4-16-2012

Food Availability as a Determinant of Weight Gain Among Renal Transplant Recipients

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FOOD AVAILABILITY AS A DETERMINANT OF WEIGHT GAIN AMONG RENAL TRANSPLANT RECIPIENTS

by

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A Thesis

Submitted in Partial Fulfillment of the

Requirements for the Degree of

Master of Public Health

Major: Public Health

The University of Memphis

May, 2012

Abstract

Bloodworth, Robin Frances. MPH. The University of Memphis. May, 2012. Food Availability as a Determinant of Weight Gain Among Renal Transplant Recipients. Major Professor: Kenneth Ward.

Renal transplantation is the treatment of choice for End Stage Renal Disease (ESRD), but one of the risks associated with this treatment is excessive weight gain. Several potential causes of this increased risk have been evaluated, but environmental factors have not yet been explored. This study used Geographic Information Systems (GIS) to evaluate the effect of number and ratio (number of one compared to all) of food sources (fast food restaurants, convenience stores, and grocery stores) within a one, two, and three mile buffer around transplant recipients' residences (n = 484) on BMI change during the first year post-transplant. Multiple linear regression found that the only significant effect was seen in fast food ratio within a three-mile buffer of residence (p = .024). Future research is needed with larger sample sizes to explore the potential of effects food environment on post-renal transplant weight gain more accurately.

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Introduction

End Stage Renal Disease: Causes and Effects

The kidneys' main function is to rid the body of waste and excess fluid, but they also serve several other purposes, such as controlling electrolyte and glucose balance, producing hormones that regulate blood pressure, maintaining bone health, and producing red blood cells (American Kidney Fund, 2008). Differing from chronic kidney disease (CKD), which occurs when the kidneys are damaged and perform at a less than satisfactory rate, End Stage Renal Disease (ESRD) occurs when damage causes the kidneys to function at less than 10% of normal capacity (Silberg, 2010). At this point, without the intervention of dialysis or transplant, death will occur (Silberg, 2010).

End stage renal disease is increasing at an alarming rate and has become a major public health issue in the United States. The United States Renal Data System (USRDS) works with Centers for Medicare and Medicaid Services and United Network for Organ Sharing (UNOS) to compile data sets from across the nation in order to portray an accurate representation of ESRD in the United States (2003). The USRDS Report for 2003 found that incidence per million people of ESRD in the United States increased from under 100 in 1981 to over 300 in 2003 (2003). Reasons for this increase in ESRD are unclear, but it is not due entirely to overall increases in chronic kidney disease. A birth cohort analysis in the U.S., linking nationally representative data from the Second and Third National Health and Nutrition Examination Surveys (NHANES) with data from the USRDS, found that incidence of progression from CKD to ESRD

increased from 9 per 1,000 between 1978-1983 to 16 per 1,000 between 1991-1996 (*RR* = 1.7, *95% CI* = 1.1-2.7) (Hsu, Vittinghoff, Lin, & Shlipak, 2004).

The two main causes of ESRD, and also CKD, are diabetes and hypertension (Silberg, 2010). Diabetes affects kidney function in two ways (National Kidney Foundation, 2011a). First, diabetes damages small blood vessels throughout the body, and when this damage occurs to blood vessels in the kidneys, they are no longer able to rid the body of waste properly (National Kidney Foundation, 2011a). Also, diabetes can lead to neuropathy, which can result in difficulty emptying the bladder (National Kidney Foundation, 2011a). This can damage the kidneys by creating excess pressure from the bladder, and it can increase risk for infection by providing bacteria an opportunity to flourish in the urine (National Kidney Foundation, 2011a). Hypertension and ESRD are interrelated. Hypertension can cause damage to blood vessels as well as filtration units in the kidneys, leading to decreased function (National Kidney Foundation, 2011b). Decreased kidney function can also cause hypertension, perpetuating a destructive cycle (National Kidney Foundation, 2011b).

In addition to hypertension, ESRD causes a wide array of other complications that affect quality of life for its sufferers, including internal bleeding, fluid retention, dementia, peripheral nerve damage, cardiovascular complications, and increased risk of infection (Silberg, 2010). The 2003 USRDS report found that patients with even mild to moderate renal failure are at increased risk of death due to ischemic heart disease.

Renal Transplantation as a Treatment for ESRD

There are three treatment options for ESRD: hemodialysis, peritoneal dialysis, and renal transplantation. During hemodialysis, a patient is hooked up to a machine that filters out impurities and waste in the blood, as kidneys would do, then returns it to the body (Silberg, 2011). Peritoneal dialysis consists of implanting a permanent tube in the patient's abdomen, through which fluids are pushed then flushed, using the patient's own peritoneum to flush out impurities from the blood that have been exchanged to the fluids (Silberg, 2011). In renal transplantation, a new kidney, either from a living or deceased donor, is implanted into the patient's abdominal cavity (Miller, 2011).

Renal transplantation is the preferred option in treating ESRD (Neipp, Jackobs, & Klempnauer, 2009). Not only does it extent survival time, but it also improves quality of life (Neipp et al., 2009) and psychological functioning (Sayin, Mutluay, & Sindel, 2007) for recipients. Long-term survival of ESRD patients who undergo renal transplantation is much better than that of ESRD patients who undergo dialysis (Briggs, 2001). For example, in the largest prospective study conducted to date on this topic, ESRD patients who received renal transplantation (n = 46,164) had a 68% lower long-term (three to four years) mortality risk compared to ESRD patients on a transplantation wait-list matched for follow-up time (n = 23,275) (Wolfe et al., 1999). In a population-based prospective study of all Michigan residents 65 years of age or younger, who began treatment for ESRD between 1984 and 1989 (n = 5020), those who underwent transplantation (n = 799) had greater long-term survival (up to five

years) than similar patients who received dialysis (*RR* = 0.36, *p* < .001) (Port, Wolfe, Mauger, Berling, & Jiang, 1993). Greater long-term survival also was documented in another prospective study comparing mortality rates between ESRD patients on the wait list for a transplant (*n* = 434) and renal transplant recipients (*n* = 722), which found that crude mortality rates between the two groups were 5.0% and 3.4%, respectively (Rabbat, Thorpe, Russell, & Churchill, 2000).

In addition to increased survival, transplantation improves health-related quality of life relative to dialysis. In several cross-sectional studies, transplant recipients report improved quality of life on several indices, relative to wait-list candidates receiving dialysis (Griva et al., 2011; Maglakelidze, Pantsulaia, Tchokhonelidze, Managadze, & Chkhotua, 2011; Neipp et al., 2006). For example, a recent study administered three widely-used, psychometrically sound instruments, the Short Form-36 (SF-36), Giessen Subjective Complaints List (GBB-24), and Zerssen's Mood Scale (Bf-S) to 120 hemodialysis patients, 43 peritoneal dialysis patients, nine transplant recipients that lost their grafts and went back on dialysis, 48 transplant recipients, and 120 healthy controls (Maglakelidze et al., 2011). In addition, a community control group was generated using probability-based methods to generate a sample that was representative of the general population (Maglakelidze et al., 2011). Groups were matched on the basis of age, sex, race, and other major covariates (not specified) (Maglakelidze et al., 2011). Transplant recipients reported better functioning than hemodialysis and peritoneal dialysis patients on all indices,

including the eight sub-scales of the SF-36 (physical functioning, social functioning, physical role, bodily pain, mental health, emotional role, vitality, and general health), the five sub-scales of the GBB-24 (total score, fatigue, limb pain, gastric, and cardiac functioning), and overall mood (from the Bf-S) (Maglakelidze et al., 2011). On the majority of these sub-scales, transplant recipients reported as good, or better, quality of life than healthy controls (Maglakelidze et al., 2011).

There is evidence that quality of life improvements after transplantation are sustained long term. In a prospective cohort study of 102 renal transplant recipients followed for six years post-transplant, several indices of emotional health-related quality of life, from the SF-36, continued to improve over time (Griva et al., 2011). Further supporting this finding is a retrospective cohort study of 139 renal transplant recipients assessed 15 years post-transplant (Neipp et al., 2006). Recipients were found to score in the satisfactory range, and similar to that of the healthy population, on several sub-scales of the SF-36, including physical role, social functioning, emotional role, and mental health. Thus, there is compelling evidence that transplantation improves several domains of physical and emotional functioning, often to levels similar to that of the healthy general population.

Transplantation also reduces depression and anxiety. Several cross-sectional studies have shown that transplant recipients are less depressed and anxious than dialysis patients (Akman, Ozdemir, Sezer, Micozkadioglu, & Haberal, 2004; Alavi, Aliakbarzadeh, & Sharifi, 2009; Haq, Zainulabdin, Naqvi, Rizvi, & Ahmed, 1991; Karaminia et al., 2007; Panagopoulou, Hardalias, Berati,

& Fourtounas, 2009; Sayin et al., 2007). For example, a cross-sectional study of 100 transplant recipients and 63 hemodialysis patients found that transplant recipients showed lower levels of both anxiety and depression (Alavi et al., 2009). Anxiety and depression were measured using Symptom Checklist-90 (SCL90), which has subscales of depression and anxiety (Alavi et al., 2009). Multiple regression analysis was conducted to identify which variables (age, gender, marital status, education level, predisposing diseases, duration, and kind of therapy) affected the incidence of depression, anxiety, and other quality of life measures (Alavi et al., 2009). Significantly fewer transplant recipients than and hemodialysis patients were depressed (39% and 65.3%, respectively ($p < .001$) or anxious (40.6% and 51.6%, respectively, $p = .03$) (Alavi et al., 2009).

In sum, renal transplantation has become the treatment of choice for ESRD, compared to dialysis treatments, due to its superior outcomes related to survival, quality of life, and psychological functioning.

Adverse Consequences of Renal Transplantation

Despite the clear benefits of renal transplantation, there are some serious adverse consequences. As with any surgery, risk of wound infection is an issue (Miller, 2011). Also, there is the risk that the body will reject the kidney (Miller, 2011). In order to reduce the risk of these two negative outcomes, recipients are often put on antibiotics and immunosuppressant medications, which can cause many unpleasant side effects (Miller, 2011). As mentioned above, renal function is closely associated with diabetes and hypertension. It is not uncommon for one, if not both, of these conditions to develop or become exacerbated post-

transplant (National Kidney Foundation, 2011c). Lastly, overweight and obesity are also common side effects of transplant (National Kidney Foundation, 2011c).

Effect of Obesity on Renal Transplant Outcomes

Renal transplant recipients have a tendency to gain a significant amount of weight post-transplant. Among a sample of Methodist University Transplant Institute (MUTI) recipients, an average 14 pound weight gain occurred during the first year post-transplant, with some gaining as much as 70 pounds (Cashion et al., 2007). In a prospective study of 418 renal transplant recipients in Poland, the percentage of overweight or obese recipients increased dramatically from 38% at time of transplant to 65% 4.5 years post-transplant (Jezior et al., 2007). Risk was greatest for severe weight gain, whereas the percentage of recipients classified as overweight ($25 < BMI \leq 30$) increased from 32% to 38%, the percentage of obese ($30 < BMI \leq 35$) nearly quadrupled (4% to 15%), and risk of morbid obesity ($BMI > 35$) nearly doubled (1.4% to 2.9%) (Jezior et al., 2007). Similar findings were reported in a retrospective chart review study of 115 adult renal transplant recipients, where prevalence of overweight doubled from 21% to 43% from time of transplant to one year post-transplant, and 57% of all recipients experienced a weight gain greater than 10% (Johnson et al., 1993). Another retrospective study of 165 renal transplant recipients found that average BMI increased from 25.3 (normal range) at time of transplant to 33.0 (obese range) at one year post-transplant and 36.2 at 5 years post-transplant (Thoma, Grover, & Shoker, 2006), indicating that risk of excessive weight gain remains a problem relatively long-term after transplantation.

Post-transplant obesity, as well as excessive post-transplant weight gain, increases mortality. A retrospective analysis of 51,927 renal transplant recipients from the USRDS found obesity was correlated with graft loss and recipient death (Meier-Kriesche, Arndorfer, & Kaplan, 2002). Similarly, el-Agroudy, Wafa, Gheith, Shehab el-Dien, and Ghoneim (2004) found that obese recipients trended more towards graft loss and decreased survival rate five and 10 years post-transplant. A study examining predictors of survival among adult renal transplant recipients one and two years post-transplant (n = 3899 and n = 3419) found that weight gain of more than 20% during the first year post-transplant and more than 10% during the second year post-transplant were associated with increased risk of death (Chang & McDonald, 2008). A prospective study of 292 renal transplant recipients, measuring both anthropometric and biological parameters at time of transplant and one year post-transplant, further supported these findings, showing that post-transplant weight gain significantly reduces graft survival, especially in those individuals who increase their body weight by more than 5% (Ducloux, Kazory, Simula-Faivre, & Chalopin, 2005).

The effect of post-transplant obesity and weight gain on mortality is mediated through a number of pathophysiological conditions, especially hypertension and diabetes. Obese transplant recipients (Body Mass Index [BMI] $\geq$ 30) are more likely to develop hypertension, diabetes, and ischemic heart disease (el-Agroudy et al., 2004). Weight gain after transplant increases serum cholesterol and triglyceride levels significantly (Johnson et al., 1993). Obesity also is a risk factor for the development of post-transplant diabetes mellitus

(PTDM) (Baltar et al., 2005) and metabolic syndrome (Luan, Langewisch, & Ojo, 2010), which increase mortality risk (Cosio et al., 2002; Demirci et al., 2010; Goldsmith & Pietrangeli, 2010; Gonzalez-Posada et al., 2006; Porrini et al., 2006; Salvadori, Bertoni, Rosati, & Zanazzi, 2003).

Previously, it was assumed that post-transplant weight gain was due to the effects of corticosteroids, used chronically in transplant recipients for their immunosuppressant effects, to prevent graft rejection. A common side effect of chronic steroid use is weight gain (Manson, Brown, Cerulli, & Vidaurre, 2009). There is recent evidence, however, that steroid use is not typically responsible for the excessive weight gain observed in renal transplant recipients. A retrospective study of 123 renal transplant recipients found that neither cumulative nor maintenance-steroid dose post-transplant was associated with one year post-renal transplant weight gain (van den Ham, Kooman, Christiaans, & van Hooff, 2000). Another study found that cumulative steroid dose during the first five years post-transplant was not associated with weight gain during this time interval (Johnson et al., 1993).

Several studies have reported sociodemographic correlates of post renal-transplant weight gain, including race, gender, age, and socioeconomic status (Baum et al., 2002; Cashion et al., 2007; Clunk, Lin, & Curtis, 2001; Diaz et al., 2005; Jezior et al., 2007; Johnson et al., 1993). In the U.S., several studies have reported that African Americans are at greater risk of post-transplant weight gain than Caucasians (Baum et al., 2002; Cashion et al., 2007; Clunk et al., 2001; Johnson et al., 1993). For example, in a secondary data analysis of 506 renal

transplant recipients transplanted between 1983 and 1998, African Americans were found to be affected by obesity more severely than Caucasians, where 39% and 27% were obese one year post-transplant (Baum et al., 2002).

Several studies also have found that women are more likely than men to gain weight post-transplant (Cashion et al., 2007; Clunk et al., 2001; Jezior et al., 2007; Johnson et al., 1993). For example, a retrospective review of 977 renal transplant recipients found that women gained more weight than men over one year post-transplant (118.4% vs. 112.15 of initial body weight at transplantation, $p = .0001$) (Clunk et al., 2001).

Younger recipients are at higher risk for weight gain than older recipients (Clunk et al., 2001; Diaz et al., 2005; Johnson et al., 1993). For example, a retrospective chart review of 115 renal transplant recipients found an inverse relationship between age and weight gain, where older recipients (> 50 years) gained the least amount of weight at one year post-transplant, and younger recipients (18-29 years) gained the most amount of weight one year post-transplant (8.3% vs. 13.3%, $p = .047$) (Johnson et al., 1993).

Socioeconomic status also is inversely associated with weight gain. In a retrospective review of 977 transplant recipients in Alabama, median yearly household income was negatively associated with risk of weight gain during the first year post-transplant (Clunk et al., 2001). Univariate analysis showed low-income recipients ($n = 141$) on average gained more weight than medium- ($n = 726$) and high-income ($n = 70$) recipients (120.3%, 114.1%, 110.6%, of body weight at transplantation, $p = .0001$) (Clunk et al., 2001). In sum, transplant

recipients in the U.S. at most risk of weight gain are women, African Americans, younger individuals, and those of lower socioeconomic status.

In the general population, physical inactivity and excessive energy intake are strongly correlated with weight gain (Fogelholm & Kukkonen-Harjula, 2000; Hankinson et al., 2010; McCrory, Suen, & Roberts, 2002; Waller, Kaprio, & Kujala, 2008). These relationships also have been demonstrated in the renal transplant population, albeit relatively few studies have been conducted to date. A systematic review of observational and interventional studies among renal transplant recipients *(n* = 21, which includes six observational and 15 interventions) found that habitual physical activity was strongly inversely related to body fat (Macdonald, Kirkman, & Jibani, 2009). Two non-randomized intervention studies found support for the efficacy of early dietary intervention to reduce post-transplant weight gain (Moreau et al., 2006; Patel, 1998). Although shedding insight into the possible causes of weight gain post-transplant, these individual-level factors are not adequate to fully explain post-transplant weight gain.

Within the socio-ecological framework, health behavior is influenced by several levels of determinants (National Cancer Institute, 2005). These include intrapersonal factors, such as attitudes and beliefs; interpersonal factors, such as social and familial relationships; institutional or organizational factors, such as rules and informal regulations; community factors, such as societal norms; and public policy factors, such as local, state, or federal policies (National Cancer Instute, 2005). Therefore, weight gain can be influenced by a variety of different

factors, whether they are those of individual behavior, environmental influence, or societal level factors.

A major gap in the literature is an examination of how environmental factors affect weight gain after renal transplantation. A sizable body of literature, however, has evaluated environmental influences on obesity in the general population. These factors can be broadly grouped as environmental characteristics, access to physical activity, and access to food.

GIS and Obesity

Geographic Information Systems (GIS) technology has been widely used to assess the associations between environmental factors and obesity. GIS refers to a system that is used to store and manipulate geographical information on a computer. Because environmental influences on obesity, assessed using GIS methodology, are likely to be relevant to renal transplant recipients, they will be reviewed here.

Environmental characteristics. Several environmental characteristics that have potential effects on obesity can be measured by GIS technology. These include land use, walkability, neighborhood greenness, and neighborhood safety.

***Land use*.** Land use refers to how land is used in a given area, such as a neighborhood, which can influence how much exercise individuals in the area get, as well as what types of resources they have access to. For example, a neighborhood with a high level of mixed land use, meaning there are various different types of buildings in the area (both commercial and residential), would

be more conducive to residents walking to the store instead of driving, etc. A study of potential childhood obesity research methods in 412 Chicago public schools found that GIS could be used to accurately characterize distinct neighborhoods in regards to land mix use, allowing this methodology to be used successfully to measure obesity risk (Zhang, Christoffel, Mason, & Liu, 2006). A cross-sectional study used number of residents walking to work and median age of housing as land use indicators in Utah (Smith et al., 2008). Older neighborhoods tend to be built for better walking and newer neighborhoods tend to focus on the flow of traffic (Smith et al., 2008). It was determined that these two measures had a significant effect on obesity risk (Smith et al., 2008). Specifically, doubling the amount of people walking to work resulted in a 10% decrease in obesity risk, and adding a decade to the median average age of the neighborhood decreased obesity risk by 8% for women and 13% for men (Smith et al., 2008). In a study of Latino residences using the same data set, it was found that mixed land use is a factor that can be modified to decrease obesity rates of this minority population (Wen & Maloney, 2011). Evaluating adiposity in 1221 older adults (aged 50-75) in Portland, OR, a cross-sectional study found that a 10% increase in land use mix led to a 25% reduction in the prevalence of overweight/obesity (Li et al., 2008). In a review of the built environment in influencing physical activity in children and adolescents, mixed land use was positively associated with greater physical activity levels (Giles-Corti, Kelty, Zubrick, & Villanueva, 2009). However, a study using 5000 randomly chosen licensed drivers in Utah found that mixed land use was not associated with

healthier weight (Brown et al., 2009). Furthermore, a study examining 546 older adults in Portland, OR found that the built environment was not associated with walking level; however, of those adults who did walk, they were more likely to do so with an increase in mixed land use (Nagel, Carlson, Bosworth, & Michael, 2008). Thus, GIS technology has been widely used to evaluate land use characteristics and has generally found associations with obesity risk, though there are some inconsistencies in the literature.

Walkability. Walkability is a measure of how "walking-friendly" a neighborhood is, with higher levels of walkability characterized by more sidewalks, footpaths, access to buildings, safety, and other pedestrian-friendly features. Neighborhoods that are more "walkable" are associated with greater level of physical activity among residents. In an observational study of 2199 adults (ages 20-65) taken from a sample of 32 neighborhoods in Seattle, WA and Baltimore, MD, moderate physical activity was associated with neighborhoods with higher walkability (Sallis et al., 2009). Higher walkability was also found to be linked to higher levels of physical activity in a cross-sectional study of 1221 older adults in 120 neighborhoods in Portland, OR (Li et al., 2008). A cross-sectional study of 577 older adults in 56 neighborhoods in Portland, OR found that the level of walkability was positively associated with walking activity levels on a neighborhood scale (Li, Fisher, Brownson, & Bosworth, 2005). In a study examining 546 older adults in Portland, OR, the built environment, which included such measures as percentage of high-, medium-, and low-volume streets; percentage of sidewalk coverage; number of intersections, bus lines,

commercial establishments; and distance to the nearest park, was not associated with walking level; however, of those adults who did walk, they were more likely to do so with an increase in neighborhood walkability (Nagel et al., 2008).

Not only is walkability associated with greater physical activity, but also with lower body weight and obesity. In an observational study conducted with 1145 residents of 120 neighborhoods in Portland, OR, a one year change in body weight of -1.2kg ($p < .05$) among older adults (aged 50-75) was associated with higher-walkability neighborhoods (Li, et al., 2009). Examining the same population, Zick et al. (2009) found that neighborhoods with higher walkability were associated with lower obesity risk, but the strength of this effect was dependent on neighborhood income, with stronger effects in low-income neighborhoods. A cross sectional study examining overweight among 501 preschool aged children (262 female, 239 male) in Canada found that for girls, the odds of being overweight or obese were lower if they lived in a neighborhood with higher walkability (*OR* = .78, *95%CI* .66-.91), but no such effect was found in boys (Spence, Cutumisu, Edwards, & Evans, 2008). Using 5000 randomly chosen licensed drivers in Salt Lake City County, Utah, Brown et al. (2009) found that walkability was associated with healthier weight. In a status report summarizing current reviews, the general consensus was that people who live in neighborhoods with higher walkability are less likely to be overweight or obese (Sallis & Glanz, 2009). Hence, the literature shows a negative association between walkability and obesity.

Neighborhood greenness. Greenness is a measure of how much open/green space, such as parks, are in an area. In Massachusetts, a cross-sectional study of 21008 children found that the amount of open (green) space was inversely associated with BMI (Oreskovic, Winickoff, Kuhlthau, Romm, & Perrin, 2009). A similar study (*n* = 6680) found that lower-income towns had a lower density of open (green) space and a higher level of obesity (Oreskovic, Kuhlthau, Romm, & Perrin, 2009). In a secondary data analysis of 7334 children (ages 3-18) obtained from Indiana pediatric clinics, increased neighborhood vegetation (greenness) produced a decreased risk for overweight, but only for children who lived in areas with a high population density (Liu, Wilson, Qi, & Ying, 2007). A cross-sectional study of 1221 older adults in 120 neighborhoods in Portland, OR found that higher levels of neighborhood greenness were found to be linked to higher levels of physical activity (Li et al., 2008). Examining the walking activities of 577 older adults in 56 Portland area neighborhoods, Li et al. (2005) found that amount of greenness was positively associated with walking activity levels on a neighborhood scale. In sum, the general consensus of the literature is that neighborhood greenness decreases obesity.

Neighborhood safety. Lack of safety in an area can discourage physical activity, which can lead to higher obesity rates in the area. In a review of 45 studies evaluating the effects of the built environment on obesity in disadvantaged populations, neighborhood safety was found to have a strong effect on obesity (Lovasi, Hutson, Guerra, & Neckerman, 2009). Higher levels of neighborhood safety were found to increase individual-level walking activity in a

study of 577 older adults in 56 Portland, OR neighborhoods (Li et al., 2005). Examining obesity levels in women with young children (*n* = 2445), a cross-sectional study found that obesity rates increased across neighborhood safety tertiles (safest to least safe: 37%, 41%, and 46%, respectively) after controlling for sociodemographic factors, smoking, depression, and television time (Burdette, Wadden, & Whitaker, 2006). However, a cross-sectional study of obesity rates in children from 20 large U.S. cities found that neighborhood safety, measured in tertiles, was not significantly associated with obesity prevalence (safest to least safe: 20%, 17%, and 18%, respectively) (Burdette & Whitaker, 2005). Although not all literature was consistent, neighborhood safety has been shown to affect physical activity levels and obesity rates.

Access to physical activity resources. The amount of physical activity resources, such as recreational facilities, in an individual's area can influence physical activity levels. Examining the effects of the built environment on obesity in disadvantaged populations, a review of 45 studies found that amount of places to exercise was a factor that affected obesity in these populations (Lovasi et al., 2009). Summarizing current reviews, a status report found that people who live in neighborhoods with more access to recreational facilities are less likely to be overweight or obese (Sallis & Glanz, 2009). Examining how disparities in access to physical activity resources can affect adolescent overweight, Gordon-Larsen, Nelson, Page, and Popkin (2006) used GIS technology to link national and satellite data to residential location of 20,745 adolescents from the National Longitudinal Study of Adolescent Health. Lower SES neighborhoods were less

likely to have access to recreational facilities, and an increased number of facilities was associated with a decrease in overweight (Gordon-Larsen et al., 2006). Access to recreational facilities was found to be positively associated with greater physical activity levels in a review of the built environment in influencing physical activity in children and adolescents (Giles-Corti et al., 2009). Individual-level walking activity was found to be positively associated with number of accessible recreational facilities in a cross-sectional study of 577 older adults in 56 neighborhoods in Portland, OR (Li et al., 2005). However, in an examination of the contributors to overweight in 7020 low-income preschool children in Cincinnati, OH, Burdette and Whitaker (2004) found that distance to playground had no effect. Ergo, more access to physical activity resources decreases obesity risk, although there are some inconsistencies in the literature.

Access to food. There is an abundance of research today showing that food availability has a direct impact on individuals' weight. During the last few decades, there has been a dramatic shift in where and what people eat. Fast foods provided only 1% of per capita total fat intake in the U.S. diet in 1965 compared to 11% in 1996 (Popkin, Siega-Riz, Haines, & Jahns, 2001). Additionally, the amount spent on fast food in the United States has increased from $6 billion in 1970 to more than $110 billion in 2000 (Schlosser, 2001). One of the main factors in deciding what to eat is convenience (Frazao, 1999). As such, the food availability in an individual's personal environment, as in what is found nearby their home, affects what people eat and ultimately their health.

Access to fast food restaurants. Greater access to fast food restaurants has been shown to increase obesity levels. Several reviews have reported positive associations between access to fast food restaurants and weight or obesity (Giskes, van Lenthe, Avendano-Pabon, & Brug, 2011; Holsten, 2009; Larson, Story, & Nelson, 2009; Rahman, Cushing, & Jackson, 2011; Sallis & Glanz, 2009). Access to fast food restaurants can affect obesity by influencing diet. In a secondary data analysis of 15 years' worth of data from the Coronary Artery Risk Development in Young Adults (CARDIA) study of 5115 young adults (ages 18-30 years at baseline), fast food consumption was related to its availability, but only for low-income individuals (Boone-Heinonen et al., 2011). Timperio et al. (2008) found that children (*n* = 461, ages 10-12) in Australia were less likely to eat fruit two or more times per day as a function of having more fast food outlets (*OR* = .82, *95%CI* .67-.99) in their neighborhood. Further, they were less likely to consume vegetables three or more times per day the further away they lived from a fast food restaurant (*OR* = 1.19, *95%CI* 1.06-1.35) (Timperio et al., 2008).

In addition to healthy diet, the effect of access to fast food restaurants on obesity can also be assessed by directly measuring BMI and/or obesity rates. Linking self-report BMI data from a representative sample of more than 700,000 U.S. adults to restaurant data from the U.S. Economic Census, a higher quantity of fast food restaurants and higher ratio of fast food to full service restaurants in an individual's county of residence was associated with higher individual BMI (Mehta & Chang, 2008). This study also found that the ratio of fast food

restaurants to other food outlets, in comparison to other types of food eaten away from home, was most predictive of higher BMI (Mehta & Chang, 2008). Similar results were reported in a study of BMI and food availability among more than 2,000 adults from 63 neighborhoods in Los Angeles County, CA, where the association of neighborhood density of fast food restaurants and weight gain was especially strong among individuals dependent on their immediate environment for their food choices, due to lack of transportation (Inagami, Cohen, Brown, & Asch, 2009). This is especially remarkable in that car owners typically have higher BMIs than non-car owners, due to lower levels of physical activity. A secondary data analysis of the Stanford Heart Disease Prevention Program found that the percentage of obese women (*n* = 3145) rose by 28%, and the percentage of obese men (*n* = 2625) increased by 24%, due to large increases in the number and density of fast food restaurants from 1981 to 1990 (Wang, Cubbin, Ahn, & Winkleby, 2008). Exposure to fast food restaurants in this study was defined as the quantity and density (quantity divided by area in miles^2) in each neighborhood, and regression model and trend analyses were conducted to examine the relationship between fast food exposure and increase in obesity rates (Wang et al., 2008). In a cross-sectional study of fast food restaurants in 120 neighborhoods in Portland, OR, residents in neighborhoods defined as having a high density of fast food restaurants had a 1.878 odds (*95%CI* 1.006-3.496) of being obese than residents of low fast food density neighborhoods (Li, Harmer, Cardinal, Bosworth, & Johnson-Shelton, 2009). Using the same data set, it was determined that a 1-*SD* increase in density of fast food outlets would

lead to a 7% increase in overweight/obesity (Li et al., 2008). An observational study conducted with 1145 residents of 120 neighborhoods in the Portland, OR area found that a one year change in body weight of 1.4kg ($p < .05$) among older adults (aged 50-75) was associated with higher density of fast food outlets (Li, et al., 2009). A cross-sectional examination of 21008 children in Massachusetts found that the closer the nearest fast food restaurant, the higher the BMI, and a higher density of fast food restaurants in a neighborhood was positively associated with BMI (Oreskovic et al., 2009). However, in an examination of the contributors to overweight in 7020 low-income preschool children in Cincinnati, it was found that distance to nearest fast food restaurant had no effect on obesity (Burdette & Whitaker, 2004). In sum, there is a strong correlation between environmental food access and weight gain, particularly in regards to fast food availability.

Access to convenience stores. An additional environmental contributor to obesity is a relatively high density of convenience stores, rather than supermarkets. Several reviews have shown a positive association with convenience store access and obesity (Lovasi et al., 2009; Rahman et al., 2011; Larson et al., 2009; Sallis & Glanz, 2009). Convenience stores have more unhealthy food options and less healthy food options, compared to grocery stores (Glanz, Sallis, Saelens, & Frank, 2007). The ratio of convenience to grocery stores in neighborhoods is related to socioeconomic status. Lower SES neighborhoods tend to have a greater amount of convenience stores and a lower

amount of grocery stores compared to higher SES neighborhoods (Gibson, 2011).

One way access to convenience stores affects obesity is by its effect on diet. In an analysis of the effect of travel times to food sources on fruit and vegetable consumption, Pearce, Hiscock, Blakely, and Witten (2008) found that people in neighborhoods with the highest level of convenience stores had a 25% lower odds (O*R* = .75, *95%CI* = .6-.93) of attaining the recommended daily vegetable intake. A cross-sectional study of the eating habits of 1721 children (ages 9-10) in England found that density of super markets in the neighborhood was associated with increased vegetable intake (.31 portions/week, $p < .05$), and density of convenience stores in the neighborhood was associated with more intake of several unhealthy foods (Skidmore et al., 2010) A study of children (*n* = 461, ages 10-12) in Australia found that having more convenience stores (*OR* = .84, *95%CI* = .73-.98) in their neighborhood resulted in children being less likely to eat fruit two or more times per day (Timperio et al., 2008). They were also less likely to consume vegetables three or more times per day as a function of the density of convenience stores (*OR* = .84, *95%CI* = .74-.98) in their neighborhood (Timperio et al., 2008).

Several studies have reported a direct association between convenience store access and body weight or obesity. Looking at food access in the environment in an attempt to assess childhood obesity (*n* = 1669), Jennings et al. (2011) found that living in a neighborhood with poor access to healthy food choices (such as one with high levels of convenience stores) was associated with

higher body weight. Conversely, lower body weight was associated with more access to healthy food options in the neighborhood (Jennings et al., 2011). In a recent longitudinal study that combined individual-level data on adults from the 1998 through 2004 survey years of the National Longitudinal Survey of Youth 1979 with zip code-level data on the neighborhood food environment, it was found that in urban areas, a higher neighborhood density of small grocery stores, or convenience stores, was directly and significantly associated with obesity and BMI (Gibson, 2011). Secondary data analysis of the Stanford Heart Disease Prevention Program found that large increases in the number and density (number divided by neighborhood area in miles^2) of convenience stores from 1981 to 1990 was associated with a 28% increase in obesity among women, and a 24% increase among men (Wang et al., 2008). Examining a population in Utah, an association was found between access to convenience stores and obesity rate, but the strength of this effect was dependent on neighborhood income, with stronger effects in low-income neighborhoods (Zick et al., 2009). A three year longitudinal secondary analysis of young girls taken from a nation-wide survey found that the higher the concentration of convenience stores around young girls' homes, the more likely they were to be overweight or obese (Leung et al., 2011). Thus, it has been demonstrated by several studies and reviews that there is an obesogenic effect seen in those who live in areas with higher access to convenience stores.

Access to grocery stores. There have been several reviews conducted that found a negative association between access to grocery stores and obesity

(Giskes et al., 2011; Rahman et al., 2011; Sallis & Glanz, 2009; Lovasi et al., 2009; Larson et al., 2009). Grocery store access has been found to vary by socieoeconomic status. A study examining distance to grocery stores in 68 low income neighborhoods in California found that 31% of neighborhoods had no grocery stores within census tract boundaries (Ghirardelli, Quinn, & Foerster, 2010). In an assessment of healthy versus unhealthy food options conducted in Melbourne, Australia, residents of neighborhoods with higher socioeconomic status were found to live in closer proximity to supermarkets, whereas residents of neighborhoods with lower socioeconomic status lived in closer proximity to fast food outlets (Burns & Inglis, 2007).

One way access to grocery stores has been shown to be linked to obesity is by means of a healthier diet. A cross-sectional study of the eating habits of 1721 children (ages 9-10) in England found that density of super markets in the neighborhood was associated with increased vegetable intake (.31 portions/week, $p < .05$), and density of convenience stores in the neighborhood was associated with more intake of several unhealthy foods (Skidmore et al., 2010). The further a child lived from a supermarket was found to make them less likely to consume vegetables three or more times per day in Australia (*OR* = 1.27, *95%CI* = 1.07-1.51) (*n* = 461, ages 10-12) (Timperio et al., 2008).

Access to grocery stores has also been shown to have a direct effect on obesity. Examining a population in Utah, an association was found between access to grocery stores and obesity rate, but the strength of this effect was dependent on neighborhood income, with stronger effects in low-income

neighborhoods (Zick et al., 2009). Analyzing data from Indiana pediatric clinics on 7334 children (ages 3-18), Liu et al. (2007) found that decreased access to grocery stores produced an increased risk for overweight, but only for children who lived in areas with a low population density.

In sum, and consistent with the socioecological model, environmental factors play an important role in obesity. Specifically, living in neighborhoods in which access to unhealthy foods, via fast food restaurants and convenience stories, is much greater than access to healthier foods, via grocery stores and supermarkets, substantially increases one's risk of obesity. These associations have been found in the general U.S. population, but no research has been conducted to determine whether these environmental conditions affect weight gain among a particularly high-risk sub-population, renal transplant recipients.

Implications for Present Research

Although there are studies that evaluate the potential demographic factors that contribute to weight gain post-renal transplant, the literature is lacking in studies that examine potential environmental factors that could have an effect on post-transplant weight gain.

Food availability has been shown to directly affect weight gain in the normal population. However, there are no studies examining whether food availability is associated with weight gain among renal transplant recipients. The current study aims to explore how food availability affects BMI change during the first year post-renal transplant.

Methods

Design

This was a retrospective cohort study of renal transplant recipients from Methodist University Transplant Institute (MUTI) in Memphis, TN. Both clinical data, from the recipients' medical records, and publicly available environmental data referenced to the recipients' residential addresses were utilized. Clinical data was obtained from the recipients' online medical record, via an electronic system designed by Cerner Corporation that integrates electronic medical records into one system (CERNER), so no recipient contact or follow-up was required. The primary outcome variable was BMI change during the first year post-transplant, and exposures were quantity and ratio to total food sources of fast food restaurants, convenience stores, and grocery stores in one, two, and three mile buffer zones around recipient residential addresses.

Approval to conduct the study was sought from the Institutional Review Boards at The University of Tennessee Health Science Center, which is combined with the IRB from MUTI, and The University of Memphis. Since there was no recipient contact, interaction, or follow up, and therefore recipients would not be exposed to any substantial risks, approval as exempt status was granted by both IRBs.

Subjects

The study sample was MUTI renal transplant recipients who received their renal graft between January 1, 2004 and July 31, 2010 and who did not die or lose their graft during the first year post-transplant. Sixty-two percent of

recipients were African American, and 38% were female. There were also 5 Hispanics (1%), 7 Asians (1%), and 1 Native Hawaiian/Pacific Islander (0.2%) who were excluded from analyses due to small sample sizes.

Measures

Outcome variables. The primary outcome variable was change in BMI from baseline (time of transplant) to one year post-transplant, obtained from the recipients' online medical records at MUTI, via CERNER. Weight and height are routinely documented at all MUTI visits, including at admission for the transplant procedure, and at a one year post-transplant follow-up visit. BMI was calculated as: (weight in kg) / (height in m)^2.

Exposure variables. A total of 18 exposure variables were utilized. These include the quantity of fast food restaurants, convenience stores, and grocery stores within one, two, and three mile buffers of recipient households, as well as the ratio of each food source (fast food restaurants, convenience stories, or grocery stores) to total food sources (sum of the three sources) for each buffer zone.

Recipient race, gender, and age at transplant was also measured and examined in statistical models as potential confounders or moderators, as well as latitude and longitude coordinates of the recipient's residential address to account for clustering effects.

Procedures

Obtaining clinical data. A list of renal transplant recipients who received an organ between January 1, 2004 and July 31, 2010, along with race, gender,

date of transplant, age at time of transplant, residential address (house number, street, city, state, and zip code), height, weight at time of transplant, and weight 12 months post-transplant were obtained from CERNER.

Obtaining food availability data. Local business data containing fast food restaurants, conveniences stores, and grocery stores were obtained from a data provider (InfoGroup, Inc.; Papillion, NE). These data were obtained for the zip codes in which recipients reside. The data were in the form of names and addresses of all locations, allowing for precise mapping.

Mapping. Residential addresses were geocoded and spatially mapped on a coordinate plane using GIS. Each individual food source was then geocoded and spatially mapped over the residential addresses using the same coordinate plane. This resulted in a map of all residential addresses and food sources as they are located in relation to each other. GIS was then used to calculate the quantity of each food source in a one, two, and three mile buffer of each recipient's residential address using the point distance tool. See Figure 1 for further details.

Developing independent variables. After GIS was used to calculate the number of each food source (fast food restaurants, convenience stores, and grocery stores) in each buffer zone (one, two, and three mile radii), a ratio of each food source to total food sources was calculated. For example, if 12 of the 28 total food sources in a one mile radius of recipient 42's residence are fast food restaurants, then the total for fast food in this buffer zone would be 12, and the fast food ratio in this buffer zone would be 43%. This process was repeated for

each food source in each buffer zone for each recipient, resulting in 18 independent variables.

Developing dependent variables. After height, weight at time of transplant, and weight 12 months post-transplant were obtained from recipient charts, these values were used to calculate BMI change during the first year post-transplant for each recipient, which was the dependent variable for analysis.

Data Analysis

Descriptive analyses of data were conducted, measuring frequencies, means, standard deviations, and ranges of variables. Since all independent variables were extremely negatively skewed, they were recoded from continuous to categorical variables, where zero represented no food source (fast food restaurants, convenience stores, or grocery stores, depending on the analysis) being present within the buffer zone, a one represented the bottom tertile (all values greater than zero but below the 33rd percentile of the number of food sources within the buffer zone), two represented the middle tertile (all values greater than or equal to the 33rd percentile but below the 67th percentile), and three represented the highest tertile (all values above the 67th percentile). For each independent variable, three dummy-coded vectors were created, comparing the bottom, middle, and highest tertiles to zero food sources within the buffer zone.

Age, race, and gender were examined as potential moderators by evaluating their interactions with food availability on BMI change. No significant

interactions were observed, thus moderator terms were not included in outcome analyses.

Ordinary least squares multiple linear regression analyses were conducted to assess the effect of food availability to BMI change. Prior to running the models, normality of each variable was confirmed by visual inspection of histograms. In each of 18 models, BMI change from baseline to one year post-transplant was regressed on the food availability variable, adjusting for age, gender, race, and latitudinal and longitudinal coordinates of the recipient's residence. Multicollinarity was assessed by examining the variance inflation factors of all variables in each model.

Results

Descriptive Statistics

Sample. This sample (n = 484) was 38% female (n = 184) and 64% black (n = 310), with an average age of 48.6 (SD = 12.2 years). Baseline BMI averaged 28.2 (SD = 5.2), while 12 month post-transplant BMI averaged 29.4 (SD = 5.7), and mean BMI change was 1.2 BMI units (SD = 3.4). A paired t-test showed a significant average increase in BMI from baseline to 12 month follow-up ($p < .001$). Mean body weight, in pounds, was 185.4 and 193.4 at baseline and follow-up, respectively. This increase was statistically significant ($p < .001$). See Table 1 for more details.

The vast majority of recipients lived in the Memphis, TN metropolitan area and surrounding areas, with 38 total living in Arkansas, 82 in Mississippi, and 349 in Tennessee. However, this study also included recipients that lived in Alabama

(*n* = 2), Arizona (*n* = 1), California (*n* = 1), Colorado (*n* =1), Florida (*n* = 2), Georgia (*n* = 2), Illinois (*n* = 1), Missouri (*n* = 1), North Carolina (*n* = 1), Texas (*n* = 2), and Washington (*n* = 1). National state-wise and tri-state area zip code-wise population distributions can be viewed in Figures 2 and 3.

Food sources. In general, the distribution of quantity and ratio of food sources was as expected, with amounts increasing as the size of the buffer increased. However, many of these variables had a large amount of zeros (indicating lack of food sources within the buffer), especially the variables within a one mile buffer of recipient residences. Considering the small number of recipients living in rural areas, this was not unexpected.

Fast food restaurants. The mean number of fast food restaurants within one, two, and three mile buffers of recipient residences was 3.0 (*SD* = 4.5), 10.9 (*SD* = 11.1), and 20.9 (*SD* = 19.4), respectively. Fast food ratio within a one mile buffer around recipient residences averaged 26.6%, while ratios within two and three mile buffer both averaged 29.7%. See Table 2 for further details.

Convenience stores. The mean number of convenience stores within a one, two, and three mile buffer of recipient residences averaged 3.7 (*SD* = 5.0), 14.4 (*SD* = 15.6), and 29.7 (*SD* = 30.8), respectively. Convenience store ratio within a one mile buffer of recipient residences averaged 28.3%, whereas ratio within a two mile buffer averaged 35.9%, and ratio within a three mile buffer averaged 39.9%. See Table 2 for further details.

Grocery stores. The mean number of grocery stores within a one, two, and three mile buffer of recipient residences averaged 1.2 (*SD* = 2.3), 5.4 (*SD* =

7.2), and 10.7 (*SD* = 13.2), respectively. Ratio of grocery stores to total food sources averaged 8.1% within a one mile buffer around recipient residences, 16% within a two mile buffer, and 18.7% within a three mile buffer. See Table 2 for further details.

Regression Analysis

Fast food restaurants. BMI change was not associated with the number of fast food restaurants within a one mile buffer of recipient residences, with *p* values ranging from .535 to .770 for dummy-coded variables representing comparisons between recipients in the bottom, middle, and top tertiles to recipients who had zero fast food restaurants in a one mile buffer around their residences. Similar non-significant results were obtained for analyses using a two mile buffer zone (*p* values ranging from .345 to .711) and a three mile buffer zone (*p* values ranging from .339 to .859). See Table 3 for more details.

Likewise, BMI change was not associated with the ratio of fast food restaurants to total number of food sources within a one mile buffer of recipient residences, with *p* values ranging from .490 to .971, nor was it associated with fast food ratios within a two mile buffer zone of recipient residences, with *p* values ranging from .084 to .974. However, BMI change was positively associated with the ratio of fast food to total food sources within a three mile buffer of recipient residences. Examining the unstandardized betas for this significant association indicated that an increase of 0.134 units in the ratio of fast food to total food sources within three miles of the residence was associated with a one unit BMI change from baseline to 12 months post-transplant for recipients

in the bottom tertile of the ratio compared to those with zero fast food restaurants (b = 0.134, p = .024). Significant relationships were not observed for either the middle or highest tertiles of the ratio within the three mile buffer zone, nor for any of the comparisons in the one and two mile buffer zones. See Table 3 for more details.

Convenience stores. BMI change was not associated with the number of convenience stores within a one mile buffer of recipient residences, with p values ranging from .282 to .441 for dummy-coded variables representing comparisons between recipients in the bottom, middle, and top tertiles to recipients who had zero fast food restaurants in a one mile buffer around their residences. Similar non-significant results were found in a two mile buffer around recipient residences, with p values ranging from .206 to .785, and in a three mile buffer, with p values ranging from .435 to .676. See Table 4 for more details.

In addition to not being associated with convenience store quantity, BMI change was also not associated convenience store ratios within a one mile buffer of recipient residences, with p values ranging from .061 to .956, nor was it associated with fast food ratios within a two mile buffer zone of recipient residences, with p values ranging from .096 to .562, or within a three mile buffer zone, with p values ranging from .166 to .634. See Table 4 for more details.

Grocery stores. BMI change was not associated with the number of grocery stores within a one mile buffer of recipient residences, with p values ranging from .200 to .569 for dummy-coded variables representing comparisons between recipients in the bottom, middle, and top tertiles to recipients who had

zero fast food restaurants in a one mile buffer around their residences. The same non-significant results were also found in a two mile buffer around recipient residences, with *p* values ranging from .188 to .959, and in a three mile buffer, with *p* values ranging from .327 to .917. See Table 5 for more details.

Correspondingly, BMI change was also not associated grocery store ratios within a one mile buffer of recipient residences, with *p* values ranging from .242 to .537, grocery store ratios within a two mile buffer zone of recipient residences, with *p* values ranging from .289 to .859, or grocery store ratios within a three mile buffer zone, with *p* values ranging from .253 to .876. See Table 5 for more details.

Discussion

The present study largely failed to confirm, in a sample of renal transplant recipients, findings from the general population that the availability of food sources influences weight gain. Neither the number of fast food restaurants, convenience stores, and grocery stores, nor the ratio of each of these three food sources to the total number of food sources, with one, two, and three mile buffer zones around the recipients' residences, were generally associated with the amount of weight gain that occurred during the first year post-transplant. The one exception was a statistically significant result for the ratio of fast food restaurants to total food sources, indicating that having a greater ratio within a three mile buffer of one's residence was positively associated with BMI change.

Access to fast food restaurants has been shown to affect weight gain in several ways, including its influence on diet, as well as body weight and obesity

rates. Timperio et al. (2008) found that children in Australia were less likely to get the recommended fruit and vegetable intake based on the amount of and distance to fast food restaurants. In an examination of quantity and ratio of fast food restaurants and how these factors influence the BMIs of over 700,000 adults, an association was found between county-wide high quantities and ratios of fast food restaurants and higher individual BMIs (Mehta & Chang, 2008). Using data from the Stanford Heart Disease Prevention Program, it was determined that a 28% increase in obese women (*n* = 3145) and a 24% increase in obese men (*n* = 2625) could be attributed the large increases found in the number and density of fast food restaurants during a 9- year time period (Wang et al., 2008). Although the current study did find one significant relationship between fast food ratio within a three mile buffer and BMI change, the vast majority of comparisons were non-significant. The inconsistency of these results with previous studies may be due to our smaller sample size and sampling region. Previous studies all had several thousand subjects from nation-wide samples, whereas the current study had just less than 500, with the vast majority in the greater Memphis area.

Convenience store access also has been found in several previous studies to be associated with weight and weight gain. Pearce et al. (2008) found that those in neighborhoods with the highest levels of convenience stores had 25% lower odds of consuming the recommended daily amount of vegetables. Using a nationally representative sample of youths, it was found that, in urban areas, higher neighborhood density of convenience stores was directly

associated with obesity and BMI (Gibson, 2011). Convenience store access and obesity rate were found to be related in a study in Utah, especially in lower-income neighborhoods (Zick et al., 2009). Unlike the current study, these studies found significant effects between convenience store access and obesity. Small sample size is more than likely again to blame in this case, as well as sampling area. Although one study's sample consisted of only people from Utah, its sample still consisted of over 1000 subjects, allowing for better evaluation of potential effects.

The same inconsistencies were found between the current study and others examining the effect of grocery store access to obesity. A study of the eating habits of 1721 English children found that a higher density of super markets in the area was associated with increased vegetable intake (Skidmore et al., 2010). According to a review of 54 studies, neighborhoods with more access to grocery stores had lower levels of obesity (Larson et al., 2009). Again, it is likely that the lack of significant effects found in the current study was due to small sample size and the fact that the population consisted mostly of individuals who lived in and around Memphis, TN. It also is possible that, unlike the general population of adults, weight gain in renal transplant patients may not be largely affected by environmental factors such as food availability. Other research indicates that behavioral factors such as dietary and physical activity changes after transplantation (Macdonald et al., 2009; Moreau et al., 2006; Patel, 1998) are associated with weight gain. Further work is needed to explore the relative

contributions of genetic, behavioral, and environmental determinants of weight gain in this population.

There were several notable limitations in regards to this study. First and foremost was the relatively small sample size. Sample size in this study was limited by necessity to the number of transplant recipients at Methodist University Transplant Institute who meet eligibility criteria, which was 484. Studies in the general population that have reported statistically significant effects of food availability on weight-related variables typically have used larger sample sizes than that which was available to us in the current study, ranging from 826 (Casey et al., 2008) to more than 700,000 (Mehta & Chang, 2008) with several studies using sample sizes in the range of 2,000 to 4,000 (Bodor, Rice, Farley, Swalm, & Rose, 2010; Inagami et al., 2009). In the current study, it was expected that sample size needs would be lower than in general population studies, because weight gain is much higher among renal transplant recipients than in the general population. Indeed, weight change in this study averaged 8.02 pounds over one year, which is more than twice as high as weight change over one year in the general population of U.S. adults. A study using data from the National Health and Nutrition Examination Survey (NHANES) concluded that for the general population of U.S. adults, a major weight gain over 10 years would be 15.5 kg and 13.6 kg (34.2 and 30.0 pounds) for males and females, respectively (Kuczmarski, 1992). This averages to 3.21 pounds per year, which is less weight gain than our population experienced, showing our population gained more than the national average of “major” weight gain for one year, but we were generally

unable to detect significant effects. Nevertheless, the study makes a useful contribution in being the first study to examine these associations in a transplant population and providing data that can be used to help estimate effects to power larger studies in the future.

Another limitation involves the food source data. Ideally, food source data would be matched temporally. For example, if in the zip code 38114 there were recipients who were transplanted in 2005, 2008, and 2010, then fast food restaurant, convenience store, and grocery store data would be purchased for all three years for that zip code, with analyses done by compiling recipients and food source data into temporal groups. Unfortunately, limited funds for this study did not allow for this approach. As a compromise, food source data was purchased for all zip codes for the year 2007 only, which was the mid-point in the range of transplant dates (2004-2010). Although substantial changes in the numbers of food sources are unlikely within this relatively short time period, the lack of exact temporal matching of transplant follow-up period to the food source capture period is likely to add error to the analyses.

Another potential limitation of this study involves the quality of the data used. The clinical data (recipient residential address, weights, height, etc.) was obtained through a data analyst at MUTI, and any missing data were filled in by accessing the recipients' online medical records via CERNER. Although data were obtained from the same source, there were two people, the MUTI data analyst and the author, obtaining the data, which leaves room for inconsistencies. Also, data was collected for clinical purposes rather than

research, and is entered into CERNER by numerous medical staff members, which leaves room for data entry errors.

One of the eligibility criteria of this study was that recipients must have survived and maintained graft function the first year post-transplant. This was a necessity in order to determine BMI change, but it could have skewed the results. For example, as mentioned above, post-transplant obesity increases the likelihood of post-transplant mortality and graft loss. Therefore, those that gained a tremendous amount of weight could have been excluded due to death or graft loss. In addition, those who lost a large amount of weight could have also been excluded for the same reasons.

An advantage of this study is the large number of African American transplant recipients who were included, which is representative of the recipient population served by MUTI. A disadvantage of analyzing recipients from a single transplant center, however, is that the results may not be generalizable to other renal transplant recipients.

All things considered, this study and its results are important to the transplant community, because they provide a novel insight into the extensive issue of post-transplant obesity and its potential causes and contributing factors. Efforts should be made to conduct future research with more appropriate sample sizes and greater funding, as well as conducting a national study, evaluating racial differences, and evaluating weight change over a longer period of time to adequately assess effects and interactions and explore these associations more thoroughly.

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Appendix A

Table 1.

Sample Characteristics.

	% (*n*)
Total	100 (484)
Gender	
Female	38 (184)
Male	62 (300)
Race	
Black	64 (310)
White	36 (174)
	Mean (*SD*)
Age	48.6 (12.2)
BMI	
Baseline	28.2 (5.2)
1 year	29.4 (9.7)
Change	1.2 (3.4)
Weight (lbs)	
Baseline	185.4 (40.1)
1 year	193.4 (42.8)
Change	8.02 (22.4)

Table 2.

Number and Ratio of Food Sources within Buffer Zones.*

	Mean	*SD*	Range
Fast Food Restaurants			
1 mile buffer	3.0	4.5	26.0
2 mile buffer	10.9	11.1	45.0
3 mile buffer	20.9	19.5	71.0
1 mile ratio*	26.6	34.1	100.0
2 mile ratio*	29.7	25.8	100.0
3 mile ratio*	29.7	21.6	100.0
Convenience Stores			
1 mile buffer	3.7	5.0	24.0
2 mile buffer	14.4	15.6	61.0
3 mile buffer	29.7	30.9	103.0
1 mile ratio*	28.3	32.6	100.0
2 mile ratio*	35.9	28.1	100.0
3 mile ratio*	39.9	25.1	100.0
Grocery Stores			
1 mile buffer	1.2	2.3	14.0
2 mile buffer	5.4	7.2	35.0
3 mile buffer	10.7	13.2	58.0
1 mile ratio*	8.1	14.4	100.0
2 mile ratio*	16.0	19.7	100.0
3 mile ratio*	18.7	19.6	100.0

*Ratios are calculated as the number of food sources (fast food restaurants, convenience stores, grocery stores) divided by the total number of food sources (fast food restaurants + convenience stores + grocery stores).

Table 3.

Multiple linear regression results regressing change in body mass index on the number of fast food restaurants and the ratio of fast food restaurants to the total food sources within one, two, and three mile buffers around recipient residences, adjusted for age, gender, race, and latitude and longitude coordinates of recipient residences.

	Standardized Beta	Unstandardized Beta	Standard Error	*95% CI*		*p*
One mile buffer						
Number of fast food restaurants						
Bottom tertile vs. zero	-0.159	-0.014	0.542	-1.224	0.905	0.770
Middle tertile vs. zero	0.118	0.015	0.394	-0.656	0.892	0.765
Top tertile vs. zero	0.272	0.030	0.439	-0.590	1.135	0.535
Fast food restaurant ratio						
Bottom tertile vs. zero	0.053	0.006	0.461	-0.853	0.959	0.908
Middle tertile vs. zero	0.301	0.034	0.437	-0.556	1.159	0.490
Top tertile vs. zero	-0.016	-0.002	0.440	-0.880	0.848	0.971
Two mile buffer						
Number of fast food restaurants						
Bottom tertile vs. zero	0.167	0.020	0.449	-0.716	1.049	0.711
Middle tertile vs. zero	0.396	0.051	0.437	-0.463	1.256	0.365
Top tertile vs. zero	0.425	0.053	0.449	-0.458	1.308	0.345
Fast food restaurant ratio						
Bottom tertile vs. zero	0.256	0.032	0.456	-0.640	1.152	0.575
Middle tertile vs. zero	0.769	0.097	0.445	-0.105	1.643	0.084
Top tertile vs. zero	0.014	0.002	0.434	-0.838	0.867	0.974
Three mile buffer						
Number of fast food restaurants						
Bottom tertile vs. zero	0.219	0.027	0.457	-0.679	1.117	0.632
Middle tertile vs. zero	0.431	0.056	0.450	-0.454	1.316	0.339
Top tertile vs. zero	0.082	0.010	0.461	-0.824	0.988	0.859
Fast food restaurant ratio						
Bottom tertile vs. zero	1.062	0.134	0.470	0.138	1.986	**0.024**
Middle tertile vs. zero	0.062	0.008	0.451	-0.824	0.948	0.890
Top tertile vs. zero	-0.145	-0.018	0.443	-1.016	0.725	0.743

Bold indicates a statistically significant ($p < .05$) finding.

Table 4.

Multiple linear regression results regressing change in body mass index on the number of convenience stores and the ratio of convenience stores to the total food sources within one, two, and three mile buffers around recipient residences, adjusted for age, gender, race, and latitude and longitude coordinates of recipient residences.

	Standardized Beta	Unstandardized Beta	Standard Error	*95% CI*		*p*
One mile buffer						
Number of convenience stores						
Bottom tertile vs. zero	0.347	0.037	0.451	-0.538	1.233	0.441
Middle tertile vs. zero	0.372	0.040	0.467	-0.546	1.290	0.426
Top tertile vs. zero	0.480	0.054	0.445	-0.395	1.354	0.282
Convenience store ratio						
Bottom tertile vs. zero	0.835	0.090	0.445	-0.040	1.709	0.061
Middle tertile vs. zero	0.340	0.037	0.448	-0.540	1.219	0.448
Top tertile vs. zero	-0.025	-0.003	0.457	-0.923	0.873	0.956
Two mile buffer						
Number of convenience store						
Bottom tertile vs. zero	0.119	0.015	0.438	-0.740	0.979	0.785
Middle tertile vs. zero	0.334	0.042	0.449	-0.549	1.216	0.458
Top tertile vs. zero	0.574	0.072	0.453	-0.316	1.464	0.206
Convenience store ratio						
Bottom tertile vs. zero	-0.254	-0.032	0.438	-1.116	0.607	0.562
Middle tertile vs. zero	0.743	0.093	0.445	-0.132	1.618	0.096
Top tertile vs. zero	0.533	0.066	0.441	-0.334	1.400	0.228
Three mile buffer						
Number of convenience store						
Bottom tertile vs. zero	-0.380	-0.049	0.486	-1.335	0.567	0.435
Middle tertile vs. zero	0.210	0.028	0.501	-0.774	1.194	0.676
Top tertile vs. zero	0.393	0.051	0.510	-0.609	1.394	0.441
Convenience store ratio						
Bottom tertile vs. zero	-0.675	-0.087	0.487	-1.631	0.282	0.166
Middle tertile vs. zero	0.233	0.031	0.489	-0.728	1.194	0.634
Top tertile vs. zero	0.431	0.056	0.498	-0.548	1.409	0.388

Table 5.

Multiple linear regression results regressing change in body mass index on the number of grocery stores and the ratio of grocery stores to the total food sources within one, two, and three mile buffers around recipient residences, adjusted for age, gender, race, and latitude and longitude coordinates of recipient residences.

	Standardized Beta	Unstandardized Beta	Standard Error	*95% CI*		*p*
One mile buffer						
Number of grocery stores						
Bottom tertile vs. zero	0.375	0.036	0.486	-0.579	1.329	0.440
Middle tertile vs. zero	0.323	0.026	0.567	-0.791	1.437	0.569
Top tertile vs. zero	0.569	0.062	0.444	-0.303	1.441	0.200
Grocery store ratio						
Bottom tertile vs. zero	0.300	0.029	0.485	-0.654	1.254	0.537
Middle tertile vs. zero	0.565	0.055	0.483	-0.384	1.514	0.242
Top tertile vs. zero	0.471	0.046	0.494	-0.500	1.442	0.341
Two mile buffer						
Number of grocery stores						
Bottom tertile vs. zero	0.087	0.011	0.431	-0.761	0.934	0.841
Middle tertile vs. zero	0.024	0.003	0.462	-0.884	0.931	0.959
Top tertile vs. zero	0.574	0.074	0.435	-0.282	1.429	0.188
Grocery store ratio						
Bottom tertile vs. zero	0.077	0.009	0.436	-0.779	0.934	0.859
Middle tertile vs. zero	0.438	0.058	0.412	-0.372	1.247	0.289
Top tertile vs. zero	0.091	0.010	0.485	-0.862	1.045	0.851
Three mile buffer						
Number of grocery stores						
Bottom tertile vs. zero	0.145	0.018	0.486	-0.811	1.100	0.766
Middle tertile vs. zero	-0.048	-0.007	0.468	-0.967	0.870	0.917
Top tertile vs. zero	0.484	0.063	0.494	-0.468	1.455	0.327
Grocery store ratio						
Bottom tertile vs. zero	-0.145	-0.019	0.482	-1.093	0.803	0.764
Middle tertile vs. zero	0.548	0.071	0.479	-0.393	1.489	0.253
Top tertile vs. zero	0.074	0.010	0.478	-0.865	1.014	0.876

Appendix B

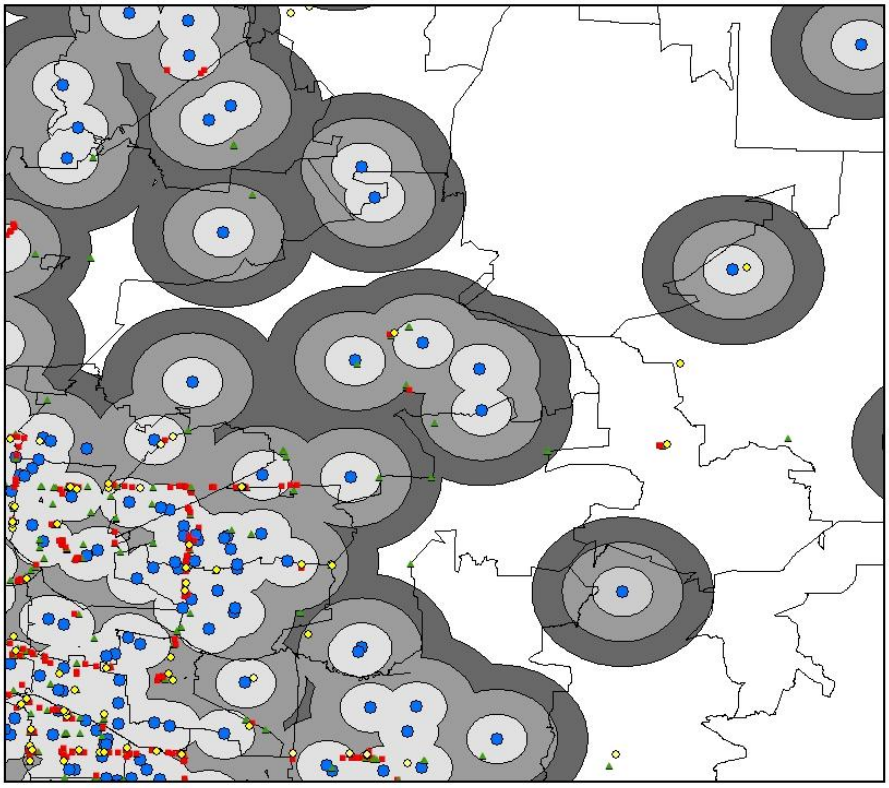

Figure 1. **Example of Food Source Distribution Around Residences***

*Data Obtained from U.S. Census, 2010 and Methodist University Transplant Institute

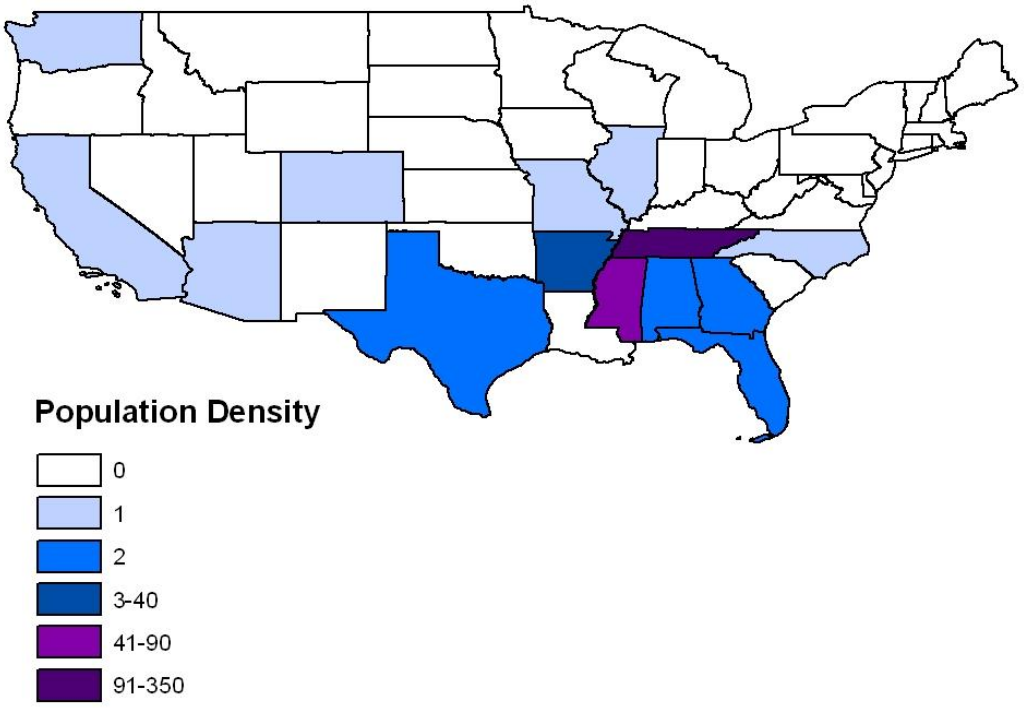

Figure 2. **Subject Population Distribution by State***

*Data Obtained from U.S. Census, 2010 and Methodist University Transplant Institute

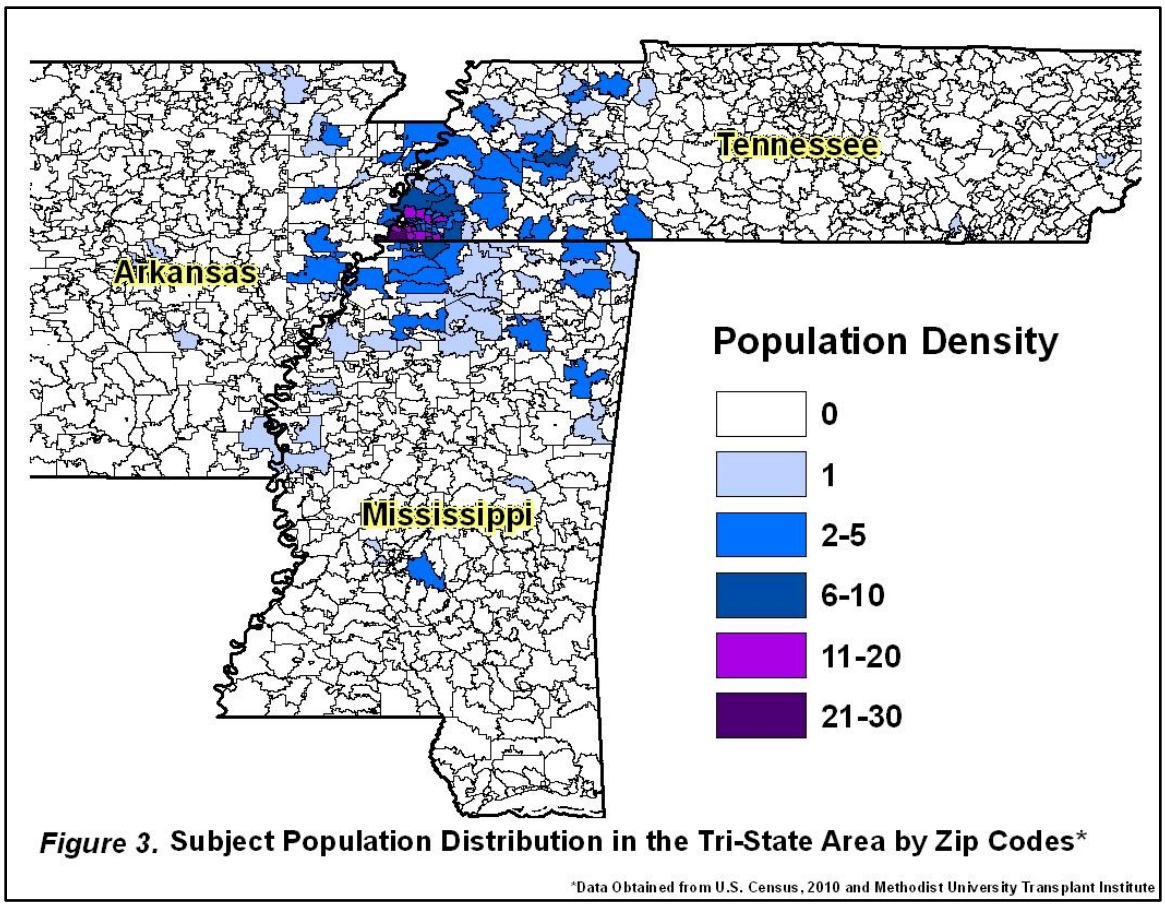

Figure 3. **Subject Population Distribution in the Tri-State Area by Zip Codes***

*Data Obtained from U.S. Census, 2010 and Methodist University Transplant Institute

Appendix C

THE UNIVERSITY OF MEMPHIS

Institutional Review Board

To:	Robin F. Bloodworth School of Public Health
From:	Chair or Designee, Institutional Review Board For the Protection of Human Subjects irb@memphis.edu
Subject:	Food Availability as a Determinant of Weight Gain Among Renal Transplant Recipients (100311-929)
Approval Date:	October 21, 2011

This is to notify you that the Institutional Review Board has designated the above referenced protocol as exempt from the full federal regulations. This project was reviewed in accordance with all applicable statuses and regulations as well as ethical principles.

When the project is finished or terminated, please submit a Human Subjects Research Completion Form (COMP) to the Board via e-mail at irbforms@memphis.edu. This form can be obtained on our website at http://www.memphis.edu/irb/forms.php.

Approval for this protocol does not expire. However, any change to the protocol must be reviewed and approved by the board prior to implementing the change.

Digitally signed by
Jacqueline Y. Reid
Date: 2011.10.21
11:51:57 -05'00'

Chair or Designee, Institutional Review Board
The University of Memphis

Cc: Dr. Kenneth D. Ward

Appendix D

THE UNIVERSITY OF TENNESSEE
Health Science Center

Institutional Review Board
910 Madison Avenue, Suite 600
Memphis, TN 38163
Tel: (901) 448-4824

August 24, 2011

Robin Frances Bloodworth
College of Nursing
Department of Nursing
507N 920 Madison Building

Re: 11-01454-XM
Study Title: Food Availability as a Determinant of Weight Gain in Renal Transplant Recipients

Dear Ms. Bloodworth,

The Administrative Section of the UTHSC Institutional Review Board (IRB) has received your written acceptance of and/or response dated August 22, 2011 to the provisos outlined in our correspondence of August 11, 2011 concerning the application for the above referenced project. The IRB determined that your application is eligible for **exempt** review 45CFR46.101(b)(4) in that it involves the study/project of existing data or other materials that are publicly available or the information will be recorded in a way that subjects cannot be individually identified. Informed consent is waived in accord with 45CFR46.116 (d). Your application has been determined to comply with proper consideration for the rights and welfare of human subjects and the regulatory requirements for the protection of human subjects. Therefore, this letter constitutes full approval of your application (version 1.1) for the above referenced study.

This study may not be initiated until you receive approval from the institution(s) where the research is being conducted.

In addition, the request for waiver of HIPAA authorization for the conduct of the study itself and to identify potential subjects is approved. The waiver applies to renal transplant patients at Methodist University Hospital from January 1, 2004 through July 31, 2011.

In the event that volunteers are to be recruited using solicitation materials, such as brochures, posters, web-based advertisements, etc., these materials must receive prior approval of the IRB.

Any alterations (**revisions**) in the protocol must be promptly submitted to and approved by the UTHSC Institutional Review Board prior to implementation of these revisions. You have individual responsibility for reporting to the Board in the event of unanticipated or serious adverse events and subject deaths.

Sincerely,

Signature applied by Donna L Stallings on 08/24/2011 12:26:25 PM CDT

Signature applied by Terrence F Ackerman on 08/24/2011 12:27:30 PM CDT

Donna Stallings, CIM
IRB Administrator

Terrence F. Ackerman, Ph.D.
Chairman

Appendix E

Sent From:	REXANN G PICKERING
Send To:	Robin Frances Bloodworth, REXANN G PICKERING, RN, PhD, Ann K Cashion
IRB Number:	11-01454-XM
IRB Expiration:	
Working Title:	Txp Food Availability
Title:	Food Availability as a Determinant of Weight Gain in Renal Transplant Recipients
Study Status:	Approved
Principal Investigator:	Robin Frances Bloodworth
Message Content:	This correspondence is to acknowledge that Methodist Le Bonheur Healthcare has reviewed the study, 11-01454, PI - Robin Bloodworth, as currently submitted through iMedRIS. MH-LB action – approved Sponsor – none *Dr. Cashion - please notify R. Pickering when this study is completed since the PI is a student.* Please submit your medical record requests to the HIM manager at the respective hospital from which records are needed. [record date range 01/01/2005 - 07/31/2011] data to be extracted: [Please be aware that Methodist Le Bonheur Healthcare may, at its expense and upon reasonable notice to you, audit or have audited your research records. Rexann G. Pickering, Ph.D., R.N., CIP, CIM Administrator, Human Protection Methodist Le Bonheur Healthcare