kidney. According to the NIDDK (2001b), as of November 2, 2001, there were 50,305 people waiting for kidney transplants. To be a potential candidate for kidney transplantation, a person must have kidney function estimated to be below 15% and must not be positive for certain diseases, such as unstable coronary artery disease, infection, or glomerulonephritis. (Glomerulonephritis is inflammation of the tiny blood vessels in the nephrons where blood is filtered in the kidneys. It is usually caused by an autoimmune disease, but can also be caused by infection.)

Can Renal Replacement Be Deferred? A study was conducted to determine if a very low-protein diet could defer renal replacement therapy (RRT) in patients with chronic renal failure. High protein intake is known to be stressful for the kidneys and over time can be a contributing factor to a slow, pervasive decline in kidney function. Two groups of patients (23 and 53 patients, respectively) were put on a very low-protein diet (0.3 g/kg) combined with supplemental amino acids. The patients in these groups were well-motivated RRT candidates who were closely monitored for nearly 1 year. During the course of the study, indications of malnutrition did not occur, and the patients were able to maintain acceptable kidney function (glomerular filtration rate or GFR < 10 mL/min or < 15/mL/min for diabetic patients) (Walser 1999).

Note: Since 1973, Medicare has picked up 80% of ESRD treatment costs, including the costs of dialysis and transplantation and of some medications. To qualify for benefits, a patient must be insured or eligible for benefits under Social Security or be a spouse or child of an eligible American. Private insurance and state Medicaid programs often cover the remaining 20% of treatment costs.

Natural and Adjuvant Treatment

- Dietary Management
- Inflammation
- Dietary Supplements

Dietary Management

In the early stages of kidney disease, careful dietary management may slow down the process of kidney disease. A diet low in sodium, potassium, and phosphorus, three substances regulated by the kidneys, is essential in managing kidney disease. Other dietary restrictions, such as reducing protein, may also be required. Your physician might suggest that you consult a renal dietitian who has special training in diets for persons with kidney disease. Persons who are vegetarians naturally have diets high in potassium and phosphorus and therefore need good nutritional advice. If you have to limit phosphorus, sodium, or protein, remember the following:

- **Phosphorus** is especially high in dairy products (milk, cheese, ice cream); dried beans and peas; nuts and peanut butter; some salt substitutes; and cocoa, beer and cola soft drinks.
- **Sodium** is especially high in table salt, canned soup, processed cheese, snack foods, prepared and "fast foods," pickles, olives, sauerkraut, and smoked and cured food (ham, bacon, luncheon meat).
- **Protein** is found in large amounts in food from animal sources (poultry, meat, seafood, eggs, dairy products). Protein is found in smaller amounts in food from plant sources (bread, cereal, grain, vegetables, fruit).

However, a certain amount of phosphorus, sodium, and protein is necessary for good health. To keep yourself healthy, it is important to learn to read labels and make better choices. For example, non-dairy creamers and milk substitutes are a good way to lower dietary phosphorus.

Avoid losing too much weight. It is important to maintain a good level of calories because calories give you energy. If you are limiting protein, you will need to get more calories from other foods. Good ways to increase calories are to:

- Increase unsaturated fats from vegetable oils (corn, cottonseed, safflower, soybean, sunflower), olive oil, and mayonnaise salad dressings.
- Use sugar from gum drops, jelly beans, marshmallows, honey, jam, and jelly.
- Use canned or frozen fruits in heavy syrup.

Note: The recommendations for using sugar may not be appropriate for diabetics or overweight individuals. If you are diabetic, consult your physician or dietitian for alternative recommendations.

Protecting Kidneys Against Inflammatory Attack

Pentoxifylline (PTX) is a prescription drug approved by the FDA to treat peripheral vascular disease. The standard dose is 1200 mg a day to improve circulation. To suppress proinflammatory cytokines often involved in age-related renal impairment, a lower dose of 400 mg twice a day can be used. (Refer to pentoxifylline precautions in the summary section before using this drug.)

A controlled study on human diabetics with advanced renal failure showed that 400 mg a day of PTX reduced tumor necrosis factor-alpha (TNF-alpha) levels by approximately 35%. In the pentoxifylline group, a measurement of kidney impairment was reduced 59%. There were no changes in those given placebo. The researchers noted that inflammatory cytokines such as TNF-alpha have long been implicated in the development and progression of diabetic kidney failure (Navarro et al. 1999a). Organ failure induced by

TNF-alpha has been confirmed by other studies (Boldt et al. 2001).

In advanced kidney failure, anemia can be induced by an inflammatory cytokine attack on erythropoietin, the major natural hormone responsible for red blood cell (RBC) production. In a group of 7 anemic patients with advanced renal failure, PTX suppressed TNF-alpha and reversed the anemic state (Navarro et al. 1999b).

Dietary Supplements

Dietary supplements are often recommended by physicians and renal dietitians (National Kidney Foundation 2001e). Their recommendations are guided by the results of blood tests that you will be required to take regularly as part of monitoring your condition and treatment results. Always speak with your physician or renal dietitian before using or adding any supplements or herbal products.

Multivitamins. In addition to eating a diet that contains appropriate nutrients and levels of protein, a comprehensive multivitamin is often required to replace vitamins that are lost during dialysis treatments (National Kidney Foundation 2001e).

Vitamin B. Vitamins B6, B12, and folate (folic acid) are members of the B vitamin group. The B vitamins are known for having many beneficial qualities, including promoting growth; improving heart function; lowering homocysteine; protecting against atherosclerosis caused by excess homocysteine; helping with the formation and regeneration of red blood cells and preventing anemia; and increasing energy and endurance (McGregor et al. 2000).

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Kidney Disease

Vitamin C. Vitamin C is an antioxidant that helps keep many different types of tissues healthy. Vitamin C helps wounds and bruises heal faster and may aid in preventing infection (2001e).

Vitamin D. Additional vitamin D, which promotes the absorption of calcium, along with calcium supplements, may also be recommended. Some physicians prescribe vitamin D in a pill form called vitamin D3 (National Kidney Foundation 2001e).

Vitamin E. Supplementation with vitamin E may protect the kidneys from free-radical damage, a major factor in renal health. In experiments in rats, Sadava et al. (1996) found that a dietary deficiency of vitamin E caused progressive and pronounced renal damage. Vitamin E has been shown to restore tubular flow to rats with severe kidney disease by suppressing the free radicals that cause tubulointerstitial damage (Hahn 1998).

Calcium. Calcium along with vitamin D helps keep your bones healthy. Calcium is also used to bind to phosphorus from dietary food. Your physician will advise you about taking calcium (National Kidney Foundation 2001e).

Phosphorus. The proper amount of phosphorus is needed for healthy bones. As noted earlier, when the kidneys do not work properly, blood levels of phosphorus can get too high, causing calcium to be taken from the bones. Calcium taken from the bones will make them weak. It is important to keep phosphorus and calcium balanced to maintain strong bones (National Kidney Foundation 2001c).

Potassium. Another function of the kidneys is to maintain the right amount of potassium. Potassium plays an essential role in keeping your heartbeat regular and your other muscles working properly, but high blood levels of potassium are to be avoided. You can help control your potassium level by avoiding foods that are high in potassium (National Kidney Foundation 2001d).

Iron. A low level of red blood cells is known as anemia. Because red blood cells carry oxygen to all the tissues and organs throughout the body, low levels of oxygen may result in reduced performance of vital organs including the kidneys. Anemia is common in people who have kidney disease. Healthy kidneys produce a hormone called erythropoietin (EPO). EPO stimulates the bone marrow to produce red blood cells. However, diseased kidneys often do not make enough EPO and therefore the bone marrow makes fewer red blood cells. Other common causes of anemia are from loss of blood during hemodialysis and low levels of iron and folic acid. Anemia often starts in the early stages of kidney disease and tends to worsen as the disease progresses. According to the NIDDK (2001a), nearly everyone with end-stage kidney failure has anemia.

A CBC (complete blood count) determines the hematocrit (Hct) or the percentage of the blood that consists of red blood cells. It also measures the amount of hemoglobin (Hgb) in the blood. If at least half of normal kidney function (serum creatinine is greater than 2 mg/dL) has been lost and Hct is low, the most likely cause of anemia is decreased EPO production. The National Kidney Foundation's Dialysis Outcomes Quality Initiative (DOQI) recommends that a detailed evaluation of anemia in men and postmenopausal women on dialysis should begin when the Hct value falls below 37%. For women of childbearing age, evaluation should begin when the Hct falls below 33%.

If no other cause for EPO deficiency is found, the deficiency can be treated with a genetically engineered form of the hormone (usually injected under the skin 2 or 3 times a week). Hemodialysis patients who cannot tolerate EPO injections in their skin can receive EPO intravenously during dialysis treatment. However, intravenous dosing requires a larger, more expensive dose and it may not be as effective.

Many people who need EPO treatment also need iron supplementation because EPO alone will not relieve the effects of anemia if iron levels are too low. Sometimes iron can be taken in a pill form, but according to the NIDDK, iron pills often do not work as well in people with kidney failure as iron given intravenously. Iron supplements should only be taken if prescribed by a physician based on blood analysis (National Kidney Foundation 2001e).

In addition to EPO and iron, some people also need vitamin B12 and folic acid supplements. Supplementation with EPO, iron, and appropriate B vitamins helps raise hemoglobin levels and most patients with kidney disease feel better, have more energy, and live longer (NIDDK 2001a).

L-Carnitine. For patients who are in a predialysis stage, are undergoing dialysis, or are post-transplant, nutritional supplementation with L-carnitine that has been lost during dialysis may reduce the side effects of common renal problems, such as cardiomyopathy and blood platelet aggregation, and may also help improve the patient's perception of their overall quality of life. L-carnitine is an amino acid that has shown effectiveness in providing cellular energy in both healthy individuals and those with chronic diseases.

General muscle weakness is a common complaint among patients undergoing hemodialysis. One study that measured the serum amount of L-carnitine found that hemodialysis lowered L-carnitine levels and posed new problems for patients (Wanic-Kossowska et

al. 1998). This study measured muscle atrophy via nerve conduction and velocity testing and found indications of "neurogenic atrophy of the muscles." This well-known type of muscle weakness was further studied by doctors in Japan who reported that low dosages of L-carnitine (500 mg daily) showed improvement in two-thirds of 30 patients who were studied for 12 weeks. The patients reported less muscle weakness, general fatigue, and cramps and aches. This study concluded that low doses of L-carnitine could improve muscle weakness and should be considered as a prolonged adjuvant therapy for dialysis patients (Sakurauchi et al. 1998).

ESRD affects every aspect of a patient's life. Therefore, improved quality of life is very important for dialysis patients, potentially affecting compliance with medical, nursing, and nutritional prescriptions. In one study, patients were given the Medical Outcomes Study Short Form to assess quality of life from their perspective before taking L-carnitine and at 1.5-month intervals for the duration of the study (Sloan et al. 1998). This double-blind study was conducted on 101 patients who received L-carnitine or placebo just before and immediately after dialysis. After 3 months of supplementation (1 gram of L-carnitine before and after every hemodialysis treatment), patients reported an "improved vitality and general health." It was noted that serum albumin concentration was directly correlated with the patients' feelings of well being.

A study of L-carnitine therapy on erythropoiesis and blood platelet aggregation was conducted in patients with chronic renal failure, and it was found that L-carnitine caused a "significant rise in collagen-induced platelet aggregation." The 22-month study divided the patients into three groups. Group I received erythropoietin; Group II received erythropoietin and L-carnitine; and Group III received L-carnitine. Iron concentration and platelet count measured in urea concentration were relatively unchanged. The rise of collagen was observed after only 2 months of L-carnitine therapy (Kalinowski et al. 1999).

Curcumin. A potent antioxidant extract from the spice turmeric (*Curcuma longa*), curcumin has a wide range of health benefits: antiviral, anti-inflammatory, anticancer, and cholesterol-lowering. An interesting study in rats investigated the effect of curcumin on nephrosis caused by adriamycin. Adriamycin is a drug commonly used in chemotherapy (Venkatesan et al. 2000). The results indicated that curcumin "remarkably" prevented kidney injury caused by adriamycin. Venkatesan et al. (2000) stated that their data demonstrated that curcumin offered protection "by suppressing oxidative stress and increasing kidney glutathione content and glutathione peroxidase activity." They suggest that administration of curcumin offers promise in the treatment of nephrosis that is caused by adriamycin.

Another group (Suresh Babu et al. 1998) studied the effect of curcumin on streptozotocin-induced diabetes. Streptozotocin is also a commonly used chemotherapy drug. According to Suresh et al. (1998), their data "suggested that dietary curcumin brought about significant beneficial modulation of the progression of renal lesion in diabetes." This benefit of dietary curcumin on diabetic nephropathy may be mediated by its ability to lower blood cholesterol levels.

Ginkgo Biloba. Already known for its antioxidant effects, ginkgo biloba may also protect small blood vessels against loss of tone, prevent capillary fragility, inhibit atherosclerosis, and treat diabetic vascular disease. Naidu et al. (2000) studied gentamicin-induced nephrotoxicity in rats. Gentamicin is an antibiotic used to treat serious infections. Unfortunately, it has the undesirable side effects of causing kidney damage and irreversible hearing loss. Naidu et al. (2000) found that gentamicin treatment increased levels of blood urea and serum creatinine. However, they also found that ginkgo biloba extract (GBE) protected the rats from gentamicin-induced nephrotoxicity by preventing changes in blood urea, serum creatine, and creatine clearance.

Also in a study in rats, Umegaki et al. (2000) examined the effects of GBE extract on the development of hypertension, platelet activation, and renal dysfunction in deoxycorticosterone acetate-salt hypertensive rats. After 20 days, the rats fed a 2% GBE diet had attenuated development of hypertension.

In another interesting study in rats by Fukaya et al. (1999), encouraging results of co-administration of cisplatin and GBE were reported. Cisplatin is an effective antineoplastic agent (cancer killing) used for treating solid tumors. However, cisplatin also has the undesirable side effects of causing hearing loss and nephrotoxicity. Fukaya et al. (1999) concluded that co-administration of cisplatin with GBE was beneficial to ameliorate cisplatin-induced toxicity without attenuating the antitumor activity of cisplatin.

Grape Seed Extract. Known for its powerful antioxidant qualities, grape seed extract also acts as a smooth muscle relaxant in blood vessels to combat hypertension. Ray et al. (2000) studied the protective effects of grape seed extract against biological, pharmacological, and toxicological effects of certain drugs to the kidneys, lungs, and heart in mice (acetaminophen, amiodarone, and doxorubicin). Ray et al. (2000) found that "grape seed extract preexposure prior to acetaminophen, amiodarone, and doxorubicin provided near complete protection in terms of serum chemistry changes and significantly reduced DNA fragmentation." Moderate to massive tissue damage occurred by all three drugs in the absence of grape seed extract. Bagchi et al. (2000) also found that grape seed extract "demonstrated excellent protection against acetaminophen overdose-induced liver and kidney damage."

Green Tea. Yokozawa et al. (1999) studied the effects of green tea tannin to ameliorate cisplatin-induced renal injury in rats. They found that green tea tannin suppressed the cytotoxicity of cisplatin, "the suppressive effect increasing with the dose of green tea tannin." Additional testing showed rats given green tea tannin had decreased blood levels of urea nitrogen and creatinine and decreased urinary levels of protein and glucose, indicating less kidney damage. Yokozawa et al. (1999) concluded that "based on

the evidence available, it appeared that green tea tannin eliminated oxidative stress and was beneficial to renal function." Earlier, researchers (Wardle 1999; Yokozawa et al. 1996) reported that green tea tannin was found to be beneficial for the kidney under oxidative stress. In 1991, Mukoyama et al. found that green tea had antiviral activity, inhibiting rotaviruses and enteroviruses in rhesus monkeys.

Soy. There is evidence that dietary phytoestrogens have a beneficial role in chronic renal disease (Velasquez et al. 2001; Ranich et al. 2001). Nutritional intervention studies demonstrated that consuming soy-based protein and flaxseed reduced proteinuria and attenuated renal functional or structural damage in both animals and humans. To date the studies have been of relatively short durations and involved small numbers of subjects. However, the results are encouraging and further investigations are needed. Three groups of researchers (Tomobe et al. 1998; Aukema et al. 1999; Ogborn et al. 2000) investigated the effects of a soy protein diet on polycystic kidney disease. Although the studies were conducted in rats and mice, the research teams suggested that dietary soy protein-based diets had beneficial effects in polycystic kidney disease: soy diet prevented significant elevation in serum creatinine in diseased vs. normal animals (Ogborn et al. 2000); soy protein is effective in retarding cyst development and this beneficial effect may be unrelated to genistein (an isoflavonoid present in soy protein) content (Tomobe et al. 1998); dietary protein level and source significantly affect polycystic kidney disease, with the effects being most pronounced in female animals fed low protein diets and soy protein-based diets (Aukema et al. 1999).

Taurine. Taurine is abundant in the brain, heart, gallbladder, and kidneys and plays an important role in health and disease in these organs. Taurine is an amino acid that has been shown to protect against experimentally induced lipid peroxidation of the renal glomerular and tubular cells and may alleviate tubular disorders such as glomerular impairment (Trachtman et al. 1996). It is also thought to lower blood pressure by balancing the ratio of sodium to potassium in the blood. Taurine may also regulate the increased nervous system activity that can contribute to high blood pressure. According to Franconi et al. (1995), some people with Type I diabetes appear to be deficient in the amino acid taurine.

Trimethylglycine (Betaine). Trimethylglycine (TMG) plays a role in the manufacture of carnitine and serves to protect the kidneys from damage (Chambers 1995). TMG has been reported to play a role in reducing blood levels of homocysteine, a toxic breakdown product of amino acid metabolism that is believed to promote atherosclerosis. The main nutrients involved in controlling homocysteine levels are folic acid, vitamin B6 and vitamin B12, but TMG has been reported to be helpful in some individuals whose elevated homocysteine levels did not improve with these other nutrients. TMG has also shown to be helpful in certain rare genetic disorders involving cysteine metabolism (Wilken et al. 1983; Wendel et al. 1984; Gahl et al. 1988; Barak et al. 1996; Selhub 1999; van Guldener et al. 1999). Its primary use as a nutritional supplement is in supporting proper liver function and possibly reducing the risk of urinary tract infections.

SUMMARY

The kidneys are remarkably resilient organs and can sometimes recover normal function from acute trauma as a result of injury, overdose of drugs, or poisoning, with prompt medical attention. However, there are forms of kidney disease that include conditions that can *rapidly* reduce kidney function or *slowly* reduce kidney function over several years, producing few or no symptoms. Damage from these conditions is not reversible. When kidney function is reduced to less than 10-15%, dialysis is required. When dialysis is no longer able to support kidney function, kidney transplantation is the only recourse.

If you have healthy kidneys, protect them. Start with a healthy diet; drink lots of water; give careful attention to the over-the-counter medicines you take, particularly when combined with prescription medicines or other over-the-counter products; consume alcohol responsibly (remember, over-the-counter or prescription drugs can be very damaging to the kidneys when combined with alcohol); protect your kidneys from injury if you engage in sporting activities; and consider taking protective supplements and nutrients to support overall kidney health.

As part of an annual physical checkup, request tests for blood levels of creatinine and blood urea nitrogen and urine levels of protein. Small elevations of creatinine can be an early sign of kidney disease. Early detection leads to early treatment which can occur at a stage when there is treatment to help prevent kidney disease from advancing to a more serious stage.

Because diabetes is the leading cause of chronic kidney disease, followed by hypertension, see your physician regularly and follow prescribed dietary and drug treatment to control blood sugar levels and hypertension (National Kidney Foundation 2001a) (refer to the Life Extension protocols on Diabetes and Hypertension for additional information).

Prevent damage to the kidneys from kidney stones by increasing water intake to 12 full glasses of water every day; limiting coffee, tea, and colas because caffeine increases fluid loss; increasing calcium intake using dietary factors; and including appropriate calcium/magnesium supplementation (taken only with food).

Research into gene therapy holds great hope for genetic kidney diseases. Of particular interest is research on the PKD1 gene, which is responsible for 85% or more of all ADPKD disease. ADPKD often progresses to kidney failure in young adulthood or middle

age and accounts for the need for kidney transplantation for many persons.

If you have early stage kidney disease or chronic kidney disease, follow the dietary recommendations of your physician or a renal dietitian. For example, a diet low in sodium, potassium, and phosphorus, three substances regulated by the kidneys, is essential in managing kidney disease. Other dietary restrictions, such as reducing protein, may be required depending on the cause of kidney failure and the type of treatment being used (e.g., such as dialysis). Patients with chronic kidney failure may also need to limit their fluid intake.

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Kidney Disease

Also follow your physician's recommendations concerning the addition of daily dietary supplements. Multivitamins, minerals, and other supplements may be prescribed or recommended to help replace essential nutrients lost during dialysis treatments. Consult medical professionals who are experienced in treating kidney disorders and follow their recommendations for treatment carefully. Establish good dietary habits that are appropriate for your situation. The following supplements are supportive of overall kidney health. The recommendations are for healthy individuals. If you have any form of kidney disease, consult your physician before adding or changing any supplements that you may currently be taking.

Supplements

1. Take a multivitamin to replace vitamins and minerals that are lost during dialysis or are deficient in your diet. Life Extension Mix is an excellent source of specific nutrients to defend the body against degenerative diseases. Life Extension Mix is available in several forms. Three tablets of Life Extension Mix Caps taken 3 times daily with meals are suggested.
2. Complete B-Complex contains safe and effective levels of all B-complex vitamins. Three capsules taken daily with meals are suggested.
3. Vitamin C has been shown to improve immune function, accelerate healing, and maintain healthy blood vessels. Vitamin C can come from dietary sources or from supplements: 2.5-6 grams daily from all sources are recommended. Each capsule of Vitamin C Caps provides 1000 mg of vitamin C (1 gram).
4. Vitamin D is necessary for proper utilization of calcium and phosphorus. Take one 1000-IU capsule of Vitamin D3 with fat-containing, low-fiber meals.
5. Vitamin E is known for its healing, cardiovascular, antioxidant, and immune-boosting benefits. Take one 400-IU capsule of Vitamin E succinate daily with fat-containing, low-fiber meals and 1-2 softgels of gamma tocopherol.
6. Calcium is vital for maintaining strong, healthy bones. Postmenopausal women should consume 1500 mg of calcium daily (diet plus supplementation). Menopausal women and men should consume 1200mg daily. Bone Assure is most effective if taken at night with low-fiber meals.
7. Iron supplementation is often required for people who have anemia and need EPO treatment as a result of kidney disease and hemodialysis. If iron levels are too low, EPO alone will not relieve the effects of anemia. Sometimes iron can be taken in a pill form. However, iron may be more effective if given intravenously.
Only take iron pills if your physician prescribes them for you.
8. L-carnitine has beneficial qualities for conditions associated with low cellular energy, immune dysfunction, and diabetic complications. One to four 500-mg capsules of L-carnitine capsules taken in divided doses on an empty stomach with juice or water are suggested.
9. Curcumin is a potent antioxidant with antiviral, anti-inflammatory, anticancer, and cholesterol-lowering benefits. One 900-mg capsule of curcumin taken with meals is recommended.
10. Ginkgo biloba has been used for its protective qualities in vascular disease. Super Ginkgo Extract provides more of the active ingredients, but eliminates most of the ginkgolic acid part of the ginkgo leaf. Take one 120-mg capsule of Super Ginkgo Extract daily.
11. Grape seed extract has powerful, natural free radical scavengers. It can act as a relaxant for blood vessels to combat hypertension. One to two 100-mg capsules of grape seed extract daily are suggested.
12. Green tea has demonstrated many protective qualities, including its antioxidant benefits (neutralizing cancer-causing agents and protecting against free-radical damage); reducing cholesterol, blood glucose, and blood pressure levels; and inhibiting viruses and bacteria. Take one 725-mg capsule of Super Green Tea Extract (95%) with meals daily for prevention purposes. For those who are sensitive to caffeine or do not want to consume it, Mega Green Tea (98% decaf) is available in a decaffeinated form. Consult your physician when taking larger quantities for disease treatment purposes.
13. Soy has protective qualities for kidney function in addition to its well-known anticancer, cholesterol lowering, and post-menopausal symptom alleviating benefits. Consider taking one Super Absorbable Soy Isoflavones capsule twice daily for general disease prevention purposes.
14. Taurine has potential benefits for the kidneys. By helping to lower blood pressure, it also regulates the increased nervous system activity that can contribute to high blood pressure. People with Type I diabetes may be deficient in taurine. One to four 1000-mg capsules of Taurine Capsules taken daily either with meals or on an empty stomach are suggested.
15. TMG (betaine) is possibly one of the most important nutrients with preventative benefits for heart disease, stroke, liver disease, and to slow aging. Consider taking one to five 500-mg tablets of TMG tablets daily.
16. The docosahexaenoic acid (DHA) fraction of fish oil may be the most effective non-prescription supplement to suppress pro-inflammatory cytokines. EPA and GLA are also effective anti-inflammatory fatty acids.
17. DHEA, a hormone that decreases with age, has been shown to suppress IL-6, an inflammatory cytokine that often increases with age. Typical doses of DHEA are 25-50 mg daily (although some people take 100 mg daily). Refer to the DHEA protocol for suggested blood tests to safely and optimally use DHEA.
18. Consider taking nettle leaf (1000 mg daily) to suppress the pro-inflammatory cytokine TNF-alpha.
19. Vitamin E and N-acetyl-cysteine (NAC) are protective antioxidants with anti-inflammatory properties. Take 1-2 capsules daily of Gamma E Tocopherols/Tocotrienols. NAC is an amino acid with antiviral and liver protectant properties. One 600-mg capsule daily is recommended.

20. Vitamin K helps reduce levels of IL-6, an inflammatory cytokine. One 10-mg capsule daily is recommended for prevention purposes.
21. Magnesium deficiency leads to increase of urine alkalinity, often resulting in the formation of calcium phosphate stones. Magnesium (500 mg daily) reduces calcium absorption.

Overlooked Prescription Drug

Kidneys are especially vulnerable to attack by proinflammatory cytokines. Pentoxifylline (PTX) is a drug that has been shown to protect against this type of kidney damage. The suggested dose is 400 mg twice a day of PTX.

PTX should not be used in patients with bleeding disorders such as those with recent cerebral or retinal hemorrhage. Patients taking Coumadin should have more frequently monitored of prothrombin time. Those suffering from other types of bleeding should receive frequent physician examinations. Furthermore, we would consider evaluating the individual patient's coagulation status to see what effect PTX has on the template bleeding time. This is an inexpensive test that relates the biological effect of PTX or other agents like aspirin (nonsteroidal anti-inflammatory agents) on the function of platelets. All of these agents affect platelet aggregation and this effect can be manifested in a prolonged template bleeding time. According to two studies, PTX should be avoided by Parkinson's patients. It is important to note that the body does use TNF-alpha to acutely fight infections. If patients are showing any sign of infectious disease, drugs like Enbrel that inhibit the effects of TNF-alpha are temporarily discontinued. A new FDA advisory states that patients should be tested and treated for inactive, or latent, tuberculosis prior to therapy with another TNF-alpha inhibiting therapy (e.g., infliximab). Since PTX, fish oil, and nettle directly suppress TNF-alpha, perhaps these agents should be temporarily discontinued during the time when one has an active infection.

FOR MORE INFORMATION

Contact the National Kidney Foundation, (800) 622-9010 or <http://www.kidney.org>; the American Foundation for Urologic Disease, (800) 242-2383 or www.access.digex.net/~afud; and the National Kidney and Urologic Diseases Information Clearinghouse, (301) 654-4415 or e-mail nkudic@info.niddk.nih.gov ; (website) www.niddk.nih.gov , for more information.

PRODUCT AVAILABILITY

Life Extension Mix, Complete B-Complex, vitamin D3, vitamin E succinate, gamma tocopherol, vitamin C, Iron Protein Plus, DHEA, NAC, vitamin K, L-carnitine, curcumin, Super Ginkgo Extract, grape skin extract, Super Green Tea Extract (95%), green tea, soy isoflavones, Optizinc, taurine, TMG, calcium, and magnesium are available by telephoning (800) 544-4440 or by ordering online.



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