RELATIONSHIP OF DIET QUALITY AND WEIGHT GAIN POST-RENAL TRANSPLANTATION

A THESIS

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ABSTRACT

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The aim of this secondary data analysis was to examine the relationship of dietary quality and weight gain in post-renal transplant recipients from pre-transplant baseline through post-transplant at 3 months and 1 year. Body weight, body mass index, intake of carbohydrates, protein, fat, phosphorus, potassium, sodium, calcium, and Healthy Eating Index scores of patients were used to assess changes in dietary intake, dietary quality, and weight. Findings of this study indicated that weight gain had a strong positive correlation with fat intake. Body weight, body mass index, potassium, and fat intake significantly increased from pre-transplant baseline through post-transplant at 3 months and 1 year. In contrast, weight gain was not associated with dietary quality as assessed by the Healthy Eating Index. More research is needed in this area to identify dietary recommendations for the prevention of undesirable weight gain and potential health complications seen in patients following renal transplantation.

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LIST OF ABBREVIATIONS

AMPM	Automated Multiple Pass Model
ACE	Angiotensin Converting Enzyme
ARB	Angiotensin II Receptor Blocker
ASA24	Automated Self-Administered 24-Hour
BMI	Body Mass Index
CKD	Chronic Kidney Disease
CVD	Cardiovascular Disease
DGA	Dietary Guidelines for Americans
DNA	Deoxyribonucleic Acid
DSD	Dietary Supplements Database
ESRD	End Stage Renal Disease
FGF-23	Fibroblast Growth Factor-23
FNDDS	Food and Nutrient Database for Dietary Studies
FPED	Food Pyramid Equivalents
GFR	Glomerular Filtration Rate
HEI	Healthy Eating Index
HLA	Human Leukocyte Antigens
KDIGO	Kidney Disease Improving Global Outcome
KDOQI	Kidney Disease Outcomes Quality Initiative

MBD	Mineral and Bone Disorder
MNT	Medical Nutrition Therapy
NCI	National Cancer Institute
NHANES	National Health and Nutrition Examination Survey
NKF	National Kidney Foundation
PEM	Protein-Energy Malnutrition
PTH	Parathyroid Hormone
RAAS	Renin-Angiotensin-Aldosterone System
RBC	Red Blood Cell
RDN	Registered Dietitian Nutritionist
RRT	Renal Replacement Therapy
SAS	Statistical Analysis Software
SGA	Subjective Global Assessment
SHPT	Secondary Hyperparathyroidism
TDS	Total Diet Score
US	United States
USDA	Unites States Department of Agriculture
USRDS	United States Renal Data System

CHAPTER I

Literature Review

Introduction

Although kidney transplantation is accepted as the most favorable option of renal replacement therapy, transplant recipients are at heightened risk for health complications related to chronic kidney disease and chronic immunosuppression. It has been suggested that part of the increase in risk may be due to excessive weight gained following renal transplantation. The rationale behind excessive weight gain in transplant recipients is still unclear. Little research has been performed to evaluate the relationship of diet quality and weight gain in patients following renal transplantation.

The Kidneys: Structure and Function

The kidneys are two bean-shaped organs located below the rib cage and on each side of the spine. The primary function of the kidneys is the filtration of blood through the removal of waste and extra fluids. Located inside each kidney are a million filtering units called nephrons. Each nephron contains a glomerulus and tubule that help filter blood. The glomerulus is a cluster of blood vessels that allows certain substances to pass into the tubule. Once these substances reach the tubule, the nutrients the body needs are reabsorbed into the bloodstream that runs alongside the tubule. In addition to filtering blood, the kidneys play an important role in regulating phosphorus, potassium, sodium, and calcium concentrations, controlling blood pressure through the renin-angiotensin-aldosterone system, producing red blood cells, and regulating bone development and metabolism (National Institute of Diabetes and Digestive and Kidney Diseases, 2017).

Kidney function can be measured using the glomerular filtration rate (GFR) or albuminuria levels (Levey, Becker, & Inker, 2015). The GFR is a measurement of the amount of blood that flows through the glomeruli every minute (MedlinePlus, 2017b). The GFR can be measured directly, but it is cumbersome (24-hour urine collection) or expensive (clearance of labeled isotopes). GFR can be estimated using the results from a serum creatinine test in conjunction with equations commonly used in estimating GFR, such as the 2009 Chronic Kidney Disease Epidemiology Collaboration equation or The Modification of Diet in Renal Disease Study equation. In a healthy adult, the average glomerular filtration rate is between 120 and 130 mL/min/1.73 m^2 (Levey et al., 2015). Increasing age, disease, and other factors can cause changes to the GFR. A GFR below 60 mL/min/1.73 m^2 may suggest chronic kidney disease (Levey et al., 2015).

One method of assessing kidney damage is measuring albuminuria levels. Albumin is a protein found in blood. It is responsible for regulating fluid balance, building muscles, and repairing tissues in the body (National Kidney Foundation, 2016). Healthy kidneys do not allow albumin to pass into the urine (National Institute of Diabetes and Digestive and Kidney Diseases, 2016). When high levels of albumin are found in the urine, kidney damage is suspect (Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group, 2013). Albuminuria levels can be determined by measuring the ratio between albumin and creatinine in a spot urine sample, the preferred method to detect elevated protein (Levey et al., 2015). Normal albuminuria levels are below 30 mg/g (National Kidney Foundation, 2016).

Chronic Kidney Disease

Chronic kidney disease (CKD) is defined as "abnormalities of kidney structure or function for greater than three months with implications for health" (Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group, 2013). CKD is a progressive and irreversible condition. When the kidneys are damaged or lose function, they are unable to filter blood as before, leading to waste and fluid buildup. CKD affects an estimated 15% of the United States (US) population with the majority being women (Centers for Disease Control and Prevention, 2017). The primary causes of CKD are diabetes and high blood pressure (Nazar, 2013). Other causes may include increasing age, cardiovascular disease (CVD), obesity, and a family history of kidney failure (McManus et al., 2017).

The diagnosis criteria for CKD are kidney damage or a GFR of less than 60 mL/min/1.73 m^2 for longer than three months (Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group, 2013). The tests used to diagnose kidney disease are the GFR to estimate kidney function and albuminuria levels to assess kidney damage as previously discussed. The symptoms commonly experienced in CKD patients are loss of appetite due to uremia, nausea, fatigue, and changes in urine volume (Beto, Ramirez, & Bansal, 2014). Frequently, patients are not diagnosed with CKD until the later stages because these symptoms are often overlooked.

In 2002, the National Kidney Foundation (NKF) developed the Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines to help healthcare professionals recognize the stages and treatment of CKD to improve care (National Kidney

Foundation, 2002). The KDOQI guidelines are used to classify disease progression. According to the KDOQI classification system, CKD is divided into five stages based on kidney function, characterized as GFR levels (see Table 1).

Table 1

Stages of Chronic Kidney Disease

Stage	**Description**	**GFR (mL/min/1.73 m^2)**
1	Normal kidney function	≥ 90
2	Mildly decreased kidney function	60 – 89
3	Moderately decreased kidney function	30 – 59
4	Severely decreased kidney function	15 – 29
5	Kidney failure	< 15

Note. From "K/DOQI clinical practice guidelines for chronic kidney disease: Evaluation, classification and stratification," by National Kidney Foundation, 2002, *American Journal of Kidney Diseases*, 39, S1 – S266.

However, in 2012, the Kidney Disease Improving Global Outcomes (KDIGO) organization, which includes an expert panel of kidney practitioners, updated the original 2002 NKF-KDOQI guidelines. This update was implemented to provide advanced guidance on the evaluation, management, and treatment for all patients with CKD (National Kidney Foundation, 2017a). The three primary changes were dividing the third stage of GFR into two categories (G3a and G3b) (see Table 2) and adding albuminuria categories and the cause of CKD; for example, high blood pressure or diabetes (see Table 3), These changes were incorporated into the classification of CKD (Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group, 2013; McManus & Wynter-Minott, 2017).

Table 2

Glomerular Filtration Rate Categories in Chronic Kidney Disease

GFR Category	**GFR (mL/min/1.73 m^2)**	**Description**
G1	≥ 90	Normal or high
G2	60 – 89	Mildly decreased
G3a	45 – 59	Mildly to moderately decreased
G3b	30 – 44	Moderately to severely decreased
G4	15 – 29	Severely decreased
G5	< 15	Kidney failure

Note. From "KDIGO 2012 clinical practice guideline for the evaluation and management of chronic kidney disease," by Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group, 2013, *Kidney International Supplements*, 3, 1–150.

Table 3

Albuminuria Categories in Chronic Kidney Disease

Category	**AER** (mg/24 hours)	**ACR** (mg/mmol)	**ACR** (mg/g)	**Description**
A1	< 30	< 3	< 30	Normal to mildly increased
A2	30 – 300	3 – 30	30 – 300	Moderately increased
A3	> 300	> 30	> 300	Severely increased

Note. AER, albumin excretion rate; ACR, albumin to creatinine ratio

From "KDIGO 2012 clinical practice guideline for the evaluation and management of chronic kidney disease," by Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group, 2013, *Kidney International Supplements*, 3, 1–150.

As the disease progresses, the kidneys gradually decline in function and eventually reach Stage 5 of CKD, also known as kidney failure or end-stage renal disease (ESRD). In this stage, the kidneys are only working 10-15% of their normal capacity, which is not enough for patients to survive without treatment (National Kidney Foundation, 2015). As a result, other serious health complications may emerge, including electrolyte imbalances, cardiovascular disease, hyperlipidemia, anemia, and metabolic bone disease (Thomas et al., 2008). Optimal management of ESRD and its complications

requires close collaboration of a medical team and is dependent on the patient's diseased state.

Treatment of End-Stage Renal Disease

Unfortunately, there is no cure to CKD. The treatment modality that has been deemed effective in delaying disease progression is renal replacement therapy. Renal replacement therapy (RRT) is required in ESRD patients to enhance survival by replacing the work of the damaged kidneys for proper bodily functions. Based on the estimates of the United States Renal Data System (USRDS), the initiation of RRT occurs in a majority of patients when their GFR is between 4 and 8 mL/min/1.73 m^2 (see Figure 1) (National Kidney Foundation, 2002). The RRT options are dialysis and kidney transplantation.

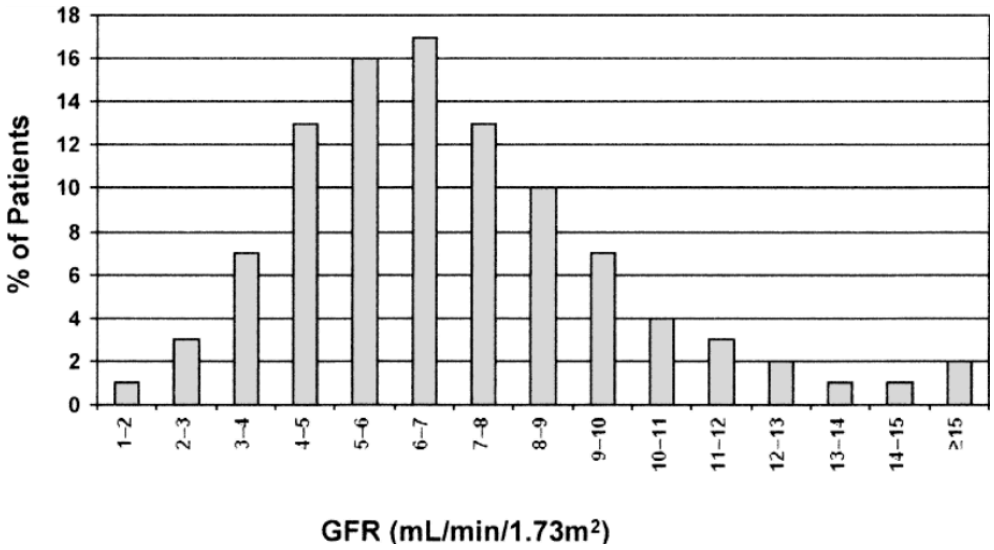


Figure 1. Initiation of Renal Replacement Therapy by Glomerular Filtration Rate. Adapted from "K/DOQI clinical practice guidelines for chronic kidney disease: Evaluation, classification and stratification," by the National Kidney Foundation, 2002, *American Journal of Kidney Diseases*, *39*, S1 – S266. Copyright 2002 by the National Kidney Foundation.

Dialysis. Dialysis is the process of removing waste and excess fluids from the blood, similar to the actions of the kidneys. ESRD patients may need dialysis treatment for the rest of their life unless they receive a kidney transplant (National Kidney Foundation, 2017b). The two types of dialysis are peritoneal dialysis and hemodialysis.

Peritoneal dialysis. Peritoneal dialysis uses the peritoneum, which is a membrane that lines the abdomen, to filter blood (National Institute of Diabetes and Digestive and Kidney Diseases, 2018a). This is accomplished by filling and emptying the peritoneum of a dialysis solution through a catheter that is surgically placed into the patient's abdomen. Throughout the day, the dialysis solution absorbs waste and fluids that is then emptied out of the body. Peritoneal dialysis can be performed at home or in any sterile environment.

Hemodialysis. Hemodialysis filters the blood by using a dialysis machine. The blood from a patient's vascular access flows through tubes connected to the dialysis machine where it is filtered through a dialyzer and then returned to the body (National Institute of Diabetes and Digestive and Kidney Diseases, 2018b). Hemodialysis can be performed at a dialysis center or at home. In general, patients receiving hemodialysis treatment visit a dialysis center three times per week. Dialysis is an effective treatment for those with ESRD. However, dialysis alone is not enough. Diet, fluid restrictions, and medications also play an important role in managing ESRD.

Kidney Transplant. Kidney transplantation is the treatment of choice for ESRD considering quality of life and survival rate when compared to a lifetime on dialysis (Gupta, Unruh, Nolin, & Hasley, 2010). The first successful kidney transplant was

performed in 1954 by Joeseph Murray and his colleagues at the Peter Bent Brigham Hospital (Kasiske et al., 2010). This procedure requires a healthy kidney that is placed in the body to compensate for the work of the damaged kidneys. It can be performed using a living or deceased donor. A living donor may be a family member or friend who gives their consent to donate their kidney. A deceased donor is someone who recently died but has allowed his or her kidneys to be donated. However, not everyone is able to receive a kidney transplant (Davis & Delmonico, 2005). Reasons for exclusion include:

- Current substance abuse
- Active systemic infection
- Uncorrectable CVD
- Current neurological impairment
- History of metastatic cancer or receiving ongoing chemotherapy

Waiting list. Once a candidate is accepted for a transplant, they are placed on the waiting list. Currently, over 90,000 people in the US are on the waiting list for a kidney transplant (United Network for Organ Sharing, 2018). They may wait months to years for a kidney transplant. If a patient already has transplant arrangements from a living donor, the surgery can be performed sooner (Davis & Delmonico, 2005).

Match process. Before kidney transplantation, blood tests are performed to determine the compatibility between the donor and recipient. This procedure helps match the donor kidney to a recipient to prevent organ rejection. The tests used are blood typing and tissue typing. To test the blood type, a blood sample is taken from the donor and

recipient. The four different blood types are A, B, AB, and O. The donor and recipient blood types must be compatible to perform the transplantation (see Table 4) (National Kidney Foundation, 2017c). Tissue typing, also called human leukocyte antigens typing (HLA), is a procedure that tests for antigens in the blood. These are found on cell surfaces and are unique to the individual. HLAs elicit an immune response when foreign substances are detected in the body. The kidneys are expected to last longer when the antigens are a perfect match for the donor and recipient (Berger, 2001).

Table 4

Blood Type Compatibility for Kidney Transplantation

Recipient Blood Type	**Donor Blood Type**
A	A or O
B	B or O
AB	A, B, AB, or O
O	O

Note. Adapted from *National Kidney Foundation*, 2017c, Retrieved June 5, 2018 from https://www.kidney.org/transplantation/livingdonors/what-blood-types-match.

Kidney transplant procedure. When patients undergo kidney transplantation, they work with a team of medical professionals. This team may include the surgeon, nephrologist, registered dietitian nutritionist (RDN), social worker, and pharmacist (National Institute of Diabetes and Digestive and Kidney Diseases, 2018c). A kidney transplant procedure usually takes two to four hours to complete. Under general anesthesia, the donor kidney is placed into the patient's pelvis. Then, the artery that carries blood to the donor kidney and the vein that carries blood away from the donor kidney are connected to the artery and vein of the pelvis. Lastly, the ureter is connected to

the bladder (see Figure 2; “Example of a Kidney Transplant.” n.d.). The patient’s inborn kidneys are not removed during surgery unless contraindicated (National Institute of Diabetes and Digestive and Kidney Diseases, 2018c).

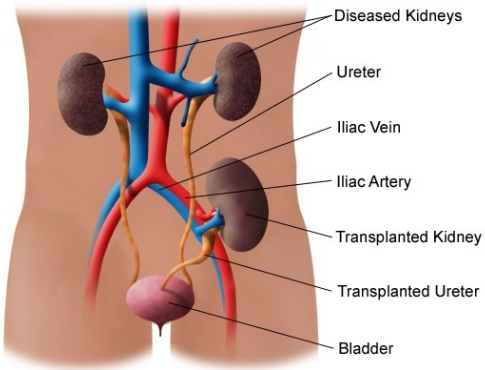


Figure 2. Anatomy of a Kidney Transplant

Management of organ rejection. Although there have been improvements in post-transplant care and graft survival, 7% of transplant recipients still experience organ rejection within the first year of receiving a kidney (Tushla, 2017). The three phases of organ rejection are hyperacute, acute, and chronic rejection. Hyperacute rejection occurs a few minutes after transplantation and is normally seen in patients who received a kidney with the wrong blood type. Acute rejections occur from one week to three months after kidney transplantation. In chronic rejection, the body is continually fighting and damaging the new kidney years after the procedure (MedlinePlus, 2017c). The prevention and treatment of organ rejection requires immune system suppression. Immunosuppression can be achieved by blocking lymphocyte activity. These are the cells of the immune system responsible for fighting infections (Halloran, 2004). To

accomplish this, immunosuppressive medications are prescribed to patients for lifelong suppression of the immune system.

The KDIGO guidelines recommend immunosuppressive medications, which include a combination of "calcineurin inhibitors and an antiproliferative agent with or without corticosteroids," during the acute phase after transplantation (Kasiske et al., 2010). The calcineurin inhibitors commonly prescribed are Tacrolimus and Cyclosporine. These drugs block T-cell proliferation by inhibiting calcineurin, which is a phosphatase protein that activates the T-cells. Some antiproliferative agents prescribed are Mycophenolate and Azathioprine (Kalluri & Hardinger, 2012). Antiproliferative agents are used to inhibit the proliferation of T- and B-cells of the immune system. The last class of drugs prescribed to patients after a kidney transplant is corticosteroids, particularly prednisone. Prednisone is a glucocorticoid and is one of the first kinds of drugs used to prevent rejection after organ transplantation. They are needed to reduce inflammation to prevent tissue damage (see Table 5) (Steiner & Awdishu, 2011).

The dosage of these medications is dependent on how patients respond to their new kidney or if they experience major side effects. Normally the dosage is higher in the acute phase because the initial post-transplant period is most sensitive to rejection. After patients are cleared of acute graft rejection, the dosage of immunosuppressive medications is reduced, typically between two and four months after transplantation for long-term maintenance immunosuppression (Kasiske et al., 2010).

Table 5

Immunosuppressive Medications in Kidney Transplantation

Medications	**Mode of Action**	**Side Effects**
Calcineurin Inhibitors Tacrolimus Cyclosporine	Inhibits activation of T-lymphocytes of the immune system	Nephrotoxicity Elevated blood sugar Elevated blood pressure Elevated cholesterol
Antiproliferative Agents Mycophenolate Azathioprine	Interrupts DNA replication	Decreased blood count Upset stomach
Corticosteroid Prednisone	Suppresses the immune system and treats inflammation	Weight gain Osteoporosis Elevated blood sugar Elevated blood pressure Elevated cholesterol

Note. From Kasiske, 2010; Steiner, 2011; Kalluri & Hardinger, 2012.

The success of organ transplantation is largely attributed to the use of immunosuppressive therapy (Taylor, Watson, & Bradley, 2005). However, some of the consequences from prolonged exposure to immunosuppressive therapy are weight gain, elevated lipid levels, bone loss, and nephrotoxicity. The comorbidities linked to immunosuppressant therapy are a major cause of dosage reductions or treatment cessations, which can have an unfavorable effect on the long-term outcome of transplant recipients (Pasha et al., 2017).

Risk of infection. Along with the complication of organ rejection, transplant recipients have an increased risk for infection due to immunosuppression. About 70% of renal transplant recipients experience an infection after three years (Jha, 2010). Patients may acquire an infection in a number of ways, whether from being exposed to viruses, bacteria, parasites, or food borne pathogens (Fishman, 2017). Contracting an infection

may lead to graft loss, prolonged illness, hospitalization, or even death. To reduce infectious episodes, patients are recommended to take special precautions.

Complications in End-Stage Renal Disease

Although patients receive renal replacement therapy to treat CKD, there are health complications that often persist. These include uremia, electrolyte imbalances, anemia, bone disorders, weight gain, malnutrition, and cardiovascular disease. RRT prevents the progression of the disease but does not improve GFR, explaining the recurrence of these problems. To promote better outcomes in these patients, alleviation of the underlying problems is imperative in managing this challenging and multifaceted condition.

Uremia. Uremia is a condition that occurs in worsening renal function. Uremia means "urine in blood" and is characterized as fluid, electrolyte, and hormone imbalances (Foris & Bhimji, 2018). In renal failure, the impaired kidneys do not sufficiently filter waste and fluid from the blood, leading to a buildup of toxins. This condition can be life-threatening if untreated because it can cause loss of consciousness, seizures, cardiac arrest, or death. The symptoms experienced from uremia are decreased appetite, nausea, vomiting, and fatigue. Normally patients with uremia are treated with dialysis to remove the toxins in the blood.

Electrolyte imbalances. The kidneys play a pivotal role in maintaining electrolyte balance. In ESRD patients, there is progressive loss of kidney function causing derangements in electrolyte homeostasis (Dhondup & Qian, 2017). The electrolytes primarily monitored in these patients are phosphorus, calcium, potassium, and sodium. Serum levels outside of the normal range of these electrolytes can be life

threatening and speed the disease process. The medical and nutritional management of these electrolytes is extremely important.

Phosphorus. Phosphorus is essential in the diet for bone and teeth formation as well as other bodily functions; however, excessive phosphorus consumption may be detrimental, especially in ESRD patients. Due to their limited kidney function, the regulation of phosphorus excretion is compromised leading to elevated phosphorus in the blood, also known as hyperphosphatemia. Recurrent hyperphosphatemia is associated with vascular stiffening and calcification, secondary hyperparathyroidism, and bone disorders (Kalantar-Zadeh, 2013). Hence, patient's serum phosphorous levels should be under control, which can be achieved through the combination of diet, dialysis, and medications.

Phosphorus status in ESRD patients is assessed using serum phosphorus levels. The 2003 NKF KDOQI clinical practice guidelines recommend lab value goals for serum phosphorus to be between 3.5 and 5.5 mg/dL (London et al., 2010). Nearly half of all dialysis patients surpass the upper range of 5.5 mg/dL. This may be influenced by insufficient phosphorus removal during dialysis treatment, limited adherence to a low phosphorus diet, and medications (Suki & Moore, 2016). On average, only 800 to 1,000 mg of phosphorus is removed during a hemodialysis session. This amount of phosphorus removal does not account for the phosphorus consumed in the remaining days of the week that patients do not receive dialysis treatment (Suki & Moore, 2016). To compensate, patients are encouraged to follow a low phosphorus diet.

Phosphorus is found in almost every food group, especially protein. Some examples include meats, dairy products, dark cola, nuts, and processed foods. Moreover, phosphorus exists as organic or inorganic. Organic phosphorus comes from plant or animal sources while inorganic phosphorus is in the form of additives. Phosphorus bioavailability is greater in phosphate additives, with up to 100%, compared to organic phosphorus where only 40 – 60% is absorbed in a mixed meal (Moore, Nolte, Gaber, & Suki, 2015). This suggests that good dietary quality may be essential in serum phosphorus control. In addition, food and nutrient databases do not always contain accurate information about phosphorus content in foods making it difficult to estimate phosphorus consumption (Moore et al., 2015). By emphasizing the importance of avoiding or limiting food sources of phosphorous to patients, hyperphosphatemia can be prevented.

In patients with constantly elevated serum phosphorus, diet and dialysis may not be sufficient to maintain phosphorus levels in the recommended range. Phosphate binders are occasionally administered for additional serum phosphorus control. When taken with meals, phosphate binders soak up phosphorus in food and eliminate it in the stool to prevent its absorption in the blood (Kalantar-Zadeh, 2013). The dosage of the phosphate binder is adjusted accordingly depending on lab values and the phosphate content in patient meals and snacks (Academy of Nutrition and Dietetics, 2010). Phosphate binders come in different forms: pill, powder, chewable tablet, and liquid. They are either too large to swallow, have an undesirable taste, or cause an upset stomach for some patients. Considering dialysis patients are already taking many medications, phosphate binders

contribute to their pill burden, which may result in non-adherence and hyperphosphatemia (Suki & Moore, 2016).

Calcium. It is known that kidney failure disrupts the homeostatic mechanism that controls serum calcium and normal bone metabolism (Gallant & Spiegel, 2017). Calcium's primary function is maintaining healthy bones and teeth. Calcium is also needed for muscle contraction and normal heart rhythm (MedlinePlus, 2017a). When calcium balance is disrupted in ESRD patients, this can cause major health issues. Negative calcium balance, or hypocalcemia, may cause osteoporosis and poor bone health. Positive calcium balance, or hypercalcemia, may cause soft tissue and vascular calcification and cardiovascular events (Gallant & Spiegel, 2017). To prevent these problems, patients are recommended to stay in the normal serum calcium range of 8.4 to 9.5 mg/dL (National Kidney Foundation, 2003).

The two hormones responsible for regulating calcium metabolism are parathyroid hormone and calcitriol. When serum calcium levels are low, these hormones are synthesized and secreted to increase calcium levels in the blood (Gallant & Spiegel, 2017). Sometimes these processes are disrupted in ESRD patients due to the underlying damage of kidney disease progression or other comorbid conditions. When this occurs, hypocalcemia or hypercalcemia may develop. As a result of these disruptions, ESRD patients may find it challenging to maintain calcium homeostasis. The primary approaches in treating calcium imbalances in the blood are increasing calcium through diet or supplementation if levels are low, dialysis, and managing the underlying causes.

Potassium. Potassium is an electrolyte needed for normal body growth, controlling the electrical signals in the heart, acid-base balance, and building protein. The kidneys maintain potassium homeostasis by matching potassium intake with potassium excretion. When potassium homeostasis is disrupted from poor kidney function, an increase in serum potassium levels or hyperkalemia is often observed (Hsieh et al., 2011). Hyperkalemia, seen in about 5 to 10% of dialysis patients, is associated with greater mortality because of its cardiac arrhythmic effect (Nakhoul et al., 2015; Korgaoankar et al., 2010).

Dietary potassium restriction is recommended in patients with hyperkalemia especially in the advanced stages of CKD (Kalantar-Zadeh & Fouque, 2017). Potassium is essentially found in fruits and vegetables but also in dairy, meats, and fish (MedlinePlus, 2016a). Fresh fruits and vegetables are considered healthy choices because of the fiber and vitamin content. However, the excessive consumption of these foods can increase potassium levels that can progress kidney disease (Kalantar-Zadeh & Fouque, 2017). To minimize the risk for arrhythmias, patients should maintain serum potassium levels between 3.5 and 5.5 mEq/L (Academy of Nutrition and Dietetics, 2010).

In addition to diet, other factors that may contribute to hyperkalemia are medications inhibiting potassium excretion or metabolic acidosis (Kovesdy, 2014). Under these circumstances, these underlying disturbances should be addressed to manage hyperkalemia.

Sodium. Sodium is naturally occurring in most foods and is commonly found as sodium chloride, or salt. It is necessary for controlling blood pressure and muscle and

nerve function (MedlinePlus, 2016b). In the general population, the Dietary Guidelines for Americans (DGA) recommend limiting sodium to 2,300 mg per day (Wright & Cavanaugh, 2010). Despite these guidelines, sodium intake in the US population remains well over the recommendations. For ESRD patients, sodium intake may be restricted further because their damaged kidneys are unable to excrete excess dietary sodium. Sodium restriction aids in the prevention of fluid retention and elevated blood pressure. As more sodium is consumed, the greater the thirst receptors are stimulated causing increased water intake, fluid retention, and blood pressure. In hemodialysis patients, major fluctuations in interdialytic weight gain strain the cardiovascular system through elevated blood pressure and extracellular volume expansion (Clark-Cutaia et al., 2014). In addition, sodium accumulation is associated with cardiovascular morbidity and mortality and increases the risk for hypertension, characterized as constantly elevated blood pressure, which is a potential indirect mechanism for CKD progression (Wright & Cavanaugh, 2010).

An important goal in reducing disease progression is blood pressure management. KDOQI recommends blood pressure goals for CKD patients to be <130/85, <125/75 with proteinuria, and <130/85 in diabetics (Thomas, Kanso, & Sedor, 2008). In order to reach these levels, patients may need a sodium-restricted diet along with multiple antihypertensive medications such as angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs), the first line therapies for hypertension (Tan & Johnson, 2008).

It has been identified that patients who restricted their sodium intake had a reduction in blood pressure (Wright & Cavanaugh, 2010). Reducing sodium intake has many benefits including maximizing the results of other therapies in CKD patients. However, there is poor adherence to a low sodium diet in this population. This may be due to their inability to understand food labels, taste preferences, poor food choices such as fast food, convenience, or processed foods, or the use of table salt at meals.

In addition to diet, blood pressure can be reduced with antihypertensive medications such as ACE inhibitors and ARBs. Blood pressure control is regulated by the renin-angiotensin-aldosterone system (RAAS). This system is composed of renin, angiotensin I and II, and aldosterone which all work together to regulate blood pressure. When low blood pressure is detected, the RAAS activity is stimulated to increase blood pressure. In CKD, the mechanism behind hypertension is sodium accumulation and overstimulation of the RAAS. To manage hypertension, ACE inhibitors and ARBs are prescribed to patients to decrease blood pressure (Tan & Johnson, 2008; Hudaifa, Tamimi, & Tamimi, 2014).

Hypertension management to promote positive outcomes in ESRD patients is primarily attributed to patient compliance to a sodium and fluid-restricted diet, dialysis treatment, and medications. The multidisciplinary team should increase education and awareness in these patients to improve adherence to prevent the complications of hypertension and disease progression.

Mineral and bone disorders. Current research has identified disorders of bone and mineral metabolism and vascular calcification as risk factors for cardiovascular

mortality and morbidity in patients with CKD. Chronic kidney disease-mineral and bone disorder (CKD-MBD), also referred to as renal osteodystrophy, is described as "a triad of interrelated abnormalities of serum biochemistry, bone, and the vasculature associated with CKD" (London et al., 2010). This disorder occurs when kidney function is impaired causing an imbalance in hormones and minerals. The alterations of CKD-MBD include elevated fibroblast growth factor-23, parathyroid hormone, and serum phosphorus and decreased calcitriol and serum calcium (Gallant & Spiegel, 2017; Mac Way, Lessard, & Lafage-Proust, 2012). These hormones and minerals help keep bones strong. When they are out of balance as a result of kidney failure, bone disease and excessive vascular and soft tissue calcification can occur (Gallant & Spiegel, 2017).

Parathyroid hormone. As the glomerular filtration rate continues to fall below 60 mL/min/1.73 m^2 in CKD patients, the prevalence of hyperphosphatemia, hypocalcemia, and secondary hyperparathyroidism increases rapidly (Ross, Caballero, Cousins, Tucker, & Ziegler, 2012). As previous mentioned, many factors could cause calcium imbalances in these patients. One factor that may explain hypocalcemia is the chemical bond between calcium and phosphorus. Calcium, a divalent cation, has a high binding affinity for phosphate, a monovalent anion (Tomasello, 2008). When phosphate is elevated in the blood, calcium binds to it causing calcium levels to drop. In response to hypocalcemia and hyperphosphatemia, the parathyroid glands secrete parathyroid hormone (PTH) (Saliba & El-Haddad, 2009). Secretion of PTH leads to:

- Stimulation of the osteoclasts in the bones which causes bone resorption to increase serum calcium and phosphorus levels

- Increased stimulation of 1-α hydroxylase activity in the kidney to increase $1,25(OH)_2$ vitamin D3 production
- Increased reabsorption of calcium in the distal renal tubules
- Decreased phosphate reabsorption and increased phosphorus excretion
- Indirect stimulation of intestinal calcium and phosphorus absorption via $1,25(OH)_2$ vitamin D3 production (Saliba & El-Haddad, 2009)

Although PTH production helps maintain calcium and phosphorus in the normal ranges, the trade-off is a gradual increase in PTH levels over time as GFR levels continue to drop. Constantly elevated PTH leads to secondary hyperparathyroidism (SHPT), which is the leading cause of renal osteodystrophy. The most common osteodystrophy is osteitis fibrosa cystica and is caused by high bone turnover from the excessive circulation of PTH (Saliba & El-Haddad, 2009). Some patients may experience poor mobility, bone and joint pain, and bone deformities or fractures.

Hypercalcemia is another problem that may result from hyperparathyroidism (Saliba & El-Haddad, 2009). This may be due to accelerated bone resorption, increased gastrointestinal absorption, or reduced excretion of calcium as a consequence of elevated PTH circulation. Hypercalcemia can lead to vascular calcification, calcium deposits in the kidneys causing poor kidney function, and high blood pressure. The preferred approach in correcting elevated serum calcium levels is through the management of PTH levels. Other treatment may include medications and dialysis (MedlinePlus, 2018).

It is important that SHPT be identified earlier on to prevent additional complications through the continuous assessment of serum calcium, phosphorus, vitamin D, and PTH levels. The ultimate goal of treating SHPT is to improve bone and mineral metabolism in CKD and reduce its associated complications (Tomasello, 2008).

Fibroblast growth factor-23. Fibroblast growth factor-23 (FGF-23) is produced by osteocytes and osteoblasts and is responsible for the regulation of phosphorus concentration in the body (Mac Way et al., 2012). It maintains serum phosphorus levels by inhibiting the intestinal absorption of phosphorus and the reabsorption of phosphorus through the proximal renal tubules (Takashi & Fukumoto, 2016). FGF-23 is normally increased in the beginning stages of CKD to prevent hyperphosphatemia and continues to increase as the GFR decreases. In the late stages of CKD, the remaining working nephrons are unable to sufficiently excrete phosphorus (Tomasello, 2008). Although FGF-23 is continually secreted, its actions are impaired. As a consequence, elevated FGF-23 and phosphate are often observed. Elevated FGF-23 is now recognized as the earliest detector for CKD-MBD and is associated with cardiovascular risk, left ventricular hypertrophy, vascular calcification, and accelerated progression of the disease process in CKD patients (Hruska, Seifert, & Sugatani, 2015; Kalantar-Zadeh & Fouque, 2017).

Vitamin D. Vitamin D is a hormone also important in calcium and phosphorus homeostasis. It is needed for bone resorption, the reabsorption of calcium and phosphorus in the renal tubules, and PTH synthesis and release (Saliba & El-Haddad, 2009; Tomasello, 2008). When calcium levels are low, PTH stimulates the synthesis of the enzyme, 1 α-hydroxylase, to produce the active form of vitamin D, $1,25(OH)_2$ or

calcitriol. Calcitriol is necessary to increase the availability of calcium and phosphorus for new bone formation (Saliba & El-Haddad, 2009). In ESRD, hyperphosphatemia stimulates FGF-23 secretion in which 1α-hydroxylase activity is inhibited. Thus, calcitriol synthesis is reduced causing vitamin D and calcium deficiency and an increase in PTH levels (Tomasello, 2008; Hruska et al., 2015).

In summary, patients with CKD-MBD experience a constant cycle of abnormal serum levels of phosphorus, calcium, calcitriol, FGF-23, and PTH. The focus should be on correcting these levels to prevent the problems frequently seen in this population such as slowed bone growth and deformities, heart and blood vessel problems, increased hospitalizations, and mortality (National Institute of Diabetes and Digestive and Kidney Diseases, 2015; Thomas et al., 2008). CKD-MBD is a complex and challenging process and requires close communication between the medical team and the patient to improve disease management.

Anemia. Another problem CKD patients may experience is anemia. One of the functions of the kidneys is erythropoietin production, which plays a role in red blood cells (RBC) production in response to low oxygen levels in the blood. As previously mentioned, CKD disrupts many processes in the body, and this includes erythropoietin production. Although anemia in CKD can emerge from other determinants (iron, folate, or vitamin B12 deficiency, gastrointestinal bleeding, inflammation, or blood loss from dialysis), insufficient erythropoietin synthesis is the most specific cause of CKD-associated anemia (Stauffer & Fan, 2014; National Institute of Diabetes and Digestive and Kidney Diseases, 2014; Thomas et al., 2008).

If anemia is not treated, CKD patients may experience irregular heartbeat and heart failure (National Institute of Diabetes and Digestive and Kidney Diseases, 2014). As the GFR falls below 60 mL/min/1.73 m^2, the prevalence of anemia increases progressively and may lead to disease progression, poor quality of life, and increased morbidity and mortality from cardiovascular complications in these patients (Ryu et al., 2017; Thomas et al., 2008). Anemia can be managed by treating the initial cause. This can be done through the supplementation of iron, vitamin B12, or folic acid, erythropoietin injections, RBC transfusions, or diet (National Institute of Diabetes and Digestive and Kidney Diseases, 2014).

Cardiovascular disease. Patients with chronic kidney disease have a significantly increased risk for mortality and morbidity from cardiovascular disease (CVD) (Sarnak, 2003). CVD risk begins in the early stages of CKD and increases as the GFR decreases. The mortality rate from CVD is rapidly increased when dialysis treatment is initiated and is fifty-fold higher compared to the general population (Liu et al., 2014). In kidney transplant patients, CVD accounts for 35 to 50% of all-cause mortality (Sarnak et al., 2003). CVD can refer to many conditions. The conditions often seen in CKD are heart disease, heart failure, and arrhythmia, all of which may be caused by coronary artery disease (American Heart Association, 2017; Alani et al., 2014). Two types of risk factors for CVD have been defined in the CKD population (Sarnak, 2003). These are traditional risk factors such as age, obesity, hypertension, and diabetes and CKD-related risk factors such as anemia, mineral metabolism disorders, and inflammation (Liu et al., 2014). Treating the modifiable risk factors involves the multidisciplinary team to optimize care.

They must be diligent in the assessment of cardiovascular disease in patients with CKD and take appropriate measures to reduce patient risk as well as work with the patient to promote a healthy lifestyle and compliance with prescribed medications (Wright & Hutchison, 2009).

Nutrition in End-Stage Renal Disease

Adequate nutrition is recommended in the current guidelines for ESRD management. Nutrition in ESRD patients is extremely intricate. RDNs play a valuable role in disease management using medical nutrition therapy (MNT), an evidence-based medical approach for treating chronic conditions (Academy of Nutrition and Dietetics, 2006). They gather and assess nutrition information, including dietary intake, anthropometric measurements, and laboratory data (Locatelli et al., 2002). In order to improve the nutritional status of each patient, on-going monitoring and counseling is necessary throughout the disease process as the requirements and utilization of certain nutrients change significantly (Nazar, 2013).

Patients with kidney damage are recommended to limit their intake of certain foods to prevent the accumulation of metabolic waste products causing uremia and electrolyte imbalances but also to protect against hypertension and poor heart and bone health (Rysz et al., 2017). MNT may help manage these health problems resulting from ESRD. The dietary recommendations in CKD are individualized and are different between dialysis and kidney transplant patients (Table 6). These dietary recommendations as well as dietary quality and nutritional status will be discussed individually for the two groups. The primary focus of MNT is to match dietary intake

with kidney function to reduce the accumulation of metabolic waste and prevent the progression of kidney damage and poor outcome in these patients (Beto et al., 2014).

Dietary recommendations for dialysis patients. In the dialysis population, patients still have health complications that cannot be resolved with dialysis and medications alone. Hemodialysis patients receive dialysis treatment at least three times a week. During the rest of the week, waste and fluid accumulation from the diet can cause a strain on many biological processes in the body, such as blood pressure, electrolyte, and hormone homeostasis. This explains how patient adherence to dietary restrictions can impact the course of the disease. The medical team must continue to emphasize the importance of securing nutritional health to optimize patient outcomes.

In the 2002 KDOQI clinical practice guidelines for nutrition in chronic renal failure, Stage 5 CKD adults on dialysis under the age of 60 are recommended a daily energy intake of 35 kcal/kg and 30 – 35 kcal/kg for adults over the age of 60 (National Kidney Foundation, 2000). The calorie ranges are higher to prevent the risk of malnutrition and weight loss because of dialysis treatment and food restrictions limiting caloric intake. Healthy adults are recommended a daily protein intake of 0.8 grams of protein per kilogram of body weight to maintain nitrogen balance (Chen et al., 2017). Adults on hemodialysis and peritoneal dialysis require more protein than the general adult population to replenish the amount that is lost during dialysis and therefore the recommended amount of protein per day is 1.2 g/kg and 1.2 – 1.3 g/kg of protein per day, respectively (National Kidney Foundation, 2000). Patients receiving peritoneal dialysis

require more protein than hemodialysis patients because dialysis treatment lasts throughout the day and night.

Dialysis patients are also recommended to limit their intake of minerals (phosphorus, potassium, calcium, and sodium) because of their increased risk for electrolyte imbalances and other health complications such as hypertension, cardiovascular disease, and bone disorders. KDOQI recommends a dietary phosphorus restriction of 800 to 1,000 mg when serum phosphorus levels are greater than 5.5 mg/dL or PTH levels are elevated in kidney failure patients (National Kidney Foundation, 2003). It is necessary for ESRD patients to maintain normal serum calcium levels of 8.4 to 9.5 mg/dL through dietary calcium, supplementation, or using calcium-phosphate binders not to exceed 2,000 milligrams per day to avoid these problems (National Kidney Foundation, 2003; Beto et al., 2014; Academy of Nutrition and Dietetics, 2010; Martins Pecoits-Filho, & Riella, 2004). To maintain serum potassium levels, patients are recommended less than 2.4 grams of dietary potassium daily (Academy of Nutrition and Dietetics, 2010). The last mineral that is restricted in these patients is sodium. Patients have a hard time restricting their dietary sodium because it occurs in many foods. The NKF KDOQI guidelines recommend CKD patients on hemodialysis to consume no more than 2 grams of sodium per day as well as decreasing sodium intake for those on peritoneal dialysis (Wright & Cavanaugh, 2010). Patients who have hypertension are recommended a sodium restriction of 1.5 grams per day or lower.

Dietary recommendations after kidney transplantation. Patient adherence to medical nutrition therapy maintains an important predictor of post-transplant clinical

outcome and graft function. In the first year after transplantation, patients are susceptible to graft loss because of their increased risk for rejection and infection as well as their co-existing conditions before transplantation. The main goals after transplantation are to achieve optimal energy and protein intake, maintain a desirable body weight, and attenuate the side effects of immunosuppressive medications (Strejc, 2000). The nutrition recommendations for kidney transplant patients are more liberalized compared to patients who received dialysis to manage CKD. Patients are encouraged to follow a balanced diet and exercise regularly to promote a better quality of life and reduce the risk of CKD-associated diseases or graft loss.

The two distinct phases after kidney transplantation in regards to nutritional changes are the early and late post-transplant phases. In the early phase after renal transplantation, patients have increased nutritional demands due to surgical stress and high doses of immunosuppressive medications (Martins et al., 2004). In this phase, the energy intake recommendation is 30 – 35 kcal/kg/day and the protein intake recommendation is 1.3 to 2 g/kg actual body weight. Energy and protein needs are much greater due to the hypercatabolic state and are similar in acute rejection episodes (Martins et al., 2004). In the late post-transplant phase, patients are recommended an energy intake of 25 – 30 kcal/kg and a protein intake of 0.8 – 1 g/kg with an adequately functioning graft to enhance graft survival and minimize the impact of comorbid conditions.

Similar to procedures before transplantation, patients' sodium, potassium, phosphorus, and calcium serum levels are regularly monitored. While successful kidney transplantation corrects the electrolyte imbalances seen prior to transplantation, there are

a few instances where these imbalances may persist. Sometimes it may be due to metabolic changes in the post-transplant period or from the use of immunosuppressive agents (Pochineni & Rondon-Berrios, 2018). Therefore, these minerals are not subjected to dietary restriction unless transplant recipients show abnormal findings in their lab results (Rho et al., 2013).

The dietary recommendations for these minerals should be individualized according to the patient's serum levels and other factors. The normal sodium recommendation is less than 2.4 g/day. There is no limitation on dietary phosphorus. However, hypophosphatemia is commonly seen in the early post-transplant phase which phosphorus supplementation may be necessary (Academy of Nutrition and Dietetics, 2010; Martins et al, 2004). The calcium recommendation is 800 – 1,500 mg/day and not to exceed 2,000 mg/day. The potassium recommendation is less than 2.4 g/day (Academy of Nutrition and Dietetics, 2010). Dietary recommendations are also adjusted dependent on these factors:

- Blood pressure
- Medications
- Catabolism
- Kidney function
- Hydration status
- Gastrointestinal issues

Dietary restrictions may be the same as before patients received a kidney transplant depending on their health condition. For example, if they have diabetes, dietary restrictions still apply to manage their blood glucose levels. In addition, since most kidney transplant patients were previously receiving dialysis treatment prior to kidney transplantation, the nutritional risk factors related to ESRD should still be considered in their disease management to improve nutritional status (Martins et al., 2004).

Table 6

Daily Dietary Recommendations for End-Stage Renal Disease

Nutrient	**Stage 5 CKD ESRD/Dialysis**		**Transplant**	
Energy	<60 years old 35 kcal/kg	>60 years old 30 – 35 kcal/kg	Acute phase 30 – 35 kcal/kg	Late Phase 25 – 30 kcal/kg
Protein	Hemodialysis 1.2 g/kg	Peritoneal dialysis 1.2 – 1.3 g/kg	Acute phase 1.3 – 2 g/kg	Late Phase 0.8 – 1 g/kg
Phosphorus	800 – 1,000 mg		No limit; increase if needed	
Calcium	<2,000 mg		800 – 1,500 mg not to exceed 2,000 mg	
Potassium	<2,400 mg		<2,400 mg	
Sodium	<2,000 mg		<2,400 mg	

Note. From National Kidney Foundation, 2000; National Kidney Foundation, 2003; Beto et al., 2014; Academy of Nutrition and Dietetics, 2010; Martins et al., 2004; Wright et al., 2010.

Nutritional Status in End-Stage Renal Disease

Maintaining good nutritional status in ESRD patients is a difficult process, especially for those who have other comorbid conditions. The assessment of nutritional status in ESRD patients allows early detection and treatment of malnutrition.

Malnutrition is defined as insufficient calorie and protein intake. Nutritional status can be assessed through dietary intake, anthropometric measurements, and biochemical parameters. Unfortunately, there is no specific nutritional marker that can be performed without being affected by other factors (Cupisti et al., 2010). However, serum albumin is mainly used to measure malnutrition in CKD patients and is the most powerful predictor of mortality (Nazar, 2013). Malnourished patients often show signs of inflammation, characterized as an increase in plasma C-reactive protein. Low serum albumin may be a consequence of inflammation, which suggests that serum albumin may not be a reliable indicator of nutritional status (Locatelli et al., 2002). For this reason, regular assessment and monitoring of nutritional status is mandatory in ESRD patients.

Dialysis patients. Many studies have presented that protein-energy malnutrition (PEM) is often seen in ESRD patients, especially those receiving dialysis treatment (Cupisti et al., 2010; National Kidney Foundation, 2000). PEM may be a result of inadequate food intake, dialysis treatment, protein catabolism, inflammation, and uremia (National Kidney Foundation, 2000). Studies have also shown that PEM is associated with morbidity and mortality, poor quality of life, and reduced physical function (Cupisti et al., 2010).

Another reliable tool used to assess nutritional status is the subjective global assessment (SGA) tool (Cupisti et al., 2010). The SGA evaluates nutritional status based on weight change, dietary intake, gastrointestinal symptoms, functional capacity, diseased state, as well as a physical examination to predict nutrition-related clinical outcomes (Steiber et al., 2004). It was reported that 44% of patients in Stages 4 - 5 of

CKD had mild to severe malnutrition based on the SGA, with 30% of patients on hemodialysis and 40% of patients on peritoneal dialysis (Nazar, 2013).

In a study comparing nutrient intake to the current dietary recommendations for dialysis patients, patients were consuming energy and protein below the recommendations. Energy intake was 23 – 28 kcal/kg of body weight per day and protein intake was 0.95 – 1 g/kg body weight per day. This suggests that dialysis patients were not meeting the ideal levels of energy and protein intake explaining the high incidence of undernutrition in this population (Kovesdy, Shinaberger, & Kalantar-Zadeh, 2010).

Epidemiological studies suggest that dietary interventions focusing on optimal energy and protein intake can improve nutritional status in ESRD patients. Another aspect of diet to acknowledge is that in addition to energy and protein intake, it is not only the quantity of food but also the quality of the diet that affects the overall health and well-being of ESRD patients, as they may be deficient in various micronutrients (Kovesdy et al., 2010). However, the aim to achieve adequate energy and protein intake can cause elevated serum levels of certain nutrients (phosphorus, potassium, calcium, and sodium). Thus, dietary intervention should be planned carefully and under the supervision of the multidisciplinary team.

Kidney transplant patients. After successful kidney transplantation, many complications associated with ESRD disappear with improved nutritional status. However, in a majority of ESRD patients, malnutrition persists even after kidney transplantation. Although weight gain after transplantation is common, approximately 15 – 23% of recipients experience malnutrition (Tutal et al., 2013). Up to the first year after

transplantation, serum albumin levels may be below normal, also suggesting protein malnutrition (Sezer et al., 2006). In general, weight gain was believed to be from overnutrition, but since these patients have followed many dietary restrictions for a long time before transplantation, many of them can experience undernutrition. The presence of malnutrition may affect graft and patient survival. Therefore, the regular evaluation of nutritional status in patients after kidney transplantation is extremely important. The major nutritional goal for kidney transplant recipients is to treat preexisting undernutrition and prevent excessive weight gain (Rho et al., 2013).

On average, patients gain between 6 and 10 kg of body weight and a body mass index (BMI; kg/m^2) increase of between 2 and 3.8 kg/m^2 in the first year following renal transplantation (Aksoy, 2016). BMI is used to classify overweight and obesity in adults. An increase in fat mass and a loss of lean body mass was also detected in these patients (Netto et al., 2012). It has been assumed that weight gain after transplantation was due to increased appetite from steroid therapy, fewer dietary restrictions, corrected uremia, and lack of physical activity (Mantoo et al., 2007).

Although weight gain is common after successful kidney transplantation, there is a greater concern for obesity in these patients. Obesity is also associated with decreased graft and patient survival, although some studies suggest otherwise (Martins et al., 2004). In Olarte et al.'s study examining obesity and its relationship with graft failure, their findings suggest that post-transplant weight gain is associated with a higher incidence of delayed and worsened graft function after one year compared to patients who maintained their BMI (Olarte & Hawasli, 2009). In agreement, el-Agroudy's study presented with a

statistically significant difference in 5- and 10-year graft and patient survival and higher severity in acute rejection episodes in the obese group, although this was not statistically significant (el-Agroudy et al., 2004). Contradicting these findings is Torres's study where the findings suggested that weight gain was associated with a better glomerular filtration rate (Torres et al., 2007). More research is needed in this area, as it is still unclear whether obesity may improve or diminish patient graft outcomes.

The weight gain in kidney transplant patients resulting in overweight and obesity is associated with serious health complications. These include delayed graft function, hypertension, dyslipidemia, cardiovascular disease, prolonged hospitalization, acute rejection, and decreased graft and patient survival (Chadban et al., 2010). In summary, early dietary interventions and ongoing assessment may be an effective approach in managing malnutrition and reducing excessive weight gain in transplant recipients to prevent these complications.

Dietary Quality in End-Stage Renal Disease

It is recognized that the dietary quality of CKD patients is suboptimal. Many factors play a role in poor dietary quality such as the restriction of certain nutrients (sodium, potassium, phosphorus, calcium) or financial barriers related to CKD which may impact disease progression (Campbell & Carrero, 2016). Recent observational studies have suggested that healthier dietary quality is associated with lower ESRD risk and mortality in patients with CKD Stage 3 to 5 (Luis et al., 2016). Also a healthy diet, including high intakes of vegetables, fruits, whole grains, legumes, and fish and low in saturated fat and sodium was associated with lower rates of age-adjusted all-cause

mortality in individuals with CKD (Rysz et al., 2017). However, many studies assessing dietary quality in CKD patients have shown low compliance to the dietary recommendations for CKD.

Dialysis patients. Many of the dietary recommendations for dialysis patients are highly restrictive which is why patients find it challenging to find anything permissible to eat. The dietary regimen for these patients is one of the most restrictive diets and may cause patients frustration, leading to suboptimal adherence and compliance (Kalantar-Zadeh et al., 2015). In a study examining hemodialysis patients' adherence to international renal-specific recommendations using 3-day food records, it was found that a large number of patients did not meet the target recommendations for energy, macronutrients, and micronutrients intake (Luis et al., 2016). These patients were consuming a high proportion of fat, mainly saturated fat, and consuming too little fiber possibly from the avoidance of fruits and vegetables to control potassium levels. The nutrients also consumed in excess were organic phosphorus, calcium, sodium, and potassium, which these minerals are normally restricted in these patients (Luis et al., 2016). Some of the nutrient deficiencies were vitamin D, vitamin E, and folic acid. The dietary information reported by these patients were suggestive of poor diet quality (Luis et al., 2016).

In an Australian study, dietary intake of hemodialysis patients was of suboptimal quality when 24-hour dietary recalls were evaluated. Dietary quality was assessed using Total Diet Score (TDS), which is a tool developed by Russel et al. The TDS aligns with the Dietary Guidelines for Australian Adults and was created to examine the diet quality

of older Australian populations (Russell et al., 2017). A score of 15/20 indicated high dietary quality. The study participants had a mean score of 10.2 out of 20 indicating low dietary quality. None of the participants scored over 15 points. The authors suggested an improvement in energy, protein, carbohydrate, fiber, potassium, and omega-3 fatty acids intake and reducing the consumption of sodium and saturated fat for better dietary quality (Roach et al., 2017).

As presented, the dialysis population would benefit from improvements in the quality of their diet. The dietary restrictions imposed in this population are demanding and make it difficult to reach optimal nutrient intake. Dietary intervention in relation with ESRD is a complex issue. The dietitian plays an important role in filling the gap between nutrition and empowering CKD patients to change their lifestyles to improve their quality of life and reduce their risk for mortality.

Kidney transplant patients. Few studies have examined dietary quality in kidney transplant recipients. Heaf et al. (2004) evaluated the dietary habits of transplant recipients using 3-day dietary histories. The findings of the study were protein and energy intake were sufficient, males and older patients had high fat intake, potassium intake was low, and patients were deficient in folic acid, iron, vitamin D, thiamin, iodine, and selenium (Heaf et al., 2004). Rho et al. also evaluated nutrient intake in transplant recipients using 3-day food records. In Rho's study, protein recommendations were met but patients did not meet the recommendations for calcium, folate, and vitamin C. The low calcium intake group consumed less fish, milk, and dairy products than the other

groups. They also found that patients with inadequate folate, vitamin C, and calcium intake consumed less milk and dairy products, vegetables, and fruits (Rho et al., 2013).

From these studies, transplant recipients need to improve their dietary quality, especially their intake of micronutrients. Although many dietary restrictions are lifted after kidney transplantation, the dietary quality of transplant patients still remains unclear. More research in this area is needed to develop specific dietary recommendations to address dietary quality in these patients.

CHAPTER II

Methods to Assess Dietary Intake and Quality

Automated Self-Administered 24 Dietary Assessment Tool

The most common methods for estimating dietary intake are food records and dietary recalls (National Kidney Foundation, 2000). Evidence has shown that a 24-hour dietary recall is the preferred tool for monitoring dietary intake in study populations (Subar et al., 2012). The National Cancer Institute (NCI) developed a public web-based dietary recall tool for researchers, clinicians, and educators called the Automated Self-Administered 24 (ASA24) Dietary Assessment Tool (Blanton et al., 2006). The NCI released its most current version, the ASA24 2016, in March 2016. The ASA24 is influenced by the United States Department of Agriculture's (USDA) Automated Multiple Pass Model (AMPM) which is a validated computerized 24-hour dietary recall tool completed through an interview in person or phone call. The interview is a 5-step multiple-pass approach that collects different information about foods consumed using memory cues for more accuracy and completeness of recalls (see Figure 3) (Steinfeldt, Anand, & Murayi, 2013).

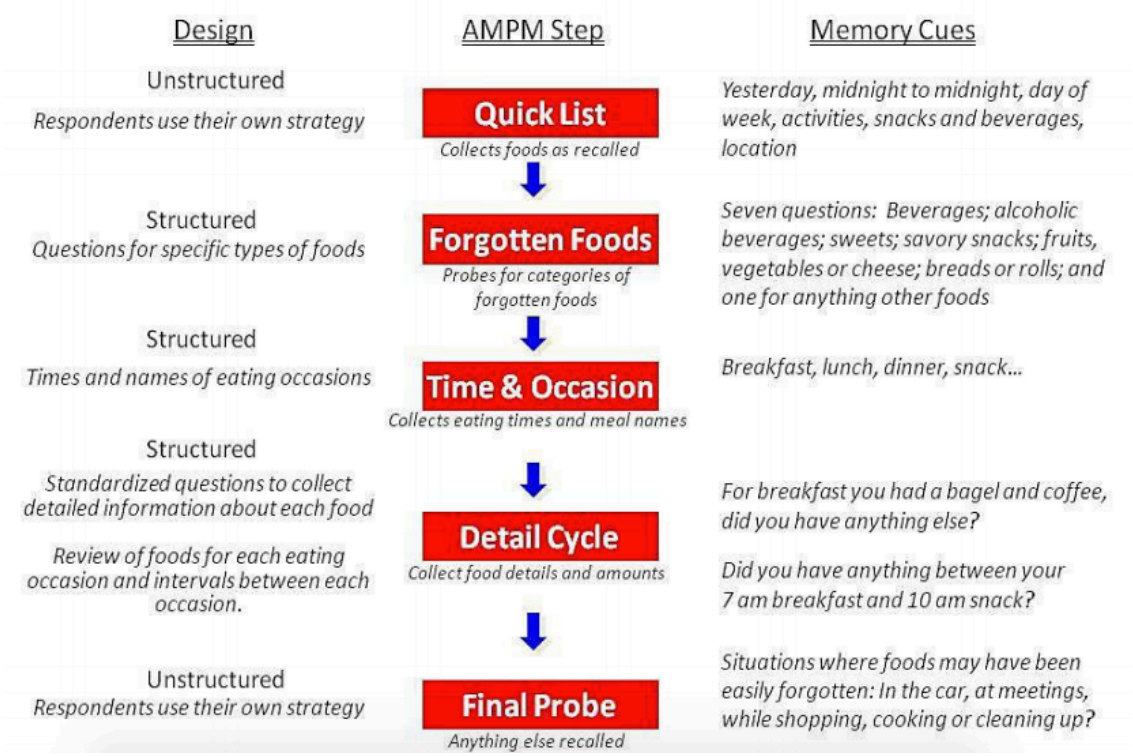


Figure 3. United States Department of Agriculture Automated Multiple-Pass Method. Adapted from "Food reporting patterns in the USDA Automated Multiple-Pass Method," by Steinfeldt, L. Anand, J., & Murayi, T., 2013, *Procedia Food Science*, *2*, 145 – 156.

The ASA24 is an online dynamic user interface that advances per a modified AMPM to collect dietary recalls (see Table 7) (Subar et al., 2012). It comprises two web-based applications, the Respondent and Researcher Websites. The Respondent Website is accessed by the participant completing the dietary recall. They are guided through a series of questions describing their food intake from the previous day by navigating through the ASA24 database of foods, supplements, and beverages derived from the Food and Nutrient Database for Dietary Studies (FNDDS) 2011-12, the Food Pyramid Equivalents (FPED) 2011-12, and the National Health and Nutrition Examination Survey (NHANES) Dietary Supplements Database (DSD) 2011-12 (National Cancer Institute,

2018). The user-friendly software also allows the respondent to report the method of food preparation, portion sizes, and meal times. The Researcher Website allows the researcher to access and evaluate the completed dietary recall data (Kirkpatrick et al., 2014).

Table 7

Steps to the Automated Self-Administered 24 Diet Interview

ASA24 passes	**Description of information collected**
Meal-Based Quick List	Respondents are asked to report meal name, time, and optionally: location, TV/computer use, who one ate with. Food and drinks consumed are reported without details by browsing or searching.
Meal Gap Review	Respondents are asked if they consume anything during any 3-hour gaps between eating occasions, between midnight and the first eating occasion, and between the last eating occasion and midnight. "Yes' responses return the respondent to the Quick List to add foods or drinks.
Details	Respondents are asked for details about the foods and drinks they recorded during the Quick List, including form, preparation methods, the amount eaten, and any additions.
Forgotten Foods	Respondent are asked about the consumption of commonly forgotten foods and drinks and report them as necessary by returning to the Quick List
Final Review	Respondents are prompted to review all the foods and drinks reported for the intake day; they can make edits and add meals and foods and drinks as desired.
Last Chance	Respondents are given another opportunity to add foods or drinks.
Usual Intake	Respondents are asked: "Was the amount of food that you ate yesterday more than usual, usual, or less than usual?"
Supplement Module (Optional)	Respondents are asked to provide information about the types and doses of supplements consumed by completing quick list, detail, and final review passes.

Note. Adapted from "The automated self-administered 24-hour dietary recall (ASA24): A resource for researchers, clinicians and educators from the National Cancer Institute," by Subar, A., Kirkpatrick, S., Mittl, B., Zimmerman, T., Thompson, F., Bingley, C., Willis, G., Islan, N., Baranowski, T., McNutt, S., Potischman, N, 2012, *Journal of Academy of Nutrition and Dietetics*, *112(8)*, 1134 – 1137.

Healthy Eating Index 2010

The Healthy Eating Index (HEI) 2010 is a tool that measures diet quality and is in conformance with the 2010 Dietary Guidelines for Americans (DGA). The DGA is evidenced based and the nutrition policy of the US government (Guenther et al., 2013). It emphasizes the importance of following a healthy eating pattern to maintain health and reduce the risk of chronic diseases. The HEI focused on the key recommendations of the DGAs; for example, increasing fruit and vegetable intake, limiting refined grains, and reducing intake of calories from solid fats and added sugars (United States Department of Agriculture and United States Department of Health and Humans Services, 2010). The HEI 2010 comprises of nine adequacy and three moderation components with their scoring standards (see Table 8). For the adequacy components, this means the greater the intake of that food component the higher the score; whereas, for the moderation components, the greater the intake, the lower the score. The highest possible score for the HEI is 100 points. Thus, higher total and component scores indicate a closer conformance with the dietary guidelines (Guenther et al., 2013). A HEI score of over 80 implies "good" diet quality, a score between 51 and 80 indicates the diet "needs improvement," and a score of less than 51 implies a "poor diet" (Basiotis, Carlson, Gerrior, Juan, & Lino, 2002). The average 2010 HEI total scores from 2011 to 2012 for the US total population, which included children and older adults, was 59/100 (see Table 9) (United States Department of Agriculture Center for Nutrition Policy and Promotion, 2012).

Table 8

Healthy Eating Index-2010 Components and Standards for Scoring

HEI-2010[1] component	Maximum	Standard for maximum score	Standard for minimum score of zero
Adequacy (higher score indicates higher consumption)			
Total Fruit[2]	5	≥ 0.8 cup equiv./1,000 kcal[10]	No fruit
Whole Fruit[3]	5	≥ 0.4 cup equiv./1,000 kcal	No whole fruit
Total Vegetables[4]	5	≥ 1.1 cup equiv./1,000 kcal	No vegetables
Greens and Beans[4]	5	≥ 0.2 cup equiv./1,000 kcal	No dark-green vegetables, beans, or peas
Whole Grains	10	≥ 1.5 ounce equiv./1,000 kcal	No whole grains
Dairy[5]	10	≥ 1.3 cup equiv./1,000 kcal	No dairy
Total Protein Foods[6]	5	≥ 2.5 ounce equiv./1,000 kcal	No protein foods
Seafood and Plant Proteins[6.7]	5	≥ 0.8 ounce equiv./1,000 kcal	No seafood or plant proteins
Fatty Acids[8]	10	(PUFAs + MUFAs)/SFAs ≥ 2.5	(PUFAs + MUFAS)/SFAs ≤ 1.2
Moderation (higher score indicates lower consumption)			
Refined Grains	10	≤ 1.8 ounce equiv./1,000 kcal	≥ 4.3 ounce equiv./1,000 kcal
Sodium	10	≤ 1.1 gram/1,000 kcal	≥ 2.0 grams/1,000 kcal
Empty Calories[9]	20	≤ 19% of energy	≥ 50% of energy

Note. [1]Intakes between the minimum and maximum standards are scored proportionately.
[2] Includes 100% fruit juice.
[3] Includes all forms except juice
[4] Includes any beans and peas not counted as Total Protein Foods
[5] Includes all milk products, such as fluid milk, yogurt, and cheese, and fortified soy beverages
[6] Beans and peas are included here (and not with vegetables) when the Total Protein Foods standard is otherwise not met.
[7] Includes seafood, nuts, seeds, soy products (other than beverages) as well as beans and peas counted as Total Protein Foods.
[8] Ratio of poly- and monounsaturated fatty acids (PUFAs and MUFAs) to staturated fatty acids (SFAs)
[9] Calories from solid fats, alcohol, and added sugars; threshold for counting alcohol is > 13 grams/1,000 kcal.
[10] Equiv. = equivalent, kcal = kilocalories

Adapted from *United States Department of Agriculture Center for Nutrition Policy and Promotion*, 2013, Retrieved September 15, 2018 from https://www.cnpp.usda.gov/sites/default/files/healthy_eating_index/CNPPFactSheetNo2.pdf

Table 9

Healthy Eating Index-2010 Total and Component Scores for the United States Total Population

HEI-2010 Dietary Component (maximum score)	Total Population ≥ 2 years (n = 7,933)	Children 2-17 (n = 2,857)	Older Adults ≥ 65 years (n = 1,032)
		Mean Score (standard error)	
Total fruit (5)	3.00 (0.11)	3.91 (0.18)	3.85 (0.22)
Whole fruit (5)	4.01 (0.17)	4.78 (0.22)	4.99 (0.05)
Total vegetables (5)	3.36 (0.08)	2.10 (0.09)	4.16 (0.19)
Greens and beans (5)	2.98 (0.15)	0.70 (0.09)	3.58 (0.47)
Whole grains (10)	2.86 (0.13)	2.50 (0.10)	4.23 (0.34)
Dairy (10)	6.44 (0.14)	9.03 (0.22)	5.99 (0.16)
Total protein foods (5)	5.00 (0.00)	4.44 (0.13)	5.00 (0.00)
Seafood and plant proteins (5)	3.74 (0.20)	3.05 (0.17)	4.91 (0.18)
Fatty acids (10)	4.66 (0.14)	3.29 (0.18)	5.60 (0.36)
Refined grains (10)	6.19 (0.15)	4.91 (0.16)	7.34 (0.31)
Sodium (10)	4.15 (0.06)	4.85 (0.25)	3.66 (0.26)
Empty calories (20)	12.60 (0.23)	11.50 (0.28)	14.99 (0.44)
Total HEI score (100)	59. 00 (0.95)	55.07 (0.72)	68.29 (1.76)

Note. Adapted from *United States Department of Agriculture Center for Nutrition Policy and Promotion*, 2012, Retrieved October 10, 2018 from https://www.cnpp.usda.gov/hei-scores-americans.

Hypotheses

The purpose of this secondary data analysis of ASA24 data of renal transplant patients was to examine nutrient intake, dietary quality, and weight gain in post-renal transplant recipients from pre-transplant baseline through post-transplant at 3 months and 1 year. The study null hypotheses were:

1. Intake of energy, macronutrients (carbohydrates, fat, and protein), potassium, phosphorus, calcium, and sodium do not increase following renal transplantation.
2. Post-transplant weight gain and BMI is not associated with increased energy, protein, fat, and carbohydrate intake.
3. Quality of diet in comparison to the 2010 Dietary Guidelines for Americans, as assessed by the HEI 2010, does not improve following renal transplantation.

CHAPTER III

Methodology

Study Design

This study was a secondary data analysis of data from the prospective cohort study, "Weight Gain After Kidney Transplantation," performed by Baylor College of Medicine and the Houston Methodist J.C. Walter Jr. Transplant Center from January 2016 to 2017. This cohort study was a pilot study that investigated post-transplant weight gain through the examination of body composition and energy metabolism in renal transplant recipients who received kidneys from living donors.

The coded de-identified data file was provided in Microsoft Excel format by Houston Methodist Hospital. The data file included patient demographics, anthropometric data, and nutrient intake data. The dietary intake, dietary quality, and anthropometric measurements of patients who received a kidney transplant were assessed using information provided in the data file.

Participants

Data was collected from 31 patients who received a kidney transplant at the Houston Methodist J.C. Walter Jr. Transplant Center at pre-transplant baseline (up to 6 months before transplant), post-transplant at three months (10 – 12 weeks after transplant), and post-transplant at one year (50 – 52 weeks after transplant). The inclusion criteria were men and women ages 18 to 75 years who were living donor recipients in the next six months, currently receiving dialysis treatment, and had internet access to provide 24-hour diet recalls. Patients who were pregnant, had a diagnosis of type 1 diabetes,

insulin dependent, participating in a clinical trial using immunosuppressive therapy or had an active malignancy or ongoing infection were excluded from the study.

Study Procedures

Patients from the cohort study were followed from pre-transplant baseline through post-transplant at three months and one year. One day prior to each study visit, patients received a phone call and were reminded of their appointments at Houston Methodist Hospital. During each visit, patient's anthropometric data and dietary recall were collected. The anthropometric measurements collected were height (centimeters) and weight (kilograms) and were used to calculate body mass index (BMI) (kg/m^2). These values were recorded into the Excel file for each subject. Dietary intake data was collected using the 2014 Automated Self-Administered 24-Hour (ASA24) Dietary Assessment Tool. Patients were provided with instructions and completed their 24-hour diet recall by accessing the Respondent Website of the ASA24 on a Houston Methodist computer. The Houston Methodist Hospital registered dietitian was available to assist patients with dietary intake entry. In between the study, the NCI introduced their most current version of the ASA24, the ASA24 2016. The ASA24 2014 dietary data for each patient was transferred to the ASA24 2016 database because the ASA24 2014 website was longer accessible after the 2016 update. Once the patients' dietary recall was submitted for each study visit day, the dietary data was exported from the Researcher Website into the same Excel file containing the anthropometric data. After review of the data, Houston Methodist Hospital provided the Excel file electronically for analysis.

From the Excel file, the anthropometric variables (body weight (kg) and BMI (kg/m2)) and daily nutrient variables (kilocalories (kcal), protein (g), fat (g), carbohydrates (g), phosphorus (mg), potassium (mg), calcium (mg), and sodium (mg)) were evaluated for change from pre-transplant baseline through post-transplant at 3 months and 1 year.

The dietary data in the Excel file provided by Houston Methodist Hospital was also used in the calculation of the HEI scores to assess the dietary quality of each patient. On the National Cancer Institute website, a Statistical Analysis Software (SAS) program was provided that automatically calculated each patient's total and component HEI score using the ASA24 dietary data. The dietary data contained specific nutrient variables that were located and retrieved by the SAS program in order to calculate the scores. The program was launched on SAS University Edition. Once the dietary data for each subject was uploaded into the SAS program and the program was run, the HEI total and food component scores were automatically calculated and saved into a separate Excel file by SAS. These steps were repeated to calculate each patient's HEI total and component scores for pre-transplant baseline and post-transplant at 3 months and 1 year. The dietary quality of the patients was assessed for change from pre-transplant baseline through post-transplant at 3 months and one year.

Statistical Analysis

Repeated Measures ANOVA was used to assess the changes in mean weight, BMI, kilocalories, protein, fat, phosphorus, potassium, sodium, calcium, and HEI scores from pre-transplant baseline through post-transplant at 3 months and 1 year. Spearman's

Rho was performed to examine the relationship of mean energy and macronutrient (protein, fat, and carbohydrates) intake with mean body weight and BMI at pre-transplant baseline and post-transplant at 3 months and 1 year. Spearman's Rho was also performed to examine the relationship between dietary quality and weight at pre-transplant baseline and post-transplant at 3 months and 1 year. Spearman's Rho was used instead of regression analysis due to a small sample size in this study. All statistical analyses were performed with IBM SPSS version 25 with level of significance set at $p < 0.05$.

CHAPTER IV

Results

Sample Characteristics

A total of 31 patients from Houston Methodist Hospital agreed to participate in the cohort study. Of the 31 patients, five did not complete the study and were excluded. The study sample comprised of 22 (85%) men and 4 (15%) women. The mean age was 47.7 ± 11.5 years. This study included 13 (50%) Caucasians, 5 (19%) Blacks, 7 (27%) Hispanics, and 1 (4%) Asian. Chronic kidney disease was caused by hypertension in six (23%) subjects, diabetes in four (15%) subjects, and other in 16 (62%) subjects. At baseline, 12 (46%) patients received peritoneal dialysis treatment, 12 (46%) patients received hemodialysis treatment, and two (8%) patients were pre-dialysis (see Table 10).

Obesity Prevalence

At pre-transplant baseline, eight (31%) subjects were normal weight (BMI between 18.5 and 24.9), 7 (27%) subjects were overweight (BMI between 25 and 29.9), and 11 (42%) subjects were obese (BMI ≥ 30 kg/m^2). One year after transplantation, five (19%) subjects were normal weight, eight (31%) subjects were overweight, and 13 (50%) subjects were obese. At pre-transplant baseline, the group was classified as overweight with a mean BMI of 28 ± 5, but after one year, they were classified as obese with a mean BMI of 31 ± 6. From baseline to 1 year after transplantation, patients gained an average of 7 kg, and their BMI increased by 3 kg/m^2 (see Table 11).

Table 10.

Participant Characteristics

	Total *n* = 26
Clinical and demographic characteristics	
Age, years, mean ± SD	47.7 ± 11.5
Sex (male), n (%)	22 (85)
Race, n (%)	
Caucasian	13 (50)
Black	5 (19)
Hispanic	7 (27)
Asian	1 (4)
Cause of chronic kidney disease, n (%)	
Hypertension	6 (23)
Diabetes	4 (15)
Other	16 (62)
Dialysis Treatment, n (%)	
Peritoneal	12 (46)
Hemodialysis	12 (46)
Pre-dialysis	2 (8)

Anthropometric and Nutrient Variables

During pre-transplant baseline, patients were consuming an average of 1978 ± 936 kilocalories per day, with approximately half in the form of carbohydrates (226 ± 140 g), 18% in the form of protein (87 ± 52 g), and 37% in the form of fat (82 ± 44 g). For the minerals, there was an average intake per day of 718 ± 477 mg of calcium, 1269 ± 646 mg of phosphorus, 2147 ± 1013 mg of potassium, and 3405 ± 1852 mg of sodium (see Table 11).

There was a significant change in mean body weight ($p = 0.002$), BMI ($p = 0.002$), fat intake ($p = 0.033$), and potassium intake ($p = 0.047$) from pre-transplant baseline through post-transplant at 3 months and 1 year. However, mean fat and potassium intake only significantly increased from baseline to 3 months but not from baseline to one year or from 3 months to one year in the pairwise comparison. On the other hand, mean body weight and BMI significantly increased over time between each time point (see Figure 4 and 5, respectively). There was no significant change in mean kilocalories, carbohydrates, protein, phosphorus, calcium, and sodium intake from pre-transplant baseline through post-transplant at 3 months and 1 year. All of the nutrient variables increased during post-transplant at 3 months but subsequently dropped at 1 year post-transplant (see Table 11).

There was a moderate negative correlation observed between BMI and carbohydrate intake at 1 year post-transplant that was statistically significant ($r = -0.394$, $p = 0.046$). A moderate positive correlation was observed between BMI and fat intake at 1 year post-transplant that was statistically significant ($r = 0.427$, $p = 0.030$). A strong

positive correlation was also observed between body weight and fat intake at 1 year post-transplant that was statistically significant ($r = 0.556, p = 0.003$). No correlation was found between energy and protein intake and body weight or BMI from pre-transplant baseline to post-transplant at 3 months and 1 year.

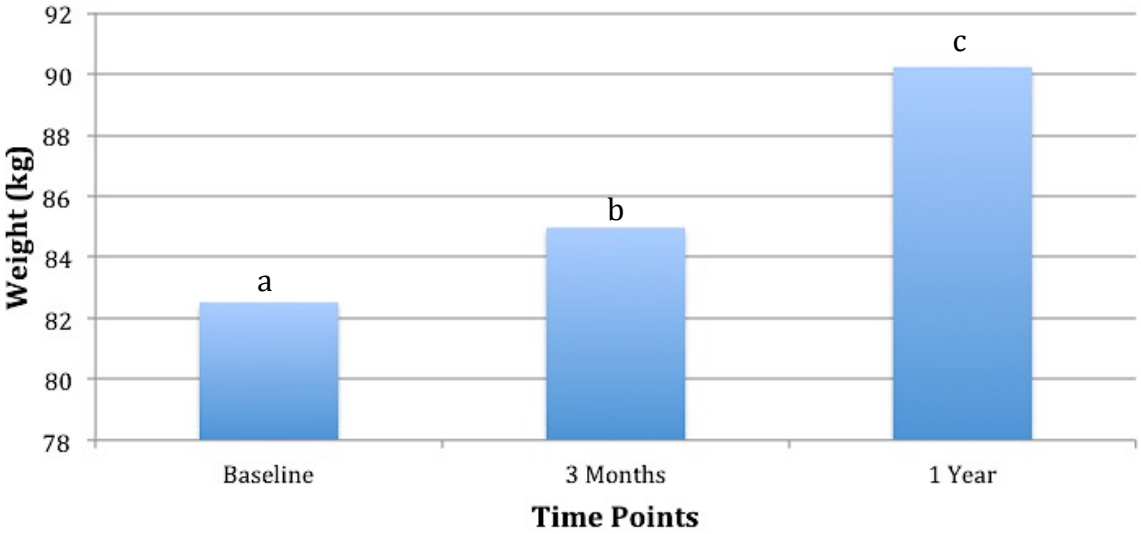
Table 11

Anthropometric and Nutrient Intake

Variable ($n = 26$)	Baseline	3 Months	1 Year	*P*-value
Weight (kg)	83 ± 18	85 ± 18	90 ± 18	0.002*
BMI (kg/m^2)	28 ± 5	29 ± 5	31 ± 6	0.002*
Energy (kcal/d)	1978 ± 936	2343 ± 828	2217 ± 702	0.227
Protein (g/d)	87 ± 52	97 ± 41	96 ± 42	0.695
Carbohydrates (g/d)	226 ± 140	249 ± 115	237 ± 108	0.761
Fat (g/d)	82 ± 44	108 ± 45	101 ± 35	0.033*
Calcium (mg/d)	718 ± 477	1079 ± 692	964 ± 409	0.068
Phosphorus (mg/d)	1269 ± 646	1614 ± 652	1475 ± 566	0.146
Potassium (mg/d)	2147 ± 1013	2866 ± 1235	2543 ± 1185	0.047*
Sodium (mg/d)	3405 ± 1852	4312 ± 1373	4039 ± 1490	0.098

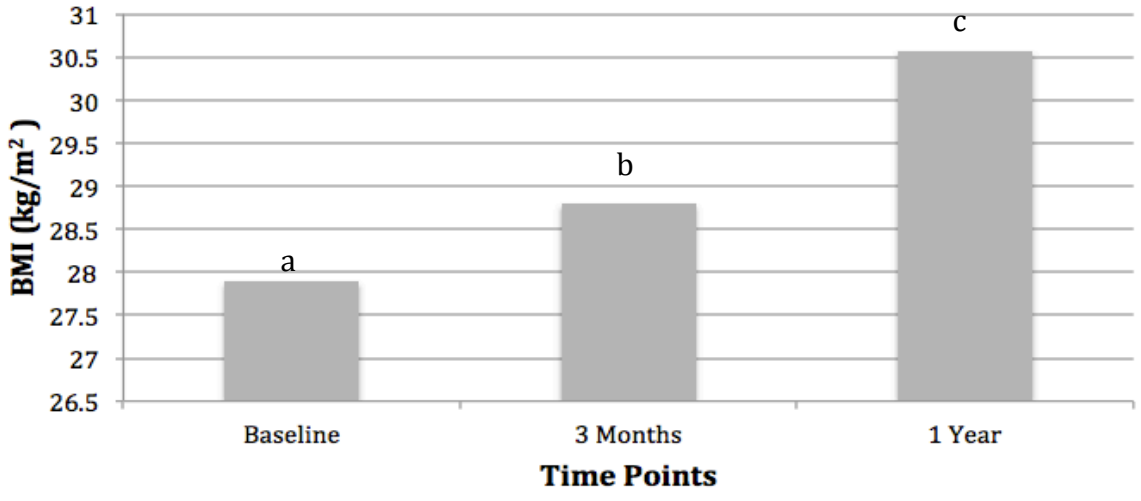
Note. [1] Mean ± standard deviation
[2] $p < 0.05$
[3] *Significant

Figure 4. Mean Body Weight From Pre-Transplant Baseline to Post-Transplant at Three Months and One Year[1,2,3]



[1] Different letters are significantly different from each other
[2] $p < 0.05$
[3] $n = 26$

Figure 5. Mean Body Mass Index From Pre-Transplant Baseline to Post-Transplant at Three Months and One Year [1,2,3]



[1] Different letters are significantly different from each other
[2] $p < 0.05$
[3] $n = 26$

Subjects Meeting or Exceeding Dietary Recommendations

The dietary intake of each patient was assessed to determine the percentage of patients that met or exceeded the nutritional recommendations for pre-transplant baseline on dialysis and post-transplant. The nutrient variables assessed were energy (adjusted for age), protein (adjusted for dialysis treatment), phosphorus, calcium, potassium, and sodium intake (see Table 12). The mid-range for the energy and protein daily intake recommendations was used to assess dietary intake in dialysis and transplant patients. A few patients met the recommendations for energy and protein intake. Over half of the patients at pre-transplant baseline exceeded the phosphorus intake recommendations. Less than half of the patients met the calcium intake at pre-transplant baseline and post-transplant at 3 months. At 3 months post-transplant, potassium intake was suboptimal. A majority of patients exceeded sodium intake recommendations for all time points.

Table 12

Percentage of Participants Meeting or Exceeding Nutrient Intake Recommendations

Variable	Pre-transplant Baseline	3 Months	1 Year
Energy	0%	12%	8%
Protein	4%	12%	19%
Phosphorus[3]	(54%)	N/A	N/A
Calcium	23%	42%	62%
Potassium	58%	38%	50%
Sodium	(85%)	(92%)	(85%)

Note. [1]Percentage meeting recommended amounts; percentage exceeding in parentheses
[2] $n = 26$
[3] Post-renal transplant no restriction

Dietary Quality

During the conversion from ASA24 2014 to ASA24 2016, dietary data was lost for six subjects. The ASA24 2014 was inaccessible to retrieve the missing data. As a result, only 20 subjects had complete dietary information to compute HEI scores for the assessment of dietary quality. Dietary quality of patients, as assessed by mean HEI total and component scores, did not significantly change from pre-transplant baseline through post-transplant at 3 months and 1 year. The mean HEI total score at baseline was 45.74 ± 14.99, which then increased to 49.66 ± 10.70 at post-transplant 3 months and decreased to 42.59 ± 12.70 at 1 year post-transplantation (see Table 13). Individual HEI total scores were categorized as "good," "needs improvement, " and "poor" to assess dietary quality at pre-transplant baseline through post-transplant at 3 months and 1 year. None of the participants had good dietary quality and a majority of participants had poor dietary quality for all time points (see Table 14).

Individual HEI total scores were compared to the national average HEI total score of 59/100 (United States Department of Agriculture for Nutrition Policy and Promotion, 2012). At pre-transplant baseline, 16 subjects had a score below 59. At 3 months post-transplant, 17 subjects had a score below 59. At one year post-transplant, 19 subjects had a score below 59 (see Figure 6). The mean HEI dietary component scores were also compared to the national average HEI component scores. At pre-transplant baseline and 3 months post-transplant, all the component scores were below the national average component scores except the empty calories and fatty acids components. At one year

post-transplant, all the component scores were below the national average component scores except the fatty acid component.

For the adequacy components, mean HEI scores decreased for greens and beans and total protein foods and increased for dairy from pre-transplant baseline through post-transplant at one year although not significant. For the moderation components, refined grains and sodium scores improved over time. The mean HEI scores for the remaining food components increased at 3 months from baseline and decreased at 1 year.

In the correlation analysis, there was no correlation found between dietary quality and weight gain in patients following renal transplantation.

Table 13

Healthy Eating Index Total and Component Score

Food Component	Baseline	3 Months	1 Year	*P*-value
Total Fruit	1.56 ± 1.98	1.69 ± 1.80	0.74 ± 1.76	0.119
Whole Fruit	1.38 ± 1.96	1.74 ± 2.12	0.79 ± 1.82	0.332
Total Vegetables	2.80 ± 1.64	2.81 ± 1.55	2.76 ± 1.61	0.995
Greens and Beans	2.06 ± 2.37	1.65 ± 2.33	0.93 ± 1.81	0.178
Whole Grains	1.71 ± 3.29	2.05 ± 3.25	1.74 ± 3.35	0.995
Dairy	3.47 ± 3.05	3.78 ± 3.01	4.48 ± 3.43	0.698
Total Protein Foods	4.63 ± 1.01	4.55 ± 1.10	4.50 ± 1.03	0.894
Seafood and Plant Proteins	0.87 ± 1.70	2.45 ± 2.44	2.0 ± 2.34	0.114
Fatty Acids	5.63 ± 3.30	6.78 ± 3.52	5.39 ± 3.23	0.320
Refined Grains	4.13 ± 3.91	4.55 ± 4.10	4.91 ± 3.66	0.544
Sodium	3.53 ± 3.39	2.51 ± 3.05	2.79 ± 3.41	0.570
Empty Calories	13.97 ± 6.26	15.09 ± 5.26	11.57 ± 6.50	0.106
Total HEI Score	45.75 ± 14.99	49.66 ± 10.70	42.59 ± 12.70	0.141

Note. [1] Mean ± standard deviation
[2] $n = 20$

Table 14 *Individual Healthy Eating Index Total Scores Categories*			
Category	Baseline	3 Months	1 Year
Good (> 80)	0	0	0
Needs improvement (51 – 80)	7	8	5
Poor (< 51)	13	12	15
Note. $n = 20$			

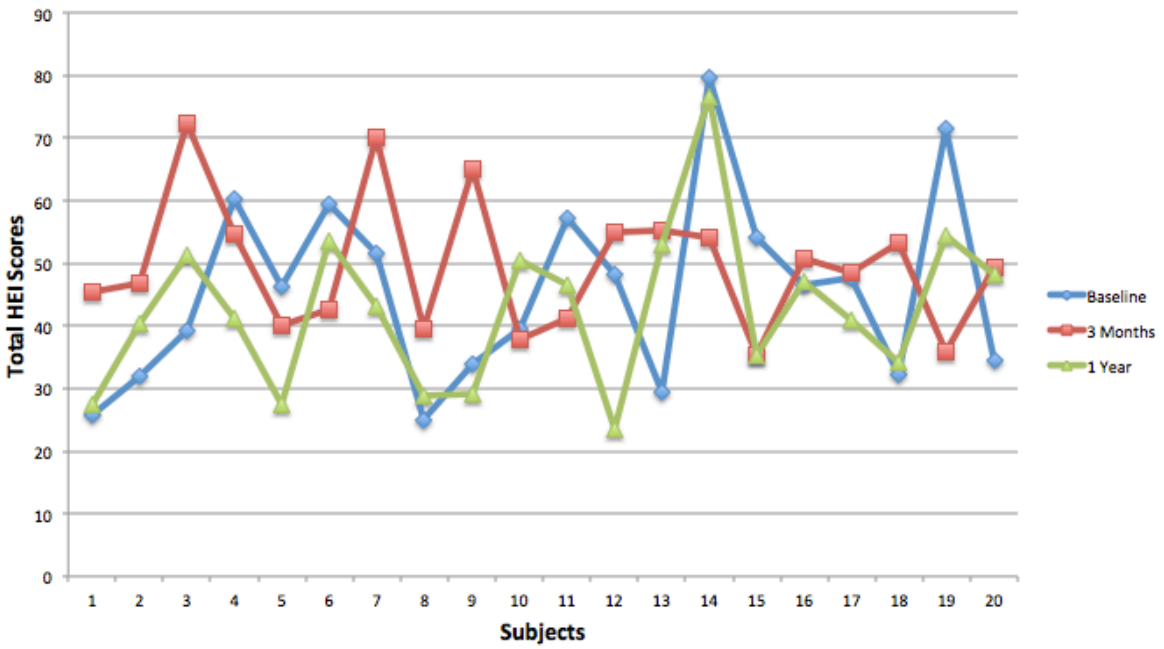


Figure 6. Individual Healthy Eating Index Total Scores

CHAPTER V

Discussion

In this secondary data analysis, a majority of patients were obese at the time of kidney transplantation. One year after transplantation, patients gained an average of 7 kg and their BMI increased by 3 kg/m^2. This finding agrees with previous studies reporting a high occurrence of patients with a weight increase of between 6 and 10 kg and a BMI increase of between 2 and 3.8 kg/m^2 in the first year following renal transplantation (Aksoy, 2016). The effect of weight gain after renal transplantation remains controversial. Weight gain may be attributable to increased appetite from steroid use for immune system suppression, the correction of uremia, fewer dietary restrictions after kidney transplantation, or lack of exercise as patients may fear injury and graft loss (Mantoo et al., 2007). There are opposing views on the impact of weight gain on graft functioning after transplantation. Some researchers suggest weight gain may increase patient survival rates, while others believe otherwise (Torres et al., 2007; Olarte et al., 2009). The definitive cause and influence of recurrent weight gain seen in kidney transplant patients remains unclear which calls for more research in this area.

For the anthropometric and nutrient intake variables, fat intake, potassium intake, body weight, and BMI significantly increased from pre-transplant baseline through post-transplant at 3 months to one year. Increased weight and BMI may be from increased energy intake that may be due to a higher proportion of fat intake. In the pairwise comparison, mean potassium intake only significantly increased from baseline to 3 months but not from baseline to one year or from 3 months to one year. The HEI

component scores reflect this finding for increased potassium intake because patients had a higher HEI score for total fruit and total vegetables at 3 months after transplantation compared to the other time points. This presents a greater intake of fruits and vegetables in patients after transplantation that may be because they are not as restricted of dietary potassium. Therefore, the hypothesis that fat and potassium intake do not increase following renal transplantation was rejected. However, the hypothesis that energy, carbohydrates, protein, phosphorus, calcium, and sodium intake do not increase was accepted.

Energy intake of patients appears to be increasing slightly; however, this observation was not reflected in the correlation between energy intake with weight gain and BMI. During dialysis, patients are in a catabolic state with an elevated basal metabolic rate compared to after a kidney transplant where the basal metabolic rate drops which may be another explanation for weight gain (Bergstrom, Wang, & Lindholm, 1998; Heng et al., 2015). The negative correlation between carbohydrate intake and BMI suggests patients may have been diagnosed with diabetes after kidney transplantation and were instructed to consume fewer carbohydrates to manage their blood glucose levels. New onset diabetes after renal transplantation is a frequent problem following renal transplantation and typically occurs after six months. This may be due to many factors such as older age (90% increase of relative risk in renal transplant patients aged 45–59 and a 160% increase in patients ≥60), being overweight or obese, and immunosuppression, essentially glucocorticoids and calcineurin inhibitors causing insulin resistance and impaired insulin secretion, respectively (Ghisdal et al., 2012). Dietary fat

intake was associated with weight gain and an increase in BMI at one year after kidney transplantation. This suggests diets high in fat may cause weight gain in transplant patients. Weight gain in transplant patients is undesirable because of its association with many health complications such as cardiovascular disease and delayed graft function or graft loss. Dietary advice for fat intake in transplant patients may be necessary (Heaf et al., 2004). Accordingly, the hypothesis that post-transplant weight gain and BMI were not associated with increased fat intake was rejected. However, the hypothesis that weight gain and BMI is not associated with increased energy, protein, and carbohydrate intake was accepted.

At 3 months post-transplantation, patients' consumption of energy and protein increased from baseline but decreased at one year. Nutrient intake may be higher at 3 months because patients may be in the acute post-transplant phase where energy and protein requirements are higher. One year after kidney transplantation, patients are in the maintenance phase where dietary requirements may be lower.

The patients' mean dietary intake was compared to the recommendations. Patients were not meeting the dietary recommendations for mean energy and protein intake for all time points. There was a similar observation in Khoueiry et al.'s study where energy intake of hemodialysis patients was significantly lower than the recommendations by NKF KDOQI (Khoueiry et al., 2011). Patients were eating above the recommendations for phosphorus and sodium for all time points. At 3 months and 1 year post-transplant, they were eating over the recommended levels for potassium. At baseline, patients were

eating below the recommendations for calcium. This is indicative that patients had poor adherence to dietary recommendations.

This study is the first to evaluate dietary quality, as assessed by the Healthy Eating Index, in dialysis and kidney transplant populations. In the evaluation of individual HEI total scores categories, none of the patients had good dietary quality for all time points, and a majority of patients had poor dietary quality. These patients were also consuming fatty acids and empty calories over the national average. As predicted, dietary quality of patients did not significantly improve after transplantation. Patients had a mean HEI total score of 45.75 ± 14.99 at pre-transplant baseline, 49.66 ± 10.70 3 months post-transplant, and 42.59 ± 12.70 one year post-transplant, all of which are less than 51 points indicating poor dietary quality. These findings suggest that renal transplant patients may need improvements in their quality of diet to improve outcomes after renal transplantation. Thus, the null hypothesis, "Quality of diet in comparison to the 2010 Dietary Guidelines for Americans, as assessed by HEI, does not improve following renal transplantation," was accepted because dietary quality of the study subjects did not improve from pre-transplant baseline through post-transplant at 3 months and 1 year.

Several strengths of the study should be noted. The most current version of the ASA24 dietary recall tool was used to assess dietary intake of renal transplant patients. The ASA24 uses the AMPM, which is a validated method in collecting dietary recalls. The AMPM was validated against the doubly labeled water method and was found to accurately estimate total energy and nutrient intake (Blanton et al., 2006). In Kirkpatrick's study comparing the performance of the ASA24 to the AMPM, the ASA24

performed well with an 80% reliability similar to the AMPM's reliability of 83% (Kirkpatrick et al., 2014). The ASA24 2016 also utilizes food composition derived from multiple sources including FNDDS, NHANES's DSD, and FPED. This study was the first to examine dietary quality in renal transplant patients using the HEI. Continuity of care was maintained because all patients were enrolled in the Houston Methodist Renal Transplant program.

Several limitations should be also noted. The original cohort study was performed in a single-center with a small sample size that comprised of mostly male patients that may not be generalizable to other transplant populations. It was an expensive study that involved dual-energy X-ray absorptiometry, BodPod test, and the doubly labeled water method to measure body composition and energy metabolism, explaining the small sample size. There was limited female participation possibly due to personal circumstances since participants spent half a day at Houston Methodist Hospital for data collection. Although dietary recalls are a validated tool to collect dietary intake in different populations, they may occasionally underestimate intake, especially in CKD patients who are more likely to underreport. Also, the study visit days when dietary recalls were obtained may not actually represent the patients' overall diet. Dietary information was lost for six subjects with the conversion from ASA24 2014 to ASA24 2016, and the missing dietary data may have affected final conclusions drawn from this data.

CHAPTER VI

Conclusion

A limited number of studies have been performed assessing nutrient intake of patients undergoing renal transplantation. In this study, an increased intake of fat was significantly associated with weight gain following renal transplantation. Dietary quality, as measured by HEI, was poor both before and after renal transplantation suggesting improvements of food intake is needed. Finally, sample size was relatively small and therefore additional multi-center studies are warranted to better understand the impact of diet quality on renal transplantation outcomes.

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APPENDIX A

TWU Institutional Review Board Study Approval

TWU
TEXAS WOMAN'S UNIVERSITY
DENTON DALLAS HOUSTON

Institutional Review Board
Office of Research
6700 Fannin, Houston, TX 77030
713-794-2480
irb-houston@twu.edu
http://www.twu.edu/irb.html

DATE: November 14, 2017

TO: Dr. Carolyn Moore
Nutrition & Food Sciences - Houston

FROM: Institutional Review Board (IRB) - Houston

Re: Exemption for Association of Changes of Nutrient intake and Dietary Quality with Body Weight following Renal Transplantation (Protocol #: 19803)

The above referenced study has been reviewed by the TWU IRB (operating under FWA00000178) and was determined to be exempt from further review.

If applicable, agency approval letters must be submitted to the IRB upon receipt PRIOR to any data collection at that agency. Because a signed consent form is not required for exempt studies, the filing of signatures of participants with the TWU IRB is not necessary.

Although your protocol has been exempted from further IRB review and your protocol file has been closed, any modifications to this study must be submitted for review to the IRB using the Modification Request Form. Additionally, the IRB must be notified immediately of any adverse events or unanticipated problems. All forms are located on the IRB website. If you have any questions, please contact the TWU IRB.

cc. Ms. Rose Bush, Nutrition & Food Sciences - Houston

APPENDIX B

Baylor College of Medicine Institutional Review Board Study Approval

October 17, 2017

BCM
Baylor College of Medicine

Baylor College of Medicine
Office of Research
One Baylor Plaza, 600D
Houston, Texas 77030
Phone: (713) 798-6970
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WILLIAM E MITCH
BAYLOR COLLEGE OF MEDICINE
MEDICINE: NEPHROLOGY

H-33305 - WEIGHT GAIN AFTER KIDNEY TRANSPLANTATION

APPROVAL VALID FROM 10/11/2017 TO 10/10/2018

Dear Dr. MITCH

The Institutional Review Board for Human Subject Research for Baylor College of Medicine and Affiliated Hospitals (BCM IRB) is pleased to inform you that the research protocol and consent form(s) named above were reviewed by Full Board procedures on 10/11/2017 by Board 3 and is now approved.

The study may not continue after the approval period without additional IRB review and approval for continuation. You will receive an email renewal reminder notice prior to study expiration; however, it is your responsibility to assure that this study is not conducted beyond the expiration date.

Please be aware that only IRB-approved informed consent forms may be used when written informed consent is required.

Any changes in study or informed consent procedure must receive review and approval prior to implementation unless the change is necessary for the safety of subjects. In addition, you must inform the IRB of adverse events encountered during the study or of any new and significant information that may impact a research participants' safety or willingness to continue in your study.

The BCM IRB is organized, operates, and is registered with the United States Office for Human Research Protections according to the regulations codified in the United States Code of Federal Regulations at 45 CFR 46 and 21 CFR 56. The BCM IRB operates under the BCM Federal Wide Assurance No. 00000286, as well as those of hospitals and institutions affiliated with the College.

Sincerely yours,

Association for the Accreditation of Human Research Protection Programs, Inc.
Full AAHRPP Accreditation

LAURA L LOFTIS, M.D.

Institutional Review Board for Baylor College of Medicine and Affiliated Hospitals