Nutrition Basics for Adult Solid Organ Transplant — An Overview
By Patsy Obayashi, MS, RD, CDE

Suggested CDR Learning Codes: 3020, 5000, 5360, 5410; Level 3

Suggested CDR Performance Indicators: 8.3.1, 8.3.6, 10.2.4, 10.4.2

The loss of organ function has a profound impact on one's ability to lead a normal life. Invariably, modification of macro- and micronutrients are necessary to compensate for changes in fluid balance, blood pressure, and lipid or carbohydrate metabolism in patients with organ failure. Methods of compensating or substituting for a failing organ may have limited success for some organs, such as hemodialysis or peritoneal dialysis for kidney failure, the left ventricular assist devices for some types of heart failure, and insulin pumps for diabetes.

Transplantation, however, offers the chance for correction of liver, lung, heart, kidney, and pancreas failure. For patients with functioning transplanted organs, immunosuppressive medications are necessary to prevent rejection of the organs. These medications, which must be used for life, have significant nutrition-related side effects, such as hypercholesterolemia, osteoporosis, hypertension, and weight gain, but many can be addressed with medical nutrition therapy (MNT) as provided by RDs.

Peri-Transplant Nutrition
The primary focus of nutrition in the pretransplant period is on minimizing the effects of progressive organ dysfunction while promoting adequate protein and calorie intake to maintain lean muscle mass, correct nutrition abnormalities associated with organ failure, and optimize weight.[1]

Fluid retention associated with cardiac, renal, or hepatic failure affects blood pressure. Sodium and fluid restrictions may be necessary to control hypertension. In patients with liver or cardiac failure, protein, phosphorus, or potassium restrictions may also be necessary if renal dysfunction occurs.

Treatment modalities such as hemodialysis (HD) or peritoneal dialysis (PD) may exacerbate fluid and electrolyte abnormalities and increase patients' needs for calories and protein to replace losses resulting from treatment.[1] Because the liver regulates gluconeogenesis and the kidneys are involved in insulin clearance, consistent control of carbohydrates and insulin is key to the management of hyper- and hypoglycemia in patients with liver and kidney failure and those undergoing transplant.

The primary goals of nutrition prior to transplant are to replace nutrient losses in order to maintain adequate muscle and energy stores, promote an adequate BMI, and maximize quality

of life while managing symptoms of organ failure such as fatigue, hypertension, and hyperglycemia in conjunction with medications and medical management.[1]

Primary post transplant complications include infection and cardiovascular risks.[2] Malnutrition prior to transplant increases an individual's risk for developing infection and decreases survival. Immediate post transplant goals are to provide adequate nutrition to support wound healing and replete nutrient losses.[2]

RDs can help patients meet long-term goals to maintain optimal organ function and minimize risks that increase morbidity and mortality by helping them make nutritionally well-balanced food choices and dietary adjustments in response to immunosuppressive medication side effects. Table 1 provides a comparative summary of potential side effects of immunosuppressive medications and symptoms of organ failure. Adjustment of medications in addition to improved organ function following transplant can improve symptoms related to immunosuppressive medications in the transplant recipient, while symptoms associated with end organ failure remain more difficult to control. Further discussion can be found below pertaining to specific organs.

Table 1: Potential Side Effects of Immunosuppressive Medications and Symptoms of End-Organ Failure.[1,2]

Side Effect	End Organ Failure	Transplant Immunosuppressive Medications
Hypertension	X	X
Hyperglycemia	X (diabetes, infection, sepsis)	X
Hyperlipidemia	X (cardiac, COPD, dialysis, diabetes)	X
Hyperkalemia	X (renal)	X
Hypomagnesemia	chronic malnutrition	X
Osteoporosis	X (renal, lung)	X
Nausea/vomiting	X	X
Diarrhea	X (cystic fibrosis, malabsorption, lactulose for liver disease, diabetic gastroparesis)	X (post transplant infection as cytomegalovirus, *Clostridium difficile*)
Weight gain	X	X
Weight loss	X (cachexia, diuresis)	
Fluid retention	X	X

REFERENCES

1. LAM, HD. NUTRITIONAL REQUIREMENTS OF ADULTS BEFORE TRANSPLANTATION. MEDSCAPE WEBSITE. HTTP://EMEDICINE.MEDSCAPE.COM/ARTICLE/431031. UPDATED APRIL 7, 2015.
2. DICECCO SR, FRANCISCO-ZILLER N, MOORE D. OVERVIEW AND IMMUNOSUPPRESSION. IN: HASSE JM, BLUE LS, EDS. *COMPREHENSIVE GUIDE TO TRANSPLANT NUTRITION*. CHICAGO, IL: AMERICAN DIETETIC ASSOCIATION; 2002:1-30.

Tables 2 and 3 summarize pre- and post transplant nutrition recommendations for each solid organ.

Table 2: Pretransplant/Organ Failure Nutrition Goals (Based on kg Estimated Dry Weight)

	Kidney[1]	Liver[2]	Heart[3]	Lung[2,3]	Pancreas[4]
Protein	1.2 to 1.4 g/kg, >50% high biologic value (HBV) (stage 5, hemodialysis [HD], maintenance) 1.2 to 1.3 g/kg, >50% HBV (peritoneal dialysis [PD], maintenance) 1.5 to 2.5 g/kg (HD, repletion)	1 to 1.5 g/kg (maintenance) 1.5 to 2 g/kg (repletion)	1.1 to 1.3 g/kg	Dietary Reference Intake	Based on individualized need such as nephropathy or chronic wound healing
Kilocalories	30 to 35 kcal/kg dry or adjusted for obesity	25 to 30 kcal/kg dry or adjusted for obesity (maintenance) 35 to 40 kcal/kg dry or adjusted for obesity (repletion)	Assess by indirect calorimetry or multiply basal energy expenditure by catabolic state	Assess by indirect calorimetry due to high individual variability	Total energy intake appropriate to weight management goals.
Fat	30% Kcal	30% Kcal	25% to 35% kcal, <200 mg cholesterol and <7% calories from saturated fat if risk factors present	30% Kcal	Individualized total fat, <200 to 300 mg cholesterol, <10% calories from saturated fat
Fluid	Urine + 500-1000 mL (HD) Urine + 1000 mL (PD)	1 to 1.5 L (if hyponatremia)	1.4 to 1.9 L (if hyponatremia)	As desired, restrict if hyponatremia	As desired
Electrolytes and Minerals	HD: <2.4 g sodium <2.4 g potassium PD: 2 g sodium, 3 to 4 g potassium, 800 to 1000 mg phosphorus <2000 mg calcium,	2 g sodium, replete calcium	< 2 g sodium, monitor potassium, magnesium, calcium	2 g sodium (if hypertension), high salt for cystic fibrosis, adequate calcium	<2300 mg sodium (if hypertention)
Vitamins	3 mcg vitamin B12, 1-5 mg folate, vitamin B1 1.5-2 mg (PD), IV iron supplementation if serum ferritin <200 ng/ml (HD) or <100 ng/mL (PD), 15 IU vitamin E, vitamin D sufficient to maintain adequacy, vitamin C within DRI	Repletion vitamin A, D, E, K, Zinc	DRI for folate, thiamin, vitamin B6, vitamin B12 (200 to 500 mcg/day)	Fat soluable vitamin supplementation, AI for Omega-3Fatty Acids, RDA for vitamin C and E, adequate vitamin D, K	RDA

REFERENCES

1. AMERICAN DIETETIC ASSOCIATION. *CHRONIC KIDNEY DISEASE EVIDENCE-BASED NUTRITION PRACTICE GUIDELINE*. CHICAGO, IL: AMERICAN DIETETIC ASSOCIATION; 2010.
2. LAM HD. NUTRITIONAL REQUIREMENTS OF ADULTS BEFORE TRANSPLANTATION. MEDSCAPE WEBSITE. HTTP://EMEDICINE.MEDSCAPE.COM/ARTICLE/431031. UPDATED APRIL 7, 2015.
3. ACADEMY OF NUTRITION AND DIETETICS. EVIDENCE ANALYSIS LIBRARY. HTTPS://WWW.ANDEAL.ORG
4. EVERT AB, BOUCHER JL, CYPRESS M, ET AL. NUTRITION THERAPY RECOMMENDATIONS FOR THE MANAGEMENT OF ADULTS WITH DIABETES. *DIABETES CARE*. 2014;37(SUPPL 1): S120-S143.

Table 3: Post Transplant Nutrition Goals (Based on kg Dry or Adjusted Weight for Obesity)

	Kidney[1,2]	Liver[2,3]	Heart[4]	Lung[4]	Pancreas[5]
Protein Short Term	1.3 to 2.0* g/kg * higher levels for dialysis	1.5 to 2.0 g /kg	1.3 to 1.5 g/kg t (maintenance) ≤ 2.5 gm/kg (infection, repletion)	1 to 1.5 g/kg (maintenance) 1.5-2 gm/kg (repletion)	1.3 to 2 g /kg
Long Term	0.8 to 1.0 g protein/kg	0.8 to 1.0 g protein/kg	1.0 to 1.3 g/kg	1.0 to 1.3 g/kg	0.8 to 1.0 g/kg
Kilocalories Short Term	30 to 35 kcal/kg	30 to 35 kcal/kg	35 kcal/kg	35 kcal/kg	30 to 35 kcal/kg
Maintenance	25 to 30 kcal/ kg or adequate to maintain desireable weight	25 to 30 kcal/kg	25 to 30 kcal/kg	25 to 30 kcal/kg	25 to 30 kcal/kg
Fat Short Term	30% to 50% non-protein kcal	25% to 35% non-protein Kcal, <300 mg cholesterol, 7 to 10% calories from saturated fat	<30% Kcal, <300 mg cholesterol, <10% calories from saturated fat	<30% Kcal, <300 mg cholesterol, <10% calories from saturated fat	30% to 50% non-protein kcal
Maintenance	<30% total kcals, <300 mg cholesterol, 7% to 10% calories from saturated fat	25 to 30% total calories, <300 mg cholesterol, <7% calories from saturated fat	<300 mg cholesterol, 7% to 10% calories from saturated fat	<300 mg cholesterol, 7% to 10% calories from saturated fat	<300 mg cholesterol, 7% to 10% calories from saturated fat
Carbohydrate Short Term	50% to 70%	50% to 70%	50% to 70%	50% to 70%	50% to 70%
Maintenance	45% to 50% total calories, 25 to 30 g fiber/day	50% non-protein kcals, 25 to 30 g fiber/day	50% to 60% total calories, 25 g fiber/day	50% to 60% total calories, 25 g fiber/day	45% to 50% total calories
Fluid	Ad Lib	Ad Lib	Ad Lib	Ad Lib	30 to 40 ml/kg/day (enteric drainage) 50 to 60 ml/kg/day or equal to output (bladder drainage)
Electrolytes & Minerals	No added salt, monitor potassium, phosphorus, magnesium, 1000 to 1500 mg calcium	No added salt, monitor potassium, phosphorus, magnesium, 1000 to 1500 mg calcium	No added salt, monitor potassium, phosphorus, magnesium, 1000 to 1500 mg calcium	2-4 g sodium if hypertension or edema, potassium, phosphorus, magnesium, 1500 mg calcium,	Additional sodium and bicarbonate (bladder drainage) monitor potassium, phosphorus, magnesium. Supplement calcium
Vitamins	Supplement vitamin D	400 to 1000 IU vitamin D	400 to 1000 IU vitamin D, 5 mg folic acid,	No specific guidelines for vitamin D 10	Supplement vitamin D

REFERENCES

1. BLUE LS. ADULT KIDNEY TRANSPLANTATION. IN: HASSE JM, BLUE LS, EDS. *COMPREHENSIVE GUIDE TO TRANSPLANT NUTRITION*. CHICAGO, IL: AMERICAN DIETETIC ASSOCIATION; 2002:44-57.
2. THIEM U, OLBRAMSKI B, BORCHHARDT K. CALCIDIOL DEFICIENCY IN END-STAGE ORGAN FAILURE AND AFTER SOLID ORGAN TRANSPLANTATION: STATUS QUO. *NUTRIENTS*. 2013;5(7):2352-2371.
3. HASSE JM. ADULT LIVER TRANSPLANTATION. IN: HASSE JM, BLUE LS, EDS. *COMPREHENSIVE GUIDE TO TRANSPLANT NUTRITION*. CHICAGO, IL: AMERICAN DIETETIC ASSOCIATION; 2002:58-89.
4. PAHWA N, HEDBERG A. ADULT HEART AND LUNG TRANSPLANTATION. IN: HASSE JM, BLUE LS, EDS. *COMPREHENSIVE GUIDE TO TRANSPLANT NUTRITION*. CHICAGO, IL: AMERICAN DIETETIC ASSOCIATION; 2002:31-43.
5. OBAYASHI PA. ADULT PANCREAS TRANSPLANTATION. IN: HASSE JM, BLUE LS, EDS. *COMPREHENSIVE GUIDE TO TRANSPLANT NUTRITION*. CHICAGO, IL: AMERICAN DIETETIC ASSOCIATION; 2002:90-105.

Kidney

The kidney is the most commonly transplanted organ.[3] Hypertension and diabetes are responsible for up to 60% of chronic renal failure, according to the National Kidney Foundation.[4] In 2011, 95,000 people waited for kidney transplant and 16,813 received transplants nationally.[5] Chronic glomerulonephritis, polycystic kidney disease, systemic lupus erythematosis, renal stones, chronic pyelonephritis, medications such as angiotensin-converting enzyme inhibitors, and dyes used for special imaging tests can also lead to chronic renal failure. If kidney function is severely diminished, dialysis becomes necessary or transplant may be an alternative for many patients. However, factors such as the presence of comorbid conditions (eg, cancer or advanced age) and, more significantly, a lack of available organs, limit the number of people who may become transplant recipients. The Centers for Disease Control and Prevention estimates that 10% of adults in the United States (more than 20 million people) may have chronic kidney disease, a number that continues to grow each year.[6] This compares to 101,170 people awaiting kidney transplant and 16,896 people receiving a transplant in 2013.[7]

Protein-calorie malnutrition in chronic renal failure is associated with anorexia; blood, protein, and nutrient losses during dialysis; and catabolism resulting from chronic disease. As kidney function deteriorates, and before dialysis becomes necessary, strict protein limits may be needed to prevent excess waste products from accumulating. Once the patient begins dialysis, protein losses due to HD may range from 2 to 8 g per day and 5 to 12 g per day during PD.[1] Patients need 1.2 to 1.3 g of protein per kilogram dry weight, with at least 50% containing all the essential amino acids for high biological value, to maintain adequate protein stores.[8]

Dietary potassium and phosphorus restrictions are necessary in patients with chronic renal failure because their kidneys cannot clear these minerals. If there is rejection of the transplanted organ or side effects of medications occur (see tip sheet), dietary potassium or phosphorus may need to be temporarily restricted. Fluid restriction is also necessary in patients with chronic renal failure because dialysis isn't as effective as the kidney in removing fluid and waste from the body. Patients on PD may be allowed a more liberal fluid, sodium, and potassium restriction than that required by HD.[9]

In chronic kidney failure, cholesterol levels may be elevated due to impaired high-density lipoprotein metabolism that disturbs the clearance of triglyceride-rich lipoproteins. Two mechanisms contribute to nephrotic dyslipidemia: overproduction and impaired breakdown of apolipoprotein B-containing lipoproteins and decreased breakdown of chylomicrons and very low-density lipoproteins.[10] Dyslipidemia may increase the risk of atherosclerotic cardiovascular disease and adversely affect the progression of renal disease and energy metabolism in chronic renal failure.[10]

Without supplementation, patients may be deficient in vitamin B6, folic acid, vitamin C, vitamin D, and iron, due to losses associated with dialysis and in conjunction with food restrictions. The kidneys have vitamin D receptors and play a major role in turning vitamin D into its active form. However, because vitamin C is excreted by the kidney, supplemental amounts of vitamin C greater than 100 mg/day should be avoided due to increased risk of oxalosis, a metabolic by-product of ascorbic acid that can deposit in the kidney.[8]

The risk of hypoglycemia is increased in patients with end-stage renal failure due to decreased clearance of insulin and antihyperglycemic medications. Impaired renal gluconeogenesis with reduced kidney mass also slows glucose production. For example, a patient has type 2 diabetes and takes Metformin and Lantus insulin to control blood sugars. As his kidney function becomes worse, he experiences blood sugars below 40 mg/dl at least twice a week because diabetes medications aren't being excreted normally and remain in his body longer, continuing to lower blood glucose; moreover, endogenous glucose production is reduced due to decreasing kidney function, so the body is unable to quickly raise blood sugar levels. About one-third of insulin breakdown occurs in the kidneys, and impairment of kidney function is associated with a prolonged half-life of insulin.[11] Careful monitoring and adjustment of diabetes medications is necessary to prevent hypoglycemia.

Post kidney transplant, excess protein losses decrease when dialysis is no longer necessary; potassium and phosphorus levels can normalize and no longer create the need for dietary restrictions. Vitamin B12, B1, folic acid, iron, and vitamin E losses are also alleviated with a functioning kidney post transplant. There's no consensus on the need for vitamin D supplementation, although needs are thought to be higher in patients after transplant than in the general population.[11]

Preexisting diabetes may be responsible for more than 40% of renal failure leading to transplant. Four percent to 25% of kidney transplant recipients develop new onset diabetes post transplant (NODPT).[12] Medications such as corticosteroids, a basic class of medications used to prevent rejection post transplant, can cause hyperglycemia and weight gain. (See medications tip sheet). Other transplant immunosuppressive medications can also exacerbate hyperglycemia. Hyperglycemia can increase risks of dehydration, delay wound healing, and increase risk of infection, which negatively affect kidney function after transplant.

A post transplant BMI higher than 28 kg/m2 is associated with increased mortality[2] due to increased risks associated with metabolic syndrome and cardiovascular disease. On average and regardless of steroid dose, 10% weight gain occurs in the first year after kidney transplantation and is largely in the form of fat accumulation in the central abdomen.[13] This increases the risk for hypertension, stroke, diabetes, coronary artery disease, and mortality.

Changes in food intake, liberalization of diet, and alterations in activity after transplant will also influence weight gain. Patients should be strongly encouraged to maintain a diet controlled in sodium, fats, and overall calories to maintain a healthy weight[2] and blood pressure. Supplemental calcium and magnesium to compensate for decreased absorption associated with immunosuppressive medications and vitamin D to support bone health are also advised.[14,15]

Liver
Less than 25% of all transplants performed involve the liver.[16] Nationally in 2011, 16,500 individuals waited for a liver transplant and only 6,342 received a transplant.[5] Transplantation is the treatment of choice for decompensated cirrhosis, acute liver failure, small hepatocellular carcinomas that result from viral hepatitis B and C, primary biliary cirrhosis, primary sclerosing cholangitis, alcoholic liver disease, and nonalcoholic steatohepatitis/nonalcoholic fatty liver

disease.[17] According to the American Liver Foundation, more than 30 million people in the United States, or one in 10 Americans, have liver disease. Up to 25% of Americans may have nonalcoholic fatty liver disease, a number that rises with the increase in obesity.[18]

Malnutrition may occur in 34% to 82% of individuals with alcoholic cirrhosis and in 27% to 87% of those with nonalcoholic cirrhosis.[1] Malnutrition prior to liver transplant results from inadequate diet, malabsorption associated with bile salt or exocrine pancreatic insufficiency, and decreased fat absorption. Ascites is defined as the accumulation of fluid in the peritoneal cavity, causing abdominal swelling, and may cause early satiety and anorexia due to increased pressure on the stomach, diaphragm, and intestinal tract that reduces gastric capacity. The addition of a bedtime snack containing complex carbohydrates is more effective in helping to prevent muscle breakdown, and improves nutrient utilization and nitrogen retention during sleep than is daytime calorie supplementation alone.[19] Nutrient-dense food choices and supplements, consumed every three to six hours, can help improve survival, and may reduce the frequency and severity of hepatic encephalopathy because when amino acids are used for glucose production, the body's protein stores become depleted and ammonia production is increased.[19,20] Control of dietary sodium to minimize fluid retention and adequate protein to promote positive nitrogen balance and prevent breakdown of protein stores are recommended. Breakdown of amino acids for energy increases loss of amino acids which increases protein needs so protein restriction is discouraged.[19] Supplementation of branched-chain amino acids (leucine, isoleucine, valine) may help improve nutritional status and anorexia; however, poor palatability interferes with long-term compliance.[1]

Glycogenesis, gluconeogenesis, glycogenolysis, and glycolysis occur in the liver to regulate the supply of glucose to the body. Hyperglycemia that develops as a complication of cirrhosis may be related to insulin resistance in muscle and adipose tissues, hyper-insulinemia, an impaired response of the pancreatic islet β-cells, and/or hepatic insulin resistance. As liver function deteriorates, glycemic control can become more difficult; hypoglycemia is often associated with liver dysfunction.[21] Hepatitis C is associated with insulin resistance related to tumor necrosis factor (TNF) production and overproduction of cytokines.[21] A consistent carbohydrate-controlled diet with frequent feedings is recommended in conjunction with appropriate antihyperglycemic medications including insulin.[21,22]

In primary sclerosing cholangitis and primary biliary cirrhosis, fat-soluble vitamins A, D, E, and K may be deficient due to steatorrhea or fatty stools; these can be supplemented in a water-soluble form. Supplementation with calcium and zinc may also be necessary.[1,23]

After liver transplant, immunosuppressive medications such as tacrolimus, cyclosporine, and corticosteroids prevent rejection of the transplanted organ but also may exacerbate weight gain, hyperglycemia, and hypertension. (Transplant medications are discussed in detail under "common immunosuppressive medications and their side effects"; also see medication tip sheet.) A prospective study in 2005 by Richards et al showed an average of 5 kg gain in the first year and 10 kg by third year after liver transplantation.[24]

The incidence of NODPT in the liver transplant population may range from 2.5% to 25% in patients with hepatitis C, and 4% to 60% of that incidence may be related to effects of

tacrolimus and hepatitis C.[12] A diet controlled in fats and overall calories to maintain a healthy weight is encouraged. A no-added-salt diet can help control hypertension associated with immunosuppressive medications. Supplemental calcium and magnesium to compensate for decreased absorption associated with immunosuppressive medications and vitamin D to support bone health are also advised.[15,22]

Heart
Less than 10% of all transplantations involve the heart.[16] Three thousand individuals wait for heart transplant in the United States, yet there are only 2,000 donor hearts available.[25] Chronic heart failure, usually due to cardiomyopathy or ischemic heart disease, is the most common indication for heart transplant.[25] Nausea and anorexia associated with chronic heart failure lead to malnutrition that causes muscle loss in the heart and increases the risk of cardiac cachexia.[26] To maintain blood pressure with a weakened heart, the body retains salt and fluid; this may reduce blood flow to the skin and gut to maintain circulation to vital organs. Less blood flow to the stomach causes gastrointestinal hypomotility with delayed gastric emptying. This is associated with edema of the stomach and bowel that interferes with appetite and hunger and may also cause nausea and/or vomiting. Reduced blood flow to the stomach causes malabsorption and exacerbates weight loss and muscle wasting.[26]

Transplantation is considered in the 10% to 15% of patients with advanced heart failure who develop severe weight loss. Within this population, 60% develop muscle wasting; those with cardiac cachexia have a 50% mortality rate in 18 months.[27] Congestion of the liver, spleen, and lungs can also be related to fluid and sodium retention. In addition, hypermetabolism is associated with increased cardiac and respiratory effort. Edema and pulmonary congestion decrease exercise capacity, making simple activities such as eating and chewing exhausting.[26] Anxiety associated with difficulty breathing may interfere with adequate food intake. Visceral muscle wasting and loss of fatty tissue increase risk of morbidity and mortality.[1] Encourage small, frequent, nutrient-dense meals for patients with early satiety or an inability to eat large enough amounts of food to meet nutritional needs. A diet restricted to less than 2,000 mg sodium and less than 2,000 mL fluid per day is prescribed to manage symptoms of heart failure.[28]

A low-cholesterol, low-saturated fat diet is indicated for any individual with risk factors for high cholesterol, high low-density lipoprotein cholesterol, and low high-density lipoprotein.[28] Because some immunosuppression medications can cause hyperlipidemia, transplant patients should also follow a low-cholesterol, low-saturated fat diet. Weight reduction in morbidly obese (>30 kg/m2) patients with chronic congestive heart failure can prevent disease progression, decrease symptoms, and improve well-being.[29] Calorie needs are best determined using indirect calorimetry due to high variability of individual needs. Resting energy expenditure (using predictive equations such as Mifflin-St Jeor or Harris Benedict) with a high catabolic state stress factor may be used if indirect calorimetry isn't available, but information may be less accurate.[28]

After a transplant, cardiac cachexia can be reversed. Weight gain of 8 kg to 10 kg within the first 12 months following transplant may lead to NODPT and metabolic syndrome.[29] Excess intra-abdominal fat is associated with metabolic syndrome. Increases in BMI may reflect

obesity as well as fluid overload.[29] Hyperglycemia occurs when renal and hepatic insulin metabolism is restored following transplant and is exacerbated by post transplant weight gain, obesity, and immunosuppressive medications.

Calciuneurin inhibitors (CNI) are a key class of immunosuppressive drugs that include cyclosporine and tacrolimus; these, along with corticosteroids and other transplant medications, such as sirolimus, impair glucose metabolism, lipid metabolism, blood pressure control, and renal function.[29] As a result, the incidence of NODPT in the heart transplant population may range from 4% to 40%.[12]

A diet controlled in fats and overall calories to maintain a healthy weight is encouraged. A no-added-salt diet can help control hypertension associated with immunosuppressive medications. Supplemental calcium and magnesium to compensate for decreased absorption associated with immunosuppressive medications and vitamin D to support bone health are also advised.[26]

Lung

Only about 5% of transplants involve the lung.[16] Lung transplantation is most often performed to treat severe COPD, emphysema, cystic fibrosis (CF), pulmonary fibrosis, bronchiectasis, and pulmonary hypertension.[26] Approximately 1,700 people wait for lung transplant nationally each year.[30] Chronic infection, seen in bronchiectasis and CF, is associated with increased cachectin production. Cachectin, or TNF, is a hormone known to cause wasting and loss of body mass.[26] In emphysema and cystic fibrosis, increased effort to breathe creates hypermetabolism and is responsible for the greatest incidence of malnutrition. Protein-calorie malnutrition impairs immune response, which increases susceptibility to lung infections.[31] Hyperinflated chest, seen in emphysema, can lead to early satiety. With this condition, blood flow through the heart and lungs is poor, causing fluid to accumulate; this results in bloating and early satiety.[32] Edema and ascites from intra-abdominal pressure and hypoxia cause anorexia.[1,26] Calorie needs are best determined by multiplying resting energy expenditure by stress factor, or indirect calorimetry, due to the high variability of individual needs.[28] Frequent, small, nutrient-dense feedings and supplements are key to meeting needs.

CF, a genetic disorder for which there is no cure, is associated with malabsorption. Glucose intolerance and diabetes related to CF often accompany the pancreatic insufficiency that's inherent to the disorder. CF and pancreatic insufficiency require supplementation with fat-soluble vitamins and pancreatic enzymes for proper absorption. Carbohydrates shouldn't be restricted. Appropriately dosed insulin will allow for proper utilization of calories to maintain optimal nutrition status.[28]

Chronic pancreatic insufficiency associated with CF is routinely managed with administration of pancreatic enzymes. Through use of pancreatic enzymes, weight gain is possible. However CF-related pancreatic insufficiency continues after transplant because the lung transplant only provides improved lung function and does not cure cystic fibrosis,[33] making weight gain more difficult. Chronic malnutrition generally increases risks of post transplant infections, particularly in lung transplantation. Infection is the most significant complication leading to death in the first year after lung transplant.[31] Five to six frequent, nutrient-dense feedings and high-calorie, high-

protein supplements in addition to pancreatic enzymes are key to maintaining/gaining weight for CF post lung transplant recipients.

In the non-CF-related lung transplant population, average weight gain of 10% due to increased body fat mass occurs in the first year post transplant and is associated with better survival.[31] Frequent, nutrient-dense feedings and supplements help with weight gain in both the CF and non-CF post lung transplant recipients. Increased weight is related to improved lung function, less inflammation, less effort to breathe, and more effective immunosuppression. In addition, cachectin levels are reduced when the explanted lungs are removed, decreasing energy expenditure related to the effort of breathing.

Before one particular patient's lung transplant, he was too tired and short of breath to eat more than a few small bites at mealtime. To prevent excess weight loss, he ate five to six small meals throughout the day and found that this habit was still helpful in the first months following his lung transplant. He used liquid high-protein, high-calorie supplements for breakfast because he was too full from taking his morning medications. He ate half of his lunch, then finished the remainder midday, had a small dinner, then another liquid supplement before bedtime. His weight stabilized and his energy and appetite gradually improved over the next several months following transplant surgery.

Underweight is a risk before transplant and leads to poorer outcome/survival post transplant. Once patients have had transplants, those who are able to gain weight survive longer. But, overall, infection is the most common cause of death post lung transplant. In a retrospective cohort study of 826 lung transplant patients done by Singer and colleagues in 2003, those who gained more weight survived longer with infection being a common cause of death.[31]

The incidence of NODPT in the lung transplant population may range from 30% to 35%.[12] Controlling fats and overall calories to maintain a healthy weight is encouraged. A no-added-salt diet can help control hypertension associated with immunosuppressive medications. Supplemental calcium and magnesium to compensate for decreased absorption associated with immunosuppressive medications and vitamin D to support bone health are also advised.[26]

Pancreas
The pancreas is involved in approximately 4% of all transplants.[16] Nationally, 1,200 people wait for pancreas transplantation, but only 287 pancreas transplants were done in 2011.[5] Indications for pancreas transplant may include type 1 diabetes; frequent severe metabolic complications such as hypoglycemia unawareness (the inability to feel warning symptoms of low blood sugar, such as shaking and sweating caused by release of stress hormones), ketoacidosis, or hyperglycemia requiring medical attention; problems with insulin therapy (failure to gain adequate control with insulin therapy or clinical and emotional difficulties with insulin administration); or pancreatectomy due to chronic pancreatitis. Although less common, pancreas transplantation may be considered in patients with type 2 diabetes depending on their levels of C-peptide,[34] an indication of insulin production by the beta cells.

Seventy-five percent of pancreas transplants are performed simultaneously with kidney transplantation as a result of diabetic nephropathy; 15% are performed after a kidney

transplant, and 10% are isolated pancreas transplant.[35] According to the National Kidney Foundation, simultaneous pancreas-kidney transplants occur twice as often as pancreas transplants alone.[36]

One of two surgical techniques is used in a pancreas transplant. Either a donor pancreas is joined or anastomosed to the recipient's bladder, or a donor pancreas is joined to the recipient's segment of duodenum for enteric anastomosis. Pancreas transplant with bladder drainage of exocrine secretions permits ease in monitoring urinary amylase for rejection; a high amylase level is a sign of rejection of the pancreas and is a reliable way to treat rejection early. However, high volume losses of exocrine secretions, fluid, and bicarbonate may lead to dehydration and metabolic acidosis. Pancreas transplant with enteric drainage is more natural or physiologic and allows for exocrine secretions to be reabsorbed in the small bowel, minimizing the need for large fluid and bicarbonate replacements; the author has been previously published on this subject.[34,37]

A BMI lower than 27 kg/m2 before transplant is thought to support early post transplant wound healing. Patients with a BMI higher than 30 kg/m2 have the most significant risk for post transplant technical failure.[1] According to the American Diabetes Association's 2014 Clinical Practice Recommendations, "Evidence suggests that there isn't an ideal percentage of calories from carbohydrate, protein, and fat for all people with diabetes; therefore, macronutrient distribution should be based on individualized assessment of current eating patterns, preferences, and metabolic goals."[38,39]

After transplant, a functioning pancreas may stabilize retinopathy, a complication of diabetes involving damage to the retina of the eye—a leading cause of blindness, according to the National Eye Institute.[34,37] Neuropathy, nerve damage due to diabetes causing pain and/or numbness, may improve, and progression of nephropathy—damage to the kidney from diabetes which leads to kidney failure—may halt.[34]

Glycemic control normalizes within hours of pancreas transplantation.[40] As with other solid organ transplantations, immunosuppressive medications such as corticosteroids, cyclosporine, or tacrolimus, are used, causing similar concerns for long-term effects on cardiovascular and bone health. Weight gain after pancreas transplant is usually less than 1 kg per year.[34] Development of NODPT related to weight gain and immunosuppressive medications may also occur.[37] A diet that's controlled in fats and overall calories to maintain a healthy weight is encouraged. A no-added-salt diet can help control hypertension associated with immunosuppressive medications.[37] Supplemental calcium and magnesium to compensate for decreased absorption associated with immunosuppressive medications and vitamin D to support bone health are also advised.[37]

Common Immunosuppressive Medications and Their Side Effects
The success of solid organ transplantation is largely a result of the discovery and development of immunosuppressive medications such as cyclosporine A in the 1970s, and tacrolimus and poly- and monoclonal antibodies in the 1990s. Poly- and monoclonal antibodies, such as rabbit antithymocyte globulin (ATG), horse ATG (ATGAM), OKT-3 (muromonab-CD3), monoclonal anti-CD25 antibody (daclizumab), and basiliximab are used in combination with other basic

immunosuppressive medications such as corticosteroids, azathioprine, mycophenolate mofetil, and sirolimus to prevent rejection of the transplanted organs.[41-43]

Although immunosuppressive medications are responsible for the increase in successful transplantations, excess immunosuppression increases the risk of infection, neoplasms, and malignancies.[41] Food-drug interactions associated with grapefruit interfere with the body's cytochrome P450 enzyme for drug metabolism and cause increased absorption of CNI, which can result in excessively high levels of CNI in the blood.[42]

Side effects can also cause metabolic syndrome and increase risk of morbidity and mortality after transplant due to hypertension, dyslipidemia, obesity, glucose intolerance, metabolic bone disease, and osteopenia in all transplanted organ populations. Medications, therefore, are adjusted as much as possible to minimize risks while preventing organ rejection. Because transplant medications must be taken for the life of the transplanted organ, changes in diet to include less sodium and animal fat, more whole grains and fiber, and adequate calcium and vitamin-rich foods are necessary to maintain optimum weight and health and help counteract side effects.[41]

Each transplant center uses some combination of the above-mentioned medications, each having different protocols dictating specific doses and duration. Therefore, RDs should be familiar with potential side effects and food-drug interactions of these medications.

There are generally two phases of immunosuppressive therapy for most transplants, induction and maintenance. Both phases rely on the use of CNI, a class of immunosuppressive medications that prevent the immune system from being activated. In the induction phase, a combination of CNI, commonly cyclosporine and/or tacrolimus, corticosteroids, and poly- or monoclonal antibodies, are given in high doses immediately after transplant. These combined medications work together to inhibit the response and proliferation of cytotoxic T cells to interleukin-2 (IL-2), prevent helper T lymphocytes from producing IL-2, suppress circulating lymphocytes and their production, and prevent T and B cell proliferation to prevent rejection in the immediate and early post operative period.[43] (See the medication tip sheet.)

In the maintenance phase, which extends throughout the life of the transplanted organ, the dosages of these medications are decreased to prevent excess immunosuppression and minimize the potential for unwanted side effects while maintaining the best organ and patient survival. CNI, in particular, have a narrow range between efficacy and toxicity and thus require routine monitoring and adjustments. They're known to be toxic to the kidneys in high doses.[41] Polyclonal antibodies such as ATG may be administered with medications stronger than the CNI or intensified maintenance immunosuppression to prevent or treat rejection.

A second stronger level of intensification of immunosuppressive medications may be needed to prevent rejection. This second level is used if the initial combination of immunosuppressive medications fails to prevent rejection or causes unwanted side effects and/or to delay introduction of CNI, especially in patients with already compromised renal function.

ATG decreases circulating lymphocytes. Monoclonal antibodies such as sirolimus, monoclonal anti-CD25 antibody (daclizumab), and monoclonal anti-CD20 antibodies (rituximab) inhibit T and B cell proliferation and also decrease circulating lymphocytes. Although effective in treating early and late rejection, they may result in a higher incidence of opportunistic infection and malignancies because of stronger immunosuppression.[43]

Transplant immunosuppressive medications may also further affect nutrition status as side effects often intensify common nutrition problems associated with end-stage organ disease, as discussed below.

Hypertension and Fluid Retention
CNI can cause acute or chronic nephrotoxicity and, particularly when used in high doses, often can lead to vasoconstriction of renal afferent arterioles, increasing serum creatinine and hyperkalemia.[44]

Corticosteroids overstimulate the mineralocorticoid receptor, resulting in sodium retention and volume expansion, causing hypertension.[45] Patients with preexisting hypertension may develop a worsening of blood pressure control when these drugs are initiated. Dietary sodium restriction and weight management should therefore be encouraged.

Hyperglycemia
Tacrolimus decreases glucose-stimulated release of insulin.[46] Sirolimus impairs insulin-mediated suppression of hepatic glucose production, may cause triglyceride deposition leading to insulin resistance, and may exhibit direct beta cell toxicity.[47] Corticosteroids stimulate gluconeogenesis and inhibit glucose uptake in the muscles, adipose tissue, and peripheral tissues to provide more glucose for glycogen formation in the liver.[47] As a result, hyperglycemia in patients with preexisting type 1 or type 2 diabetes is intensified after transplant by immunosuppressive medications. Any adjustments to immunosuppressive medications, particularly corticosteroids, will require adjustments to insulin or oral hyperglycemic agents in order to maintain optimal glycemic control. For example, lowering corticosteroids without also lowering insulin can result in hypoglycemia.

NODPT is affected by the combination, dose, and duration of immunosuppressive medications, weight gain and obesity associated with insulin resistence, and nonmodifiable risk factors such as family history of diabetes, age, and ethnicity.[12] African Americans, Mexican Americans, American Indians, Native Hawaiians, Pacific Islanders, and Asian Americans have a higher risk of developing diabetes, according to the American Diabetes Association.[48]

When these factors are considered, the incidence of NODPT ranges from 4% to 25% in the kidney transplant population, 2.5% to 25% in the liver transplant population, 4% to 40% in the heart transplant population, and 30% to 35% in the lung transplant population.[12] Because microvascular complications associated with diabetes begin to appear at glycosylated hemoglobin or hemoglobin A1c levels of less than 7%,[11,49,50] modifications of diet, lifestyle, and antihyperglycemic medications should be implemented early.[50] Education about home glucose monitoring and hyperglycemic medications also should be initiated early.[47]

Hyperlipidemia

Cyclosporine has the potential to affect many aspects of lipid and lipoprotein metabolism; however, the exact mechanism(s) by which it does so is still unclear. Sirolimus alters the insulin pathway to increase and/or decrease lipoprotein lipase activity, resulting in increased hepatic synthesis of triglyceride, increased secretion of very low density lipoproteins, and increased hypertriglyceridemia. Large doses of glucocorticoids lead to redistribution of fat to the upper trunk and face, with loss of fat in the extremities. Glucose and triglyceride accumulation occur in response to the rise in insulin levels.[45] Modification of fat quantity and quality, such as low saturated fat, high poly- and monounsaturated fat content, in addition to a low cholesterol diet, is recommended, according to the Heart Association guidelines.[41,50]

Hyperkalemia

CNIs such as cyclosporine may reduce potassium excretion by altering the function of several transporters, decreasing the activity of the renin-angiotensin-aldosterone system, and impairing tubular responsiveness to aldosterone.[51] Modification of dietary potassium may be necessary.

Hypomagnesemia

CNI suppress reabsorption of magnesium from renal tubules and can cause an intracellular shift of magnesium.[51] Magnesium replacement is necessary.

Osteoporosis

All corticosteroids increase excretion of calcium, inhibit bone formation, and suppress intestinal calcium absorption. CNI are also associated with an inhibition of a calcium-binding protein, resulting in a calcium transport defect in the distal tubule of the kidney.[51] Calcium and vitamin D supplementation is recommended, in addition to weight-bearing activities, to help prevent osteoporosis.

Nausea, Vomiting, and Diarrhea

Tacrolimus, azathioprine, mycophenolate mofetil, ATG, ATGAM, OKT3, and sirolimus all have the potential to cause gastrointestinal upset in the form of nausea, vomiting, diarrhea, abdominal pain, and/or flu-like symptoms. Dose adjustments may alleviate symptoms.

Weight Gain and Morbid Obesity

Obesity can influence the entire course of the transplant process. Pretransplant obesity may be related to the disease conditions such as ascites, edema, and fatigue that interfere with physical activity, as well as to poor eating habits. An "obesity paradox" has been identified in some organ transplant patients—those who are obese have better survival before transplant (those on hemodialysis and with congestive heart failure), but experience more complications, higher health care costs, and worse outcomes after transplant, while those who are underweight have poorer survival before transplant due to having less muscle mass and/or unintentional weight loss.[52]

World Health Organization classifications of obesity start at 30 kg/m2 to 34.9 kg/m2 (class I), 35 kg/m2 to 39.9 kg/m2 (class II), and greater than 40 kg/m2 (class III).[53] Common BMI criteria for acceptable weight in transplant candidates is controversial, varies among transplant

centers, and may range from greater than 27 kg/m2 to 40 kg/m2 depending on the organ involved and the patients' other comorbidities.[52]

Most transplant centers advise candidates to implement changes to support gradual weight loss prior to transplant. However, DiCecco and Francisco-Ziller note, "With no potential benefit and opportunity for harm, waiting for patients to lose weight before listing for transplant may not be worth the risk"; particularly if elements of malnutrition, such as muscle wasting (obvious or temporal), cachexia, ongoing decline in protein intake or significant debility, are present.[52]

Some medications associated with weight gain used after transplant, such as corticosteroids, are also used before transplant for autoimmune diseases and inflammatory processes such as lupus and asthma. Corticosteroids are thought to cause weight gain by increasing visceral and truncal fat deposition (central obesity).[45]

After transplant, obesity is associated with delayed graft function, increased wound infections, longer operating room time,[52] and associated increases in health care costs.[1] Post transplant obesity may be related not only to medication-related fat gain, but also to decreased muscle mass.[52] Improved appetite and liberalization of dietary restrictions associated with improved organ function, changes in lifestyle and activity post transplant, and lack of education regarding exercise and nutrition may also account for weight gain.[52]

A comprehensive approach to early weight management after transplant should include calorie control, consistent exercise, and psychological support. Although there's no consensus regarding the role of exercise in transplants, regular daily activity is likely to help maintain a healthier weight and general good health.[54] Ongoing medical nutrition therapy counseling to address obesity is recommended.[49,52]

Wound Healing
Delayed wound healing may increase risk of infection in the transplant recipient.[55] Wound healing can be delayed by malnutrition both before and after transplant. It's also affected by hyperglycemia associated with high-dose steroids, immunosuppression, preexisting diabetes, or NODPT. High-dose steroids affect inflammation and local immune responses needed for proper wound healing, increasing the potential for wound dehiscence, infection, and impaired collagen synthesis.[41] Sirolimus inhibits platelet-derived growth factor and basic fibroblast growth factor that are necessary for tissue healing and repair and also inhibits growth of smooth muscle cells.[56] Hyperglycemia may increase platelet aggregation that damages the endothelium and causes platelet function abnormalities.

Higher BMI increases risks of wound infections that may delay wound healing.[52] For this reason, many transplant centers encourage diet and activity changes to support a lower weight.

Food Safety
The solid organ transplant patient requires lifelong immunosuppressive and antibacterial drugs that maintain transplant organ function but can expose the patient to increased risk of foodborne illness from bacteria, viruses, fungi, and parasites. Many of the primary

immunosuppressive medications interfere with T- and B-lymphocyte activities. This prevents the gut-associated lymphoid tissue from adequately defending against microbial infection and increases susceptibility to foodborne illnesses. Safe food practices include maintaining clean food preparation surfaces, thoroughly washing produce and hands, preventing cross-contamination between the cooked and uncooked foods and the clean and dirty, and maintaining correct time and temperature for cooking and storing foods;[57] the author has been previously published on this subject.

Detailed patient information is available from the USDA Food Safety and Inspection Service.

Putting It Into Practice
Many of the same health risks exist for all adult solid organ transplant recipients. Immunosuppressive medications have improved the success of transplantation as a cure for organ failure. At the same time, side effects associated with transplant immunosuppressive medications can accelerate or exacerbate onset of health risks such as osteoporosis, hypertension, hypercholesterolemia, and weight gain seen in the general population. A heart-healthy approach to eating is strongly encouraged in conjunction with controlled calories and regular daily exercise to maintain optimum weight. The Academy of Nutrition and Dietetics' Nutrition Care Manual contains detailed education materials, tips, and sample menus illustrating these concepts in a patient-friendly format.

RDs play a vital role in communicating the science of nutrition in the context of organ dysfunction and transplantation in a practical approach to support lifelong habits that will sustain organ function and maintain optimum overall health.

—*Patsy Obayashi, MS, RD, CDE, is a specialist in adult kidney, liver, and pancreas transplant nutrition since 1991 and a certified diabetes educator since 2000. She's written and lectured on pancreas transplantation, transplant diabetes, and food safety for the transplant patient.*

Click here *for tip sheet "Common/Traditional Transplant Medications."*

References

1. Lam, HD. Nutritional requirements of adults before transplantation. Medscape. http://emedicine.medscape.com/article/431031. Updated April 7, 2015.

2. Ward HJ. Nutritional and metabolic issues in solid organ transplantation: targets for future research. *J Ren Nutr.* 2009;19(1):111-122.

3. What you need to know about organ transplants. WebMD. http://www.webmd.com/a-to-z-guides/organ-transplants-what-you-need-know. Updated July 12, 2012. Accessed July 5, 2014.

4. About chronic kidney disease. National Kidney Foundation website. www.kidney.org/kidneydisease/aboutckd.cfm. Updated July 13, 2011. Accessed July 5, 2014.

5. Organs and tissues for transplant. Center for Organ Recovery & Education website. http://www.core.org/understanding-donation/organs-tissues-for-transplant/. Accessed March 15, 2015.

6. Centers for Disease Control and Prevention. *National Chronic Kidney Disease Fact Sheet: General Information and National Estimates on Chronic Kidney Disease in the United States, 2014*. Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention; 2014.

7. Organ donation and transplant statistics. National Kidney Foundation website. https://www.kidney.org/news/newsroom/factsheets/Organ-Donation-and-Transplantation-Stats. Updated September 8, 2014. Accessed March 14, 2015.

8. American Dietetic Association. *Chronic Kidney Disease Evidence-Based Nutrition Practice Guideline*. Chicago, IL: American Dietetic Association; 2010.

9. Nix S. *Williams' Basic Nutrition & Diet Therapy*. 14th ed. St. Louis, MO: Elsevier Mosby; 2013.

10. Blaton V. Dyslipidemia at chronic renal failure. *J Int Fed Clin Chem Lab Med.* 2009;20(1):58-65.

11. National Kidney Foundation. KDOQI Clinical Practice Guideline for Diabetes and CDK: 2012 Update. *Am J Kidney Dis*. 2012:60(5):850-886.

12. Pham PT, Pham PM, Pham SV, Pham PA, Pham PC. New onset diabetes after transplantation (NODAT): an overview. *Diabetes Metab Syndr Obes*. 2011;4:175-186.

13. Johnson CP, Gallagher-Lepak S, Zhu YR, et al. Factors influencing weight gain after renal transplantation. *Transplantation*. 1993;56(4):822-827.

14. Blue LS. Adult kidney transplantation. In: Hasse JM, Blue LS, eds. *Comprehensive Guide to Transplant Nutrition*. Chicago, IL: American Dietetic Association; 2002:44-57.

15. Thiem U, Olbramski B, Borchhardt K. Calcidiol deficiency in end-stage organ failure and after solid organ transplantation: status quo. *Nutrients*. 2013;5(7):2352-2371.

16. 2009 Organ Procurement and Transplantation Network and the Scientific Registry of Transplant Recipients Annual Report: Transplant Data 1999-2008. Rockville, MD: US Department of Health and Human Services, Health Resources and Services Administration, Healthcare Systems Bureau, Division of Transplantation; 2009.

17. Education for professionals. Organ Procurement and Transplantation Network website. http://optn.transplant.hrsa.gov. Accessed March 14, 2015.

18. NAFLD. American Liver Foundation website. http://www.liverfoundation.org/abouttheliver/info/nafld/. Updated January 14, 2015. Accessed August 19, 2015.

19. Amodio P, Bemeur C, Butterworth R, et al. The nutritional management of hepatic encephalopathy in patients with cirrhosis: International Society for Hepatic Encephalopathy and Nitrogen Metabolism Consensus. *Hepatology*. 2013;58(1):325-336.

20. Plauth M, Merli M, Kondrup J, et al. ESPEN Guidelines for nutrition in liver disease and transplantation. *Clin Nutr*. 1997;16(2):43-55.

21. Garcia-Compean D, Jaquez-Quintana JO, Gonzalez-Gonzalez JA, Maldonado-Garza H. Liver cirrhosis and diabetes: risk factors, pathophysiology, clinical implications and management. *World J Gastroenterol*. 2009;15(3):280-288.

22. Hasse JM. Adult liver transplantation. In: Hasse JM, Blue LS, eds. *Comprehensive Guide to Transplant Nutrition*. Chicago, IL: American Dietetic Association; 2002:58-89.

23. McClain CJ, Hill SB, Marsano L. Liver disease. In: Erdman JW Jr., Macdonald IA, Zeisel SH, eds. *Present Knowledge in Nutrition*. 10th ed. Ames, IA: Wiley-Blackwell; 2012:889-911.

24. Richards J, Gunson B, Johnson J, Neuberger J. Weight gain and obesity after liver transplantation. *Transpl Int*. 2005;18(4):461-466.

25. What is a heart transplant? National Heart, Lung, and Blood Institute website. http://www.nhlbi.nih.gov/health/health-topics/topics/ht. Updated January 3, 2012. Accessed March 15, 2015.

26. Pahwa N, Hedberg A. Adult heart and lung transplantation. In: Hasse JM, Blue LS, eds. *Comprehensive Guide to Transplant Nutrition*. Chicago, IL: American Dietetic Association; 2002:31-43.

27. Carlson H, Dahlin CM. Managing the effects of cardiac cachexia. *J Hosp Palliat Nurs*. 2014;16(1):15-20.

28. Evidence summaries. Academy of Nutrition and Dietetics: Evidence analysis library. https://www.andeal.org

29. Amarelli C, Buonocore M, Romano G, Maiello C, De Santo LS. Nutritional issues in heart transplant candidates and recipients. *Front Biosci (Elite Ed)*. 2012;4:662-668.

30. OPTN statement regarding lung transplantation and pediatric priority. http://optn.transplant.hrsa.gov/news/optn-statement-regarding-lung-transplantation-and-pediatric-priority/. Published May 27, 2013. Accessed March 15, 2015.

31. Singer LG, Brazelton TF, Doyle RL, Morris RE, Theodore J; International Lung Transplant Database Study Group. Weight gain after lung transplantation. *J Heart Lung Transplant*. 2003;22(8):894-902.

32. Budev, MM, Arroliga AC, Wiedemann HP, Matthay RA. Cor pulmonale: an overview. *Semin Respir Crit Care Med*. 2003;24(3):233-244.

33. Lung transplantation. Cystic Fibrosis Foundation website. https://www.cff.org/Living-with-CF/Treatments-and-Therapies/Lung-Transplantation/. Accessed March 15, 2015.

34. Sutherland DE, Gruessner RW, Dunn DL, et al. Lessons learned from more than 1,000 pancreas transplants at a single institution. *Ann Surg*. 2001;233(4):463-501.

35. Kaufman DB. Pancreas transplantation. http://emedicine.medscape.com/article/429408-overview. Updated June 12, 2013. Accessed March 22, 2015.

36. A to Z health guide. National Kidney Foundation website. https://www.kidney.org/atoz/content/kidneytransnewlease. Updated September 2009. Accessed March 15, 2015.

37. Obayashi, PA. Adult pancreas transplantation. In: Hasse JM, Blue LS, eds. *Comprehensive Guide to Transplant Nutrition*. Chicago, IL: American Dietetic Association; 2002:90-105.

38. Evert AB, Boucher JL, Cypress M, et al. Nutrition therapy recommendations for the management of adults with diabetes. *Diabetes Care*. 2014;37(Suppl 1):S120-S143.

39. American Diabetes Association. Standards of medical care in diabetes — 2014. *Diabetes Care*. 2014;37(Suppl 1):S14-S80.

40. Shokouh-Amiri M, Stratta RJ, Latif KA, Gaber O. Glucose control during and after pancreatic transplantation. *Diabetes Spectr*. 2002;15(1):49-53.

41. DiCecco SR, Francisco-Ziller N, Moore D. Overview and immunosuppression. In: Hasse JM, Blue LS, eds. *Comprehensive Guide to Transplant Nutrition*. Chicago, IL: American Dietetic Association; 2002:1-30.

42. McPartland KJ, Pomposelli JJ. Update on immunosuppressive drugs used in solid-organ transplantation and their nutrition implications. *Nutr Clin Pract*. 2007;22(5):467-473.

43. Mahmud N, Klipa D, Ahsan N. Antibody immunosuppressive therapy in solid-organ transplant: Part I. *MAbs*. 2010;2(2):148-156.

44. Pallet N, Djamali A, Legendre C. Challenges in diagnosing acute calcineurin-inhibitor induced nephrotoxicity: from toxicogenomics to emerging biomarkers. *Pharmacol Res*. 2011;64(1):25-30.

45. McKay LI, Cidlowski JA. Physiologic and pharmacologic effects of corticosteroids. In: Kufe DW, Pollock RD, Weichselbaum RR, et al, eds. *Holland-Frei Cancer Medicine*. 6th ed. Hamilton, Ontario, Canada: BC Decker; 2003.

46. Radu RG, Fujimoto S, Mukai E, et al. Tacrolimus suppresses glucose-induced insulin release from pancreatic islets by reducing glucokinase activity. *Am J Physiol Endocrinol Metab*. 2005;288(2):E365-E371.

47. Crutchlow MF, Bloom RD. Transplant-associated hyperglycemia: a new look at an old problem. *Clin J Am Soc Nephrol*. 2007;2(2):343-355.

48. Chow EA, Foster H, Gonzalez V, McIver L. The disparate impact of diabetes on racial/ethnic minority populations. *Clin Diabetes*. 2012;30(3):130-133.

49. Lucey MR, Terrault N, Ojo L, et al. Long-term management of the successful adult liver transplant: 2012 practice guideline by the American Association for the Study of Liver diseases and the American Society of Transplantation. *Liver Transpl*. 2013;19(1):3-26.

50. The American Heart Association's diet and lifestyle recommendations. American Heart Association website. http://www.heart.org/HEARTORG/GettingHealthy/NutritionCenter/HealthyEating/The-American-Heart-Associations-Diet-and-Lifestyle-Recommendations_UCM_305855_Article.jsp. Updated June 10, 2015. Accessed August 3, 2015.

51. Lee CH, Kim GH. Electrolyte and acid-base disturbances induced by clacineurin inhibitors. *Electroyte Blood Press*. 2007;5(2):126-130.

52. DiCecco SR, Francisco-Ziller N. Obesity and organ transplantation: successes, failures, and opportunities. *Nutr Clin Prac*. 2014;29(2):171-191.

53. BMI classification. World Health Organization website. http://apps.who.int/bmi/index.jsp?introPage=intro_3.html

54. Gordon EJ, Prohaska T, Siminoff LA, Minich PJ, Sehgal AR. Needed: tailored exercise regimens for kidney transplant recipients. *Am J Kidney Dis*. 2005;45(4):769-774.

55. Infected wounds. Wound Care Center website. http://www.woundcarecenters.org/article/wound-types/infected-wounds

56. Kuppahally S, Al-Khaldi A, Weisshaar D, et al. Wound healing complications with de novo sirolimus versus mycophenolate mofetil-based regimen in cardiac transplant recipients. *Am J Transplant*. 2006;6(5 Pt 1):986-992.

57. Obayashi PA. Food safety for the solid organ transplant patient: preventing foodborne illness while on chronic immunosuppressive drugs. *Nutr Clin Pract*. 2012;27(6):758-766.

Examination

1. Mary is a 33-year-old recovering alcoholic who has end-stage liver disease. She complains that she's gaining too much weight. "I'm never hungry so I only eat once a day." Her usual weight last month was 120 pounds, but her current weight in clinic is now 155 pounds. What is likely to be the problem?
A. Mary's anorexia and weight gain are due to early satiety from ascites.
B. Mary is not telling the truth and has been snacking.
C. Mary is pregnant.
D. Mary is gaining muscle mass because she stopped drinking alcohol.

2. John is three months post kidney transplant and has had type 2 diabetes for five years. His blood sugar is 55 mg/dl after his morning clinic appointment. What probably happened?
A. John's insulin dose was not adjusted when his tacrolimus dose was tapered.
B. John ate a large pancake breakfast before coming to clinic.
C. John's new kidney is clearing insulin out of his body at a faster rate.
D. John has an incisional wound abscess.

3. How does cardiac cachexia increase the risk of malnutrition?
A. Accelerated losses of salt and fluid increase blood flow to the gut.
B. The stomach empties faster when edema is present.
C. Gastrointestinal hypomotility may cause nausea, vomiting, and malabsorption.
D. More efficient use of energy is associated with more activity and leads to less time for eating.

4. Tomas is a 25-year-old American Indian who just received a kidney transplant. His dry BMI is 33 kg/m2, and both of his parents have diabetes. He spends his time reading or watching movies. After learning about his cyclosporine and corticosteroids, he expresses concerns about possible side effects such as weight gain and diabetes. What advice should you offer him?
A. Instead of watching movies, start walking 30 minutes every day for more exercise to help with weight control.
B. It's OK to eat whatever you want to build yourself up after transplant; you won't gain weight because you are young.
C. Your risk of developing diabetes after transplant is low because you are a Native American.
D. The transplant medicines you are taking will not cause high blood sugars; your risk of developing diabetes are only related to your family history and obesity.

5. Paul is one month post heart transplant. His blood sugars have been in the mid- to high-200s, his BMI is 22 kg/m2 after he lost 20 pounds since transplant, and he was recently discharged to an extended care facility for rehabilitation after a prolonged hospitalization. His immunosuppression medication was changed from calciuneurin inhibitors (CNI) to sirolimus to prevent renal dysfunction. He has an impacted tooth and needs oral surgery. What factors will affect his wound healing?
A. Paul is already one month beyond surgery, at a better weight, and receiving therapy to recover his strength. He will have no problems with wound healing.
B. Sirolimus, hyperglycemia, weight loss, and weakness from poor nutrition requiring rehabilitation will delay wound healing.
C. The switch from CNI to sirolimus to protect his kidneys will promote faster wound healing.
D. Improved cardiac function, weight loss, and rehabilitation therapy at the extended care center will facilitate accelerated healing.

6. Ben had a kidney transplant six months ago. His renal function is stable and all his blood values are normal according to his nephrologist. What kind of maintenance diet would you recommend to him?
A. Very low protein, very low sodium, low potassium, low phosphorus, fluid restricted to protect the kidney from wearing out
B. Normal protein without restriction, sodium controlled for good blood pressure, and low cholesterol for good heart health
C. High protein, high fat, high calorie for post surgery healing and extra energy for staying active
D. High protein, low potassium, low phosphorus because of immunosuppressive medications

7. Three common types of immunosuppressive medications are cyclosporine, tacrolimus, and corticosteroids. Which of the following are nutrition-related side effects of these medications?
A. Weight gain, osteoporosis, hypertension
B. Constipation, hypoglycemia, rickets
C. Dehydration, hyponatremia, hypermagnesemia
D. Dysphagia, anemia, anorexia

8. Mary is a 30-year-old woman three years post kidney transplant who developed new onset diabetes post transplant one year ago. She attended a family picnic in the late afternoon and ate shish kebabs and potato salad that had been served for lunch earlier in the day. She was the only person at the picnic who developed severe nausea, vomiting, and diarrhea when she got home. What food safety considerations should be observed to minimize foodborne illness for the immunosuppressed patient?
A. Washing hands carefully and maintaining correct temperature for cooking and storing food.
B. Eat food within 15 minutes of cooking and discard any uneaten food that has been sitting out for more than 30 minutes.
C. Make sure all foods are covered or individually wrapped.
D. Only serve commercially prepared foods that come in sealed packages, bottles, or cans.

9. Jim has chronic heart failure and has been waiting for a heart transplant for three weeks. Before he got sick, he weighed 200 pounds and had a BMI of 25 kg/m2. He was athletic and worked out daily. Now, he's had too much fatigue and weakness from the heart failure and sleeps much of the time; he has lost muscle and gained 20 pounds. He eats only small amounts of food two times a day due to lack of appetite and bloating. What would you recommend for medical nutritional therapy to help him prepare for potential transplant?

A. Start total parenteral nutrition, as Jim is not able to meet his needs through oral intake alone.

B. Change to five or six small frequent feedings because of lack of appetite and bloating; provide high-calorie, high-protein foods to help with weight gain; and limit sodium to control fluid retention and blood pressure with heart failure.

C. No changes in current diet or eating habits are necessary. Jim was a little overweight, and weight loss will be better for his heart failure.

D. Encourage at least three feedings daily and offer high-calorie foods such as French fries, deli sandwiches with cheese, pastries, and other flavorful foods to encourage Jim to eat more to gain weight.

10. Jane is 48 and had a pancreas transplant seven years ago due to type 1 diabetes that had been diagnosed when she began grammar school. Her maintenance dose transplant immunosuppressive medications include tacrolimus and corticosteroids. She has gained 35 pounds since the transplant and now has a BMI of 31 kg/m2. Why is she at risk for developing new onset diabetes post transplant (NODPT)?

A. Jane has gained weight, now has a high BMI, and is taking immunosuppressive medications known to cause hyperglycemia.

B. Jane has no increased risk of developing NODPT because she has had a pancreas transplant.

C. Jane's pancreas transplant might wear out after seven years.

D. The transplant immunosuppressive medications prevent rejection of her pancreas and are not associated with any side effect that would increase risk of high blood sugars.