LONGITUDINAL ASSOCIATIONS BETWEEN THE RETAIL FOOD ENVIRONMENT, DIET QUALITY, AND CHRONIC DISEASE RISK AMONG BLACK AND WHITE YOUNG ADULTS IN THE UNITED STATES

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A dissertation submitted to the faculty of the University of North Carolina at Chapel Hill in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the Department of Nutrition of the Gillings School of Global Public Health.

Chapel Hill
2010

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ABSTRACT

Daisy Zamora: Longitudinal associations between the retail food environment, diet quality,
and chronic disease risk among Black and White young adults in the United States
(Under the direction of Barry M. Popkin, PhD)

The recognition of the public health and economic consequences of nutrition-related

chronic diseases has led to policies focused on improving the diets of the population

subgroups at highest risk: low-income people and African Americans. Sometimes, however,

the evidence base for these policies is weak. For example, in response to research suggesting

that low-income areas have less access to affordable and nutritious foods, policy

interventions have been proposed to address this access disparity; however, this association is

far from well established. Similarly, the Dietary Guidelines for Americans (DGA) are

widely promoted in public health campaigns, yet there is surprisingly little evidence that

following them is effective at reducing risk of chronic disease in the general population, in

part because most past studies have been cross-sectional, demographically homogeneous, or

conceptually flawed (e.g., potential for reverse causality). Our research fills important

substantive gaps in the understanding of these relationships using data from a geographically

diverse cohort of Black and White young adults (followed from 1985 to 2005) and analytic

methods that exploit the longitudinal structure of the data. First, we examined the

longitudinal association between neighborhood socio-demographics and the availability of

foods stores. We found that poor and high minority areas had higher availability of

supermarkets and grocery stores. This suggests that, contrary to common assumption, stores

where healthy foods can typically be purchased are available to the subgroups at highest risk. Second, we examined the prospective association between adherence to the 2005 DGA and risk of major weight gain, diabetes, and progression of other cardio-metabolic risk factors. Overall, we found little evidence that a higher diet quality led to better long-term health (except for blood pressure and HDL cholesterol outcomes). In Whites, higher diet quality was associated with less 20-year weight gain, but unrelated to diabetes incidence or insulin resistance. However, in Blacks, higher diet quality was associated with *greater 20-year weight gain*, and with slightly *higher incidence of diabetes* and increase in insulin resistance. Results of this research highlight the urgent need for effectiveness trials of the DGA.

To my family

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LIST OF ABBREVIATIONS

BMI   Body Mass Index. Calculated by weight (in kilograms) divided by height (in meters) squared (i.e., BMI = kg/m^2).

CVD   Cardiovascular diseases

DGA   Dietary Guidelines for Americans

DQI   Diet Quality Index

FE   Fixed Effects

HDL   High-density lipoprotein

GLS   Generalized Least Squares

HOMA-IR   Homeostasis model insulin resistance

kcal   Kilocalorie, equivalent to 1000 calories or about 4.184 kilojoules.

kg   Kilogram = 1,000 grams

km   Kilometer = 1,000 meters

SES   Socio-economic status

I. INTRODUCTION

A.   BACKGROUND

The burden of nutrition-related chronic diseases is rapidly increasing in the U.S. and throughout the world. Although cardiovascular diseases (CVD) are the leading causes of morbidity and mortality, obesity and diabetes have shown worrisome trends, not only because they already affect a large proportion of the population, but also because they have started to appear earlier in life (2003). According to the Surgeon General's "Call to Action" on obesity, health problems resulting from overweight and obesity could reverse many of the health gains achieved in the U.S. in recent decades (Jackson, et al., 2002). In addition, chronic diseases disproportionately affect people with low socio-economic status (SES) as well as African Americans (Harris, Gordon-Larsen, Chantala, & Udry, 2006; Krieger, 1993). The relation between SES and health is consistent across a variety of settings, yet it is complex and not fully understood. Because having a low SES has been linked to higher intake of refined carbohydrates, fast foods, soft drinks, etc., diet quality has been proposed as a potential mediator of this relationship (Deshmukh-Taskar, Nicklas, Yang, & Berenson, 2007; Glanz, Basil, Maibach, Goldberg, & Snyder, 1998; Krebs-Smith & Kantor, 2001; Popkin, Zizza, & Siega-Riz, 2003; Reicks, Randall, & Haynes, 1994; Turrell & Kavanagh, 2006).

Perhaps in part because early dietary interventions to improve health through promotion of DGA were largely unsuccessful, more attention is being given to the retail food

environment as a determinant of diet. Indeed, the spatial distribution of 'health-promoting' resources has been linked to racial and SES composition of neighborhoods, suggesting that minorities and people with low SES, who have a greater disease burden, may also have the worst access to healthy foods (Beaulac, Kristjansson, & Cummins, 2009). Since studies have found a correlation between what people eat and the foods available in their communities (Cheadle, et al., 1991; Latetia V. Moore, Diez Roux, Nettleton, & Jacobs, 2008; Morland, Wing, & Diez Roux, 2002), lack of access to healthy foods has been hypothesized to be a determinant of diet.

The growing recognition of the public health and economic consequences of nutrition-related chronic diseases has prompted actions by policymakers, the public health community, and other organizations. Many of these actions have focused on promoting adherence to the DGA among the populations at highest risk for chronic diseases: people with low SES and African Americans (Harris, et al., 2006; Krieger, 1993). Moreover, in an effort to provide people in low income areas with better food options, initiatives to encourage the development of grocery retail investments in low-income communities have been called for (Pothukuchi, 2005; Story, Kaphingst, Robinson-O'Brien, & Glanz, 2008).

The problem is that these initiatives are based on several assumptions that are not currently supported by research. For example, since the DGA are intended to "promote health and reduce the risk of chronic disease", it is often assumed that they can help prevent major weight gain or related chronic diseases. Yet there is little evidence that people who have followed diets congruent with the DGA actually gain less weight over a long period of time. The literature is mixed with regard to diabetes and CVD outcomes (Folsom, Parker, & Harnack, 2007; Fung, et al., 2008; Fung, et al., 2005; B. V. Howard, et al., 2006; Imamura,

Jacques, Herrington, Dallal, & Lichtenstein, 2009; McCullough, et al., 2000; McCullough, et al., 2002).

This study addresses several important gaps in our understanding of environmental determinants and health consequences of long-term adherence to the 2005 DGA. The chronic diseases considered in this research are those that are related to nutrition and present the greatest public health burden, either in terms of direct cost to society and government, or in terms of disability-adjusted life years. These include obesity, diabetes, and cardiovascular disease. Because our sample consists of young adults and thus CVD incidence is very low, we studied biomarkers of CVD risk as proxies of future disease incidence.

B. RESEARCH AIMS

Our specific aims were as follows:

1) To examine the longitudinal association between census block group poverty and race/ethnicity and neighborhood-level availability of supermarkets and grocery stores. Supermarkets are large stores (mostly chains) including places like Albertson's, Kroger, and Harris Teeter with any of the following Standard Industry Classification (SIC) codes: 54110100, 54110101, 54110102, 54110103, 54110104, and 54110105. Grocery stores, which are smaller than supermarkets but larger than convenience stores, have the following SIC codes: 54110000, 54119900, 54119903, 54119904, and 54119905. Food store availability was operationalized as the number of stores per 10,000 people living within 8 km (Euclidean distance) of participants' residence. Consistent with the current literature, we hypothesized that neighborhoods with high poverty and high ethnic minority composition would have lower availability of supermarkets but higher

availability of grocery stores. We estimated associations between neighborhood poverty

and minority composition on food store density using longitudinal, repeated measures

conditional regression linear models (i.e., fixed effects models).

2) To examine the associations between agreement with the 2005 DGA (assessed with the

2005 Diet Quality Index (DQI)) and subsequent risk of diabetes, major weight gain, and

progression of other cardio-metabolic risk factors. We hypothesized that higher DQI

scores would be predictive of lower disease risk, and that race would modify this

association. To avoid the issue of reverse causality (e.g., diabetes diagnosis affecting

diet, rather than diet predicting diabetes development), we only used diet data collected

before participants developed the outcome of interest. The general set up for our

analyses was for diet at an earlier time point to predict change in the outcome (e.g., diet

measured at year 0 predicted risk from year 0 to year 7, diet measured at year 7 predicted

risk from year 7 to year 20).

    a. We used survival analysis to examine the associations between DQI scores and

20-year risk of major weight gain (10 kg) from 1985 to 2005. We also examined

the longitudinal association between continuous weight change from baseline to

each subsequent examination (1985-2005) and DQI scores using generalized

estimating equations models.

    b. We used survival analysis to examine the associations between DQI scores and

20-year risk of diabetes (1985 to 2005).

    c. We examined the prospective association between DQI scores and 13-year change

in continuous high-density lipoprotein (HDL) cholesterol, plasma glucose,

homeostasis model insulin resistance (HOMA-IR), systolic and diastolic blood

pressure, waist circumference, and triglycerides using linear regression models (1992-2005).

Analyses were conducted using data from the Coronary Artery Risk Development in Young Adults (CARDIA) study, a 20-year prospective study of CVD risk factors in 5,115 young black and white men and women recruited from Birmingham, Alabama; Chicago, Illinois; Minneapolis, Minnesota; and Oakland, California. To obtain measures of the participant's environment, Geographic Information Systems software was used to link residential street addresses of participants for exam years 7 (1992), 10 (1995), and 15 (2000) to contemporaneous neighborhood information from federal and commercial databases.

II. LITERATURE REVIEW

A.   SCOPE OF THE PROBLEM

According to the Surgeon General's "Call to Action" on obesity, health problems resulting from overweight and obesity could reverse many of the health gains achieved in the U.S. in recent decades (Jackson, et al., 2002). Obesity is appearing earlier in life than in past generations (McTigue, Garrett, & Popkin, 2002) and the rapid increase in its prevalence (Flegal, Carroll, Kuczmarski, & Johnson, 1998; Flegal, Carroll, Ogden, & Johnson, 2002; Ogden, et al., 2006) is a significant public health concern not only because of the morbidity it causes, but also because it is a robust predictor of diabetes and CVD later in life (WHO, 2003). Here are some of the reasons why high rates of weight gain among young adults need urgent attention:

- Many of the factors that lead to obesity (e.g., sedentary lifestyle and poor nutrition) can also lead to chronic disease (WHO, 2003). In other words, some of the increased risk associated with obesity is not due to excess fat per se, but to a shared etiology. Hence, because chronic diseases take a long time to develop whereas obesity does not, obesity is an early indicator of unhealthy lifestyles in a population.

- Obesity leads to adverse health outcomes. For example, excess abdominal adipose tissue and excess triglyceride content in skeletal muscle, liver, and heart tissues are directly associated with hepatic and skeletal muscle insulin resistance, impaired

ventricular function, and increased coronary heart disease (Alpert, Flinn, & Flinn, 2001; Krssak, et al., 1999; Pouliot, et al., 1994). Excess adiposity also triggers an inflammatory response that interferes with cellular signaling affecting glucose tolerance and serum lipid levels and, at least in animals, leads to a decrease of cell-mediated immunity and decreased resistance to infections (Stallone, 1994). Moreover, progression of coronary artery calcification (early marker of atherosclerosis) is evident in obese people, even those that do not have any other risk factors for CVD (Cassidy, et al., 2005). Finally, the manifestations of chronic diseases such as diabetes mellitus and CVD tend to worsen as the degree of obesity increases (Allison, Fontaine, Manson, Stevens, & VanItallie, 1999; Pi-Sunyer, 1993; Resnick, Valsania, Halter, & Lin, 2000).

- Obesity is a socioeconomic problem that disproportionately affects economically disadvantaged groups (Harris, et al., 2006; Krieger, 1993). Of particular concern is that in the U.S. racial minorities are more likely to have a low socio-economic status (Harris, et al., 2006; Krieger, 1993), and minorities are expected to comprise an increasingly larger proportion of the U.S. population in coming years (U.S. Census). One of the goals of Healthy People 2010, the nation's prevention agenda, is to eliminate health disparities including differences that occur by race, education, income, and geographic location. Inequalities in health outcomes are not new (Adler, et al., 1994; Feinstein, 1993; Sundquist & Johansson, 1998); however, there is some evidence the uneven distribution of obesity and related co-morbidities among minorities and low-income groups is linked to the higher cost of healthy foods and to

characteristics of the built environment in disadvantaged areas (Drewnowski &

Darmon, 2005).

There are many factors contributing to the rising rate of nutrition-related chronic

disease in the U.S. and around the world. Unfortunately, our understanding of these factors

and their interactions is minimal. At the most basic level, weight gain is due to an energy

imbalance: less energy is expended than is consumed. The reasons that lead to this imbalance

can be broadly classified as genetic, environmental (i.e., non-genetic), or an interaction of

both. Studies of the genetic causes of obesity using twin pairs have demonstrated that a large

portion of inter-individual variation in weight can be explained by heredity (Pietilainen, et

al., 1999; Stunkard, Foch, & Hrubec, 1986). However, even though genes increase

susceptibility for obesity, research to date has not identified a set of genes responsible for

obesity in the majority of the population (Bouchard, 1995a, 1995b). Non-genetic

determinants of body weight include lifestyle behaviors that have a direct impact on energy

balance (i.e., diet, physical activity), as well as factors that influence such behaviors (e.g.,

age, transportation, cultural norms, education, job-related activity, stress, income, food prices

and marketing, and the contextual effects of the built environment). Although our

understanding of how all these factors interact to influence body weight is incomplete, it is

clear that dietary patterns have drastically changed in the last few decades (Popkin, 2006)

Rationale and conceptual framework

Little progress has been made in halting the obesity epidemic even after decades of

trying to understand (and modify) the causes of obesity at the individual level. Behavioral

approaches addressing chronic disease risk factors have not succeeded in producing

sustainable dietary and lifestyle changes (Carleton, Lasater, Assaf, Feldman, & McKinlay,

1995; Farquhar, et al., 1990; Hyman, Pavlik, Taylor, Goodrick, & Moye, 2007; Luepker, et al., 1996). Weight-loss interventions that have focused on changing individual behavior are usually effective for the first year, yet participants gain back their baseline weight shortly after (J. O. Hill, Thompson, & Wyatt, 2005). As a result, more and more studies are now focusing on the role of the community and residential environment in the development of obesity (Papas, et al., 2007).

The basic premise for studying the residential environment as it relates to obesity and related chronic diseases is the idea that the physical characteristics of an area can influence the lifestyles of people who live there (James O. Hill & Peters, 1998; James O. Hill, Wyatt, Reed, & Peters, 2003). Because spatial distribution of 'health-promoting' resources is linked to racial and SES composition, it is hypothesized that people living in disadvantaged areas are less able to engage in physical activity at recreational facilities and to buy healthy food at their local store (Booth, Pinkston, & Poston, 2005; S. Cummins & Macintyre, 2006). One example of how the environment may play a role is that food stores that offer a good selection of healthy foods tend to be less available in economically disadvantaged and predominantly black areas (Glanz 1998, Drewnoski and Specter 2004, Baker 2006, Kaufman 1997). This is known as "deprivation amplification", a situation where poorer and minority neighborhoods have fewer health-promoting resources compared to their wealthier and white counterparts (Macintyre, 2007).

In this conceptual framework, environmental constraints (e.g., lack of sidewalks or places to buy fresh produce) in low SES neighborhoods may negatively affect the health of those who live there by limiting their ability to adopt healthy behaviors (S. Cummins & Macintyre, 2006). Hence, if the environmental constrains were removed, people in

disadvantaged areas would lead healthier lifestyles. For example, if one cause of the

disproportionate obesity rates in low-income groups is that their neighborhoods lack food

stores where nutritious, affordable foods can be purchased, then addition of such resources

should help ameliorate the problem.

Figure 1. Time-varying relationships under study, numbers correspond to aims
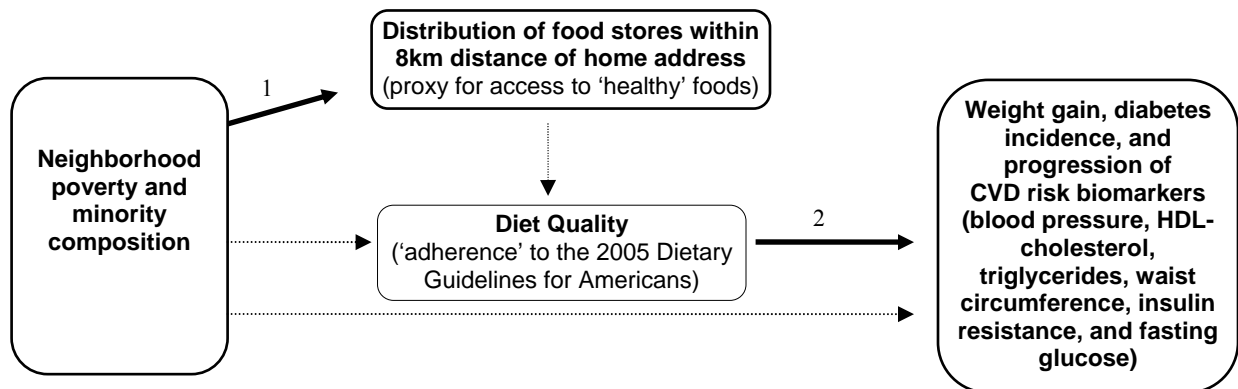
B.   DIET

Dietary determinants and trends

It is estimated that the average number of calories consumed in a day by U.S. adults

has increased from 1,969 calories in 1978 to 2,200 calories in 1990 as a result of increased

consumption of energy-dense, nutrient-poor foods and snacks (French, Story, & Jeffrey,

2001; Haines, Siega-Riz, & Popkin, 1999; Nielsen, Siega-Riz, & Popkin, 2002a). In

addition, one of the many lifestyle changes taking place in the last few decades is a shift from

eating at home to eating at restaurants and fast food places (Nielsen, Siega-Riz, & Popkin,

2002). This is problematic because as food portions have increased since the 1970's, so has

the caloric content of meals served at restaurants and fast food outlets (Briefel & Johnson, 2004). Data from the Healthy Eating Index (HEI) over time show that Americans are reducing the proportion of total fat and saturated fat in their diets and eating a wider variety of foods (Guo, Warden, Paeratakul, & Bray, 2004), yet most people still don't meet the recommended dietary intakes (Guenther, Dodd, Reedy, & Krebs-Smith, 2006; Kant, Schatzkin, Block, Ziegler, & Nestle, 1991; Li, et al., 2000).

Non-physiologic factors that influence dietary behavior include: social norms, education, health or weight concerns, marketing and advertising, convenience, taste preferences, purchasing power (family economics and food costs), and food availability (physical environment, e.g., distribution of food stores, climate, plant cultivation, rural/urban distribution patterns, transportation) (Glanz, et al., 1998; Neumark-Sztainer, Hannan, Story, Croll, & Perry, 2003; Papas, et al., 2007; Sallis & Glanz, 2006).

Healthy foods are more expensive

Several studies indicate that foods usually recommended for a healthy diet (fruits, vegetables, whole grains, low-fat dairy) are more expensive than less healthful, energy-dense refined foods (Drewnowski, 2004; Jetter & Cassady, 2006; Kaufman, Cooper, & McGee, 1997; Macdonald & Nelson, 1991). High-energy-density foods such as fats, oils, sugar, and refined grains provide energy at the lowest cost, whereas low-energy-density foods such as vegetables, fruits, seafood, and dairy products provide energy at the highest cost (Drewnowski & Darmon, 2005). Intake of energy-dense, nutrient-poor foods results in an increased risk of overeating (Drewnowski, 2004). Moreover, local food prices may potentially affect dietary intake. Sturm et al. (Sturm & Datar, 2005) conducted a study of local (metropolitan area) food prices and BMI in a nationally representative sample of

elementary-school children. They found that one-year BMI gains were positively associated with higher fruit and vegetable prices and inversely associated with meat prices.

Low-income families are more likely to consider the high price of healthy foods a barrier to healthy eating (Glanz, et al., 1998), (Drewnowski & Specter, 2004). In a study by Carlson et al., participants who paid less for their food consumed diets higher in calories and lower in nutrients, as compared to those who spent more money on food (Carlson, Andrews, & Bickel, 1999). These differences in the cost of food have a direct impact on food purchasing decisions and in turn on dietary practices (Drewnowski & Specter, 2004; Glanz & Yaroch, 2004). Thus, the high prices associated with better diet quality may be prohibitive for low-income groups and thus an important factor in the disproportionately higher rates of obesity among the poor (Drewnowski & Specter, 2004).

Researching diet patterns and health

Within the past 30 years, attention has been focused increasingly on the relationship of diet and nutrition-related chronic diseases (Baxter, et al., 2006; Ledikwe, et al., 2006; Toft, et al., 2006). When the chronic disorders of glucose intolerance, insulin resistance, hyperlipidemia, and hypertension are linked together, they are known as the metabolic syndrome (NIH, 2001). The prevalence of the metabolic syndrome is rapidly increasing in relation to obesity, and it is an important predictor of diabetes and cardiovascular disease (Carpentier, Portois, & Malaisse, 2006). A recent review by Baxter et al. (Baxter, Coyne, & McClintock, 2006) found that high intake of fruits, vegetables, whole cereals, and low-fat dairy have been associated with decreased risk of developing the metabolic syndrome, while increased consumption of meat and processed cereals are associated with increased risk. Moreover, fried foods were noticeably absent from any dietary pattern associated with

decreased prevalence of metabolic syndrome. The authors concluded that no individual dietary component could be considered wholly responsible for the association of diet with the metabolic syndrome, but rather it is the overall quality of the diet that appears to offer protection against lifestyle diseases.

There are important gaps in our understanding of the association between dietary patterns and obesity-related outcomes (Togo, Osler, Sorensen, & Heitmann, 2001). Although there is a strong consensus that a poor diet (e.g., high intake of fast-food, caloric sweeteners, salty snacks) leads to weight gain, studies on free-living individuals are often inconclusive (Kant, 1996). The imprecision and variability in dietary assessment methods along with the tendency of overweight individuals to underreport food intake (Johansson, Wikman, Ahren, Hallmans, & Johansson, 2001) may partially account for discrepancies across study results (Macdiarmid & Blundell, 1997). Inconsistent findings may also be due in part to problems of collinearity among nutrients inherent in traditional single-nutrient approaches, or to an inability to detect small effects from single nutrients (Newby, et al., 2003).

Whole diet vs. single diet components

Dietary exposures used in obesity studies typically range from single nutrients to specific foods or food groups e.g., percent energy from fat, intake of fruits and vegetables, meats, dairy, fried foods). Often, these dietary components are studied in isolation; removed from the context of the overall diet. In developed countries, however, the relationships between diet and chronic diseases are seldom attributed to the lack or excess of a single dietary component. Since each meal comprises a mixture of several foods and nutrients, health outcomes are likely a result of overall dietary quality rather than a few food groups or nutrients. Hence, it is generally unsatisfactory to examine the relationship between a single

dietary factor and disease in isolation (Togo, Osler, Sorensen, et al., 2001). Additionally, by

focusing on the overall quality of diet, the potential effect of known and unknown

interactions within foods and thereby nutrients may be taken into account (Togo, Osler,

Sorensen, et al., 2001).

There are two fundamentally different approaches used for studying whole diets in

observational epidemiological studies: *a priori* methods and *a posteriori* methods. In

contrast to exploratory data analysis methods, an index of diet quality is based on *a priori*

standards of healthy eating from which scoring criteria are derived (Haines, et al., 1999; R. E.

Patterson, Haines, & Popkin, 1994; Popkin, Haines, & Siega-riz, 1999). Diet index methods

are confirmatory by nature as they rely on previous knowledge to select which variables to

include and what cut-points to use. A major reason for making an index, rather than studying

its component variables in isolation, is to study a combined effect of the variables (Togo,

Osler, Sorensen, et al., 2001).

The *a posteriori* methods typically rely on factor or cluster analyses to identify eating

patterns of specific populations but have several limitations (Togo, Osler, Sorensen, et al.,

2001). General limitations of exploratory factor analysis methods include the following: the

exact definition of a 'factor' and the individual 'factor score' may prove difficult in readily

understandable terms. The method involves a large amount of data driven and subjective

decision-making in the course of the analysis (variable scale, number of variables, number of

factors, rotation method, interpretation, criteria for interpretation etc.), which may contribute

to the inconsistency and considerably limit the ability to generalize the results. General

limitations of cluster analysis methods include the following: there is no gold standard for

determining the number of clusters; the dietary input needs to be considered carefully in

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terms of scaling, since the differences between high, moderate or low intake are central to the analysis; typical scenarios are 1) a few distinct clusters with very few people and one or more large left over clusters, or 2) more similarly sized clusters with a minimum of inter-cluster variation in diet intake.

Diet and obesity-related outcomes

While the body of evidence supporting the use of composite measures of diet is growing (Baxter, Coyne, & McClintock, 2006; Kant, 1996; Ledikwe, et al., 2006; Toft, Kristoffersen, Lau, Borch-Johnsen, & Jorgensen, 2006), relatively few studies on obesity and related chronic diseases have used methods that attempt to account for the full complexity of the diet. However, dietary patterns are not necessarily better predictors of disease, as the methods used to define patterns varies from study to study. Togo et al reviewed 30 observational studies relating food intake patterns to BMI and found no consistent patterns associated with increased BMI or obesity. They also reported that studies using diet index scores (e.g. Healthy Eating Index, Diversity Score) were more consistent in their findings- negative associations with obesity- than those using cluster or factor analysis (Togo, Osler, Sorensen, et al., 2001).

For the most part, the studies that have found inverse associations between adherence to the DGA and body weight or obesity have been short-term or cross-sectional (Berg, et al., 2008; S. K. Gao, et al., 2008; Guo, et al., 2004; Kant & Graubard, 2005; Togo, Osler, S›rensen, & Heitmann, 2001). Studies with follow-up longer than one year are summarized in **Table 1**. While two studies found that diets consistent with the DGA were associated with lower weight gain (Barbara V. Howard, et al., 2006; Paula A. Quatromoni, Pencina, Cobain, Jacques, & D/'Agostino, 2006), the rest produced inconsistent results where DGA-like

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dietary patterns were not clearly better at preventing weight gain compared to other patterns (Burke, Giangiulio, Gillam, Beilin, & Houghton, 2003; Newby, et al., 2003; P. A. Quatromoni, Copenhafer, D'Agostino, & Millen, 2002; Thomson, et al., 2005; Togo, Osler, Sorensen, & Heitmann, 2004). Three other longitudinal studies examined the association between changes in diet quality defined using factor analysis (Newby, Weismayer, Akesson, Tucker, & Wolk, 2006; M. B. Schulze, Fung, Manson, Willett, & Hu, 2006; Togo, Osler, Sorensen, & Heitmann, 2004). Two found significant associations between increasing factor scores for a 'healthy' or 'prudent' food pattern (which have some overlap with the DGA) and decreases in weight gain among women (Newby, et al., 2006; M. B. Schulze, et al., 2006). However, as Newby et al and Schulze et al suggested, the interpretation of factor scores is subjective and results are not easily translated into dietary advice.

It is unclear whether diet quality influences energy balance. A recent trial of diet and weight change suggested that people meeting the 2005 DGA recommendations have a lower energy density diet and thus may be more likely to lose weight (Ledikwe, et al., 2007). However, in a randomized, controlled dietary intervention by Thomson et al., a healthy diet (characterized by low intake of fat and high intake of fruits, vegetables, and fiber) without specific energy goals was studied. The intervention group increased intake of plant foods and decreased fat in their diets, but changes in weight, WHR, BMI, and body composition were not different over time or by study group assignment. The authors concluded that interventions that promote a plant-based diet without specific energy restriction do not appear to promote changes in body weight.

Definition of a healthy diet

The original Dietary Guidelines for Americans were issued in 1973 by the U.S. Department of Agriculture (USDA) as a response to national concern about the increase in incidence of obesity and chronic diseases. In particular, the report stressed the potential contribution of the high consumption of fat and sugar to the development of the leading causes of death. As nutrition knowledge has evolved over time, so have the dietary guidelines. Currently, the American Cancer Society, the American Dietetic Association, the American Academy of Pediatrics, the National Institutes of Health, and the American Heart Association recommend a healthy eating program based on the USDA's Food Guide Pyramid (Nutrition and Your Health, 2000). Contrary to popular belief, the relationship between diet and disease is far from well-established; there is no standard, widely-accepted definition of a healthy diet. Even though research linking specific aspects of diet to certain cancers (G. Block, Patterson, & Subar, 1992), diabetes (M. B. Schulze & Hu, 2002), and heart disease (Ignarro, Balestrieri, & Napoli, 2007) has led to changes in the dietary recommendations, there is limited evidence at the population level supporting their effectiveness (McCullough, Feskanich, Rimm, et al., 2000; McCullough, et al., 2002; McCullough, Feskanich, Stampfer, et al., 2000; McCullough & Willett, 2006; Osler, et al., 2002; M. B. Schulze & Hu, 2002).

On the contrary, there is evidence that the DGA may not provide the best dietary advice for the prevention of chronic disease (Chiuve & Willett, 2007). Alternate dietary patterns, in particular, Mediterranean-type diets, may provide stronger health effects compared to the DGA (Chrysohoou, Panagiotakos, Pitsavos, Das, & Stefanadis, 2004; de Lorgeril, et al., 1999; Hu, 2002; W. C. Willett, 2006). For example, the Lyon Diet Heart Study, a randomized, single-blind secondary prevention trial, examined whether a Mediterranean-type diet, compared with a prudent Western-type diet, may reduce recurrence

after a first myocardial infarction (de Lorgeril, et al., 1999). In that study, 423 patients with a history of myocardial infarction were randomized to a Cretan Mediterranean diet (high in beneficial fats and low in animal fat) versus a prudent Western diet. The latter group were advised to follow a prudent diet, but were given no specific dietary instructions; dietary questionnaires at the end of the study showed that these patients were consuming a diet equivalent to the National Cholesterol Education Program Step 1 diet, with a cholesterol intake below 300 mg daily and 20% of calories from fat. This study showed an impressive protective effect of adherence to the Mediterranean diet: death and myocardial infarction were reduced by 60% in 4 years, compared to the control group. In another study, high adherence to the Mediterranean diet was associated with lower likelihood of becoming obese among overweight subjects (Mendez, et al., 2006).

C.   UNDERSTANDING THE RETAIL FOOD ENVIRONMENT

Neighborhood environment and health

Compelling evidence suggests that the geographic distribution of nutrition-related health outcomes varies across levels of neighborhood deprivation. For example, low neighborhood SES has been linked with the development of obesity, metabolic syndrome, and CVD, independently of individual-level factors (Diez-Roux, et al., 2001; Diez-Roux, Link, & Northridge, 2000; Diez Roux, Jacobs, & Kiefe, 2002; Diez Roux, et al., 2001). Although several studies have found associations between residential SES and health, little is known about the mechanism by which neighborhood environment affects health (Diez Roux, 2001; S. Macintyre, A. Ellaway, & S. Cummins, 2002).

The underlying reason for such associations is speculated to be differences in the built environment and available resources across level of RSES. The built environment includes urban design factors, land use, transportation options, and the available recreational facilities and food outlets (Papas, et al., 2007). One aspect of the built environment that may directly influence diet is the distribution of food outlets: type, number, and location of food sources in an area. This is known as the food environment and includes sources of at-home foods (i.e., supermarkets and grocery stores) and away-from-home foods (i.e., restaurants and fast-food outlets). Some studies have assessed the indirect relationship between the food environment and CVD risk factors. For example, in a cross-sectional study of over 10,000 individuals from the Atherosclerosis Risk in Communities study, Morland et al. found that having at least one supermarket in a census track was associated with a lower prevalence of obesity whereas the presence of convenience stores was associated with a higher prevalence (Morland, Diez Roux, & Wing, 2006).

Some studies have shown that availability of healthy foods is less in disadvantaged areas. For example, in a study of Horowitz et al. compared the availability and cost of diabetes-healthy foods in a racial minority neighborhood with those of an adjacent more affluent and largely White neighborhood. They found that only 18% of the stores in high minority or poorer neighborhoods stocked recommended foods, compared to 58% in the adjacent neighborhood. Sloane et al. conducted surveys of market inventories in a predominantly African American, disadvantaged area and compared them with surveys from a mostly white, more affluent area. They found that the variety and quality of fresh fruit and vegetable produce was lower in the disadvantaged areas. Products such as 1% milk, skim milk, low-fat and nonfat cheese, soy milk, tofu, whole grain pasta and breads, and low-fat

meat and poultry items were significantly less available (Sloane, et al., 2003). Similarly, Wechsler et al. found that low-fat food options (e.g. low-fat milk) are less available as well. In addition, a study found that predominantly black neighborhoods have 2.4 fast-food restaurants per square mile compared to 1.5 restaurants in predominantly white neighborhoods.

Inequalities in the availability of healthy food options in disadvantaged areas may explain some of the racial and SES disparities in nutrition and related health outcomes. A number of studies have indicated that the availability of foods is related to purchasing decisions and dietary practices (Cheadle, et al., 1991; Glanz, et al., 1998; Morland, Wing, & Diez Roux, 2002). Cheadle et al surveyed grocery stores and quantified the amount of shelf-space occupied by healthy foods (i.e., low-fat and high-fiber products). They found that the shelf-space measures were good indicators of the diets of the people who shop there. Recent findings from a cross sectional study among pregnant women in North Carolina indicate that closer proximity to supermarkets is associated with higher quality diets (Laraia, Siega-Riz, Kaufman, & Jones, 2004). Further, in a study about perceived barriers to supermarket shopping, food stamp recipients cited that proximity and cost prevented them from shopping at supermarkets (Ohls, Ponza, Moreno L, Zambrowski, & Cohen, 1999).

Areas that have limited supply of foods that can be used to meet recommended guidelines for a healthy diet are known as food deserts (S. Cummins & Macintyre, 2006). A food desert does not imply that there is a shortage of food in an area but rather that the availability of healthy food options is limited. For example, obtaining low-fat dairy, fresh produce, and lean meats may be difficult where no grocery stores or supermarkets are available, even in the presence of convenience stores. Whether food deserts actually

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influence dietary intake is unclear. Current support for the idea that food access in disadvantaged neighborhoods is a strong determinant of diet is based on inconclusive data.

Not all food stores are created equal

Generally, larger stores such as supermarkets have superior selection, quality, and affordability of healthy foods compared to smaller or non-chain stores (D. Block & Kouba, 2006; Cheadle, et al., 1991; Drewnowski & Specter, 2004; Glanz, et al., 1998; Jetter & Cassady, 2006). Mantovani et al found that lowest priced items in supermarkets were 13 percent below lowest priced items in large groceries and 33 percent below lowest priced items in small groceries and convenience stores. Moreover, in a study that analyzed food store prices across inner-city and suburban communities, Chung and Myers found that non-chain stores charge higher premiums than supermarkets for comparable food items (Chung & Myers, 1999). Food prices are likely to be lower in supermarkets because supermarkets have lower store margins compared with smaller sized outlets, allowing for lower prices.

Distribution of food stores is linked to neighborhood SES

The differences among food stores are critical because the spatial distribution of supermarkets in an area differs according to race and SES composition (Baker, Schootman, Barnidge, & Kelly, 2006). Specifically, supermarkets tend to be more common in high SES and predominantly white neighborhoods, while grocery and non-chain stores are more common in low SES and predominantly black neighborhoods (Baker, et al., 2006; Zenk, et al., 2005). A study by Moore et al. found that low-income and predominantly black neighborhoods had four times as many grocery stores and half as many supermarkets compared to the wealthiest neighborhoods (L. V. Moore & Diez Roux, 2006). Similarly, they found that predominately minority neighborhoods had over twice as many grocery

stores and half as many supermarkets compared to predominately-white neighborhoods. However, even at the same neighborhood SES, blacks have less access to supermarkets than whites do. Zenk et al. observed that among the most impoverished Detroit neighborhoods, distance to the nearest supermarket varied considerably by percentage African Americans, with the nearest supermarket averaging 1.15 miles farther in neighborhoods with high proportions of blacks than in neighborhoods with low proportions of blacks (Zenk, et al., 2005).

A 1987 report from the U.S. House of Representatives Select Committee on Hunger suggested that supermarket migration to the suburbs, inadequate transportation, and lack of competitively priced food stores restricted low-income consumers' food buying power (*House Select Committee on Hunger. Obtaining Food: Shopping Constraints on the Poor, Committee Report*, 1987). For many people living in poor areas, long distances to supermarkets means they are more likely to shop at small grocery stores with less healthful food choices. Morland et al. found that in black neighborhoods, the presence of supermarkets was associated with meeting dietary recommendations (Morland, Wing, Diez Roux, & Poole, 2002). Specifically, fruit and vegetable intake increased by 30% with each additional supermarket in the census track. Similar associations were reported for other dietary components.

This evidence suggests that inequalities in access to food could translate into substandard dietary choices for minorities and the poor (Cheadle, et al., 1991; Drewnowski, 2004; Glanz, et al., 1998; Morland, Wing, & Diez Roux, 2002; Morland, Wing, Diez Roux, et al., 2002; Reicks, et al., 1994). Thus, an environment typical of a low-SES or predominantly black neighborhood—lower quality, reduced selection, and more expensive

foods—is not supportive of healthy eating habits. This may help explain why regardless of individual income or education, people in economically disadvantaged areas tend to have worse overall diet quality, while people in high SES areas are more likely to meet healthy eating guidelines (Baker, et al., 2006; Zenk, et al., 2005).

Food store availability and diet

The studies just described have shown that there are substantial variations in diet quality across neighborhoods, and that this variation is linked to neighborhood composition and availability of food stores. However, the presence of neighborhood differences in dietary patterns does not necessarily mean that the physical or social attributes of the neighborhood matter. It is suggested that the type of foods available in a store influence the dietary habits of customers. However, it is also possible that the type of foods consumers demand in economically disadvantaged areas differs from the foods consumers demand in other areas. For example, an increased demand for low-fat food items is likely to be reflected in the quantity of low-fat foods offered for sale and in the ways that such foods are advertised and displayed (Cheadle, et al., 1991).

Few longitudinal studies that have assessed diet before and after the introduction of a supermarket-type store. Unfortunately, their results do not provide clear answers. The first study (Wrigley N, 2003) evaluated the effect of significant changes in food-retail access on food consumption patterns in a food desert. Baseline dietary intake of the target population (a sample of people expected to shop at a new store) was compared to their intake one year after the change. A slight increase in fruit and vegetable consumption was reported for participants who switched to a new store. Another study (Steven Cummins, Petticrew, Higgins, Findlay, & Sparks, 2005) took a different approach by simulating a 'natural

experiment'. Their 'control' group was a similar neighborhood where new food outlets were not introduced for the duration of the study. Fruit and vegetable intake at baseline was compared to intake one year after the opening of the new store in both the 'intervention' and 'control' groups. They found no evidence of an improvement in fruit and vegetable consumption after accounting for confounders and no difference between the control and intervention groups.

However, studies to date have not provided convincing evidence that this is the case. Reasons include methodological problems in measurement of the food environment, limited geographic areas studied, and use of cross-sectional data. One limitation to cross-sectional studies is that although they may find consistent associations between residential SES, type of food outlets, and dietary habits, there is not indication of directionality. In other words, they do not help in understanding whether the low availability of foods options in low-income areas affects eating behavior and in turn body weight. A recent review summarized existing empirical research relating the built environment to obesity (Papas, et al., 2007). Only 20 studies met inclusion criteria, which was to 1) have a direct measure of body weight and 2) have an objective measure of the built environment Of those, two were longitudinal but only one found significant associations and it was in kindergarten-age children followed for 3 years (Ewing, Brownson, & Berrigan, 2006; Sturm & Datar, 2005).

Table 1. Long-term studies of the association between diet quality and weight gain[1]

Author/Year	Diet Index/Pattern	Sample characteristics	Diet and body weight association
I. Observational studies			
a. Diet Patterns congruent with the Dietary Guidelines for Americans			
(P. A. Quatromoni, et al., 2002)	Data-driven patterns: 'Heart Healthy' (closely approximates Food Guide Pyramid's recommendations), 'Light Eating', 'Wine & Moderate eating', 'High Fat', and 'Empty Calorie'	737 non-overweight White women, 30-89 y	Incidence of OW did not significantly differ between Heart Healthy cluster and other clusters[2]
(Newby, et al., 2003)[2]	Data-driven patterns: 'Healthy', 'White Bread', 'Alcohol', 'Sweets', and 'Meat & Potatoes'	459 men and women, 30-80 y	Annual BMI change in Healthy cluster only significantly different from change in 'Meat & Potatoes' cluster
(Togo, et al., 2004)	Data-driven patterns: 'Green'(healthy), 'Sweet', 'Traditional', 'Sweet/Traditional'	2,436 Danish adults, 30-60 y	No consistent association with WG or 11y OB risk
(Paula A. Quatromoni, et al., 2006)[2]	5-point index of adherence to 1995 U.S. Dietary Guidelines (based on intake of fat, saturated fat, cholesterol, sodium, carbohydrate)	2,245 mostly overweight/ obese White men and women, 30-89 y	High DQ (baseline) = ↓ 8y WG. Difference in WG between highest and lowest adherence categories was 2.4lbs (m) and 3lbs (w)[2]
b. Other definitions of healthy diet			
(Newby, Muller, Hallfrisch, Andres, & Tucker, 2004)	Data-driven patterns: 'Reduced-fat Dairy, Fruit, & Fiber', 'Protein & Alcohol', 'Sweets', 'Vegetables & Vegetable Fats', 'Fatty Meat', and 'Eggs, Bread & Soup'.	459 healthy men and women, 30-80 y	Reduced-fat Dairy, Fruit, & Fiber pattern inversely associated with annual change in BMI[2]
(Matthias B. Schulze, et al., 2005)	Data-driven pattern: 'Healthy': high consumption of whole-grain bread, fruits, fruit juices, grain flakes/ cereals, and raw vegetables, and low consumption of processed meat, butter, high-fat cheese, margarine, and meat	24,958 middle-aged European men and women	↑ food pattern score = less WG among non-obese subjects only[3]
(Mendez, et al., 2006)[2]	Index of adherence to Mediterranean Diet[4]	27,827 non-obese Spanish adults	↑DQ = ↓ 3y OB incidence among overweight adults
(Sanchez-Villegas, et al., 2005)[2]	Index of adherence to Mediterranean Diet[4]	6,319 Spanish adults	28 month WG was not predicted by baseline DQ or change in DQ
(M. B. Schulze, Fung, Manson, Willett, & Hu, 2006)[2]	Data-driven patterns: 'Western' (red and processed meats, refined grains, sweets, potatoes) and 'Prudent' (higher intake of fruits, vegetables, whole grains, fish, poultry, salad dressing)	51,670 White women, 26-46 y	↑Prudent score = lower 4y WG No difference in WG between women who maintained Western or Prudent patterns[2]
(Yannakoulia, et al., 2009)	Index of adherence to Mediterranean Diet[4]	1,364 Greek adults	No association between Mediterranean diet and OB incidence
II. Randomized, Controlled Dietary Interventions			
(Thomson, et al., 2005)	Both groups had good DQ to begin with; intervention group increased servings of fruits, vegetables, vegetable juice, fiber, and reduced alcohol. Control: Diet intake changed little	52 White women previously treated for breast cancer, 18-70 y	Both groups gained a little weight; improving DQ did not reduce WG.
(Barbara V. Howard, et al., 2006)	Intervention group achieved high adherence to DGA (↓fat, ↑fruits, vegetables, and grains). Control: small improvements in diet quality.	48,835 post-menopausal women (50-79 y), White, Black, Asian, etc.	Both groups maintained baseline weight. Significant effect of intervention was only seen among White women.

[1] Studies of free-living adults (>1 year follow-up) that used a comprehensive measure of diet quality (several aspects of diet), had a measure of weight change as main outcome, and adjusted estimates for known confounders. DQI= Diet Quality Index, HEI= Healthy Eating Index, MDS= Mediterranean Diet Score, FOS= Framingham Offspring-Spouse study, EI= energy intake, WG= weight gain, OW= overweight, OB= obesity (BMI≥30).

[2]adjusted for energy intake, physical activity, age, and smoking

[3]adjusted for physical activity, age, and smoking, but not energy intake

[4]Based on the traditional food consumption of the Mediterranean region, which includes plenty of vegetables, fruits, cereals, nuts, fish, olive oil, red wine (in moderation), and low intake of meat and dairy products.

III. METHODS

A.   STUDY POPULATION

We used data from the Coronary Artery Risk Development in Young Adults (CARDIA) study, a prospective epidemiologic study of the determinants and evolution of CVD risk factors among young adults. The baseline examination was conducted in 1985-86 and follow-up exams were conducted 2, 5, 7, 10, 15, and 20 years later. The initial cohort consisted of 5,115 young adults recruited from Birmingham, Alabama; Chicago, Illinois; Minneapolis, Minnesota; and Oakland, California and was balanced as to age (18-24, 25-30), gender, race (Black and White), and educational level (high school completion). CARDIA's data collection procedures follow a strict protocol to ensure both information accuracy and confidentiality. Data has been collected on a variety of topics regarding the physiology, attitudes, behaviors, SES, medical and family history, and environment of participants. The retention rate among survivors at year 20 was 72%.

B.   DESCRIPTION OF ENVIRONMENTAL DATA

Using a Geographic Information System (GIS), residential street addresses of CARDIA participants in 1992, 1995, and 2000 (exam years 7, 10, and 15, respectively) were geocoded and linked to time-varying environmental information from federal and commercial databases. Economic and socio-demographic information on participants'

neighborhoods was obtained from U.S. Census databases. Data from the 1990 U.S. census was used for the 1992 and 1995 exam years; data from the 2000 census was used for the 2000 exam year.

The retail food environment was characterized within an 8 km radius circle drawn around each participant's home address. This size buffer surrounding a person's residence was chosen based on empirical evidence that this distance would likely capture relevant diet-related facilities. For example, surveys from the U.S. Department of Transportation showed that in 1995, a standard shopping trip for the average U.S. family involved a six-mile drive (USDT, 2003), and low-income consumers tend not to travel more than 3 to 5 miles to purchase food (Ohls, et al., 1999). We used retrospective commercial databases from Dun & Bradstreet (DNB) to locate food stores within each participant's 8 km buffer for the time periods of interest. The original Standard Industrial Classification (SIC) codes used by the Census Bureau are only 4-digits and do not provide enough detail to correctly classify stores. The DNB codes contain a proprietary 4-digit extension to the original SIC codes which is used to characterize the stores in more detail. For example, SIC code 5411 refers to a general category for "grocery stores", defined as stores commonly known as supermarkets, food stores, and grocery stores, primarily engaged in the retail sale of all sorts of canned foods and dry goods, such as tea, coffee, spices, sugar, and flour; fresh fruits and vegetables; and fresh and prepared meats, fish, and poultry. This broad category includes convenience food stores, food markets, frozen food and freezer plans (except meat), grocery stores (with or without fresh meat), and supermarkets. DNB's 8-digit SIC codes were used to differentiate between types of food stores.

This study examined supermarkets (SIC codes 54110100, 54110101, 54110102, 54110103, 54110104, and 54110105) and grocery stores (SIC codes 54110000, 54119900, 54119903, 54119904, and 54119905) separately. These two types of establishments differ in sales volume (in 2000, supermarkets averaged 46 times the sales volume of grocery stores), size (supermarkets have larger square footage and have about 7 times more employees), and the availability of on-site services (Powell, Slater, Mirtcheva, Bao, & Chaloupka, 2007). Smaller specialty stores (e.g., fruit and vegetable markets, meat markets) were not studied because there were too few to make into a separate category. Convenience stores (defined as food marts with or without a gas station, SIC codes: 54110200, 54110201, 54110202, 55410000, 55419900, 55419901) were excluded because this study aims to quantify access to healthy foods and the foods necessary to meet dietary recommendations (e.g., 1% milk, skim milk, low-fat and nonfat cheese, whole grain pasta and breads, and low-fat meat and poultry) are not often found at these types of stores (Sloane, et al., 2003).

SIC code	Description
54110000	Grocery stores
54119900	Grocery stores, nec
54119903	Frozen food and freezer plans, except meat
54119904	Grocery stores, chain
54119905	Grocery stores, independent
54110100	Supermarkets
54110101	Supermarkets, chain
54110102	Supermarkets, greater than 100,000 square feet (hypermarket)
54110103	Supermarkets, independent
54110104	Supermarkets, 55,000 - 65,000 square feet (superstore)
54110105	Supermarkets, 66,000 - 99,000 square feet
54319902	Vegetable stands or markets
54210200	Meat markets, including freezer provisioners

54210201	Food and freezer plans, meat
54210202	Freezer provisioners, meat
54210000	Meat and fish markets
54210100	Fish and seafood markets
54210101	Fish markets
54210102	Seafood markets
54510000	Dairy products stores

Environmental databases are vulnerable to errors due to incomplete records and out of date records. In the creation of respondent-specific environmental variables, geocoding error and inaccuracies in the street files are additional sources of error. Some research suggests that these errors exist within national business databases (such as those from Dun & Bradstreet) when measurements of these spatial datasets were validated against actual visual measurements in specific areas, but the errors seem to be random (Boone, Gordon-Larsen, Stewart, & Popkin, 2008). Boone et al. compared a commercial database of physical activity facilities to a field census in two communities and found that main sources of error resulted from facilities not included or misclassified as physical activity facilities in the commercial database. Since there is little evidence that disagreement between the two sources is correlated with the socioeconomic or demographic characteristics of neighborhoods (Bader, Ailshire, Morenoff, & House, 2010), our estimates are not likely to be biased by these errors (but they may be attenuated).

B.   ASSESSMENT OF DIET QUALITY

The CARDIA Diet History is an interviewer-administered instrument that queries usual dietary practices and includes a comprehensive food-frequency questionnaire (FFQ)

encompassing the last 28 days. The FFQ included over 700 food items and questions about

brand names, preparation methods, and frequency of consumption. Nutrient and energy

intakes were computed by the Nutrition Coordinating Center (NCC) at the University of

Minnesota. The most recent available version of the NCC database was used to calculate

nutrient intakes at each study year (version 10 for baseline and version 20 for year 7).

Additional details about the quality control and validation of the CARDIA Diet History are

available elsewhere (Liu, Slattery, Jacobs, Cutter, McDonald, Van Horn, Hilner, Caan,

Bragg, Dyer, et al., 1994; Arline McDonald, et al., 1991; Slattery, et al., 1994).

The original Diet Quality Index (DQI), published in 1994, was designed to evaluate

the overall quality of diet based on the 1989 recommendations by the National Academy of

Sciences Food and Nutrition Board (R. E. Patterson, Haines, & Popkin, 1994). It has since

been revised in adaptation to different populations and to reflect changes in nutrition

knowledge (Haines, Siega-Riz, & Popkin, 1999; Kim, Haines, Siega-Riz, & Popkin, 2003;

Laraia, et al., 2004; Popkin, Siega-Riz, & Haines, 1996).   The dietary assessment tool used

in this study is based on one of these revisions, the DQI-R, a validated instrument that

quantifies adherence to the 1995 Dietary Guidelines for Americans (Haines, et al., 1999).

Specifically, the DQI-R evaluates diet based on eating a variety of foods and meeting the

dietary recommendations for the intake of calcium, iron, fat, saturated fat, cholesterol, grains,

fruits, and vegetables, as well as moderation in the intake of added sugars, fats, alcohol, and

salt. A new index was created to use with the CARDIA Diet History and to reflect the

messages conveyed by the 2005 Dietary Guidelines for Americans (USDA, 2005). We will

refer to this new index as the 2005 DQI to differentiate from earlier versions. The major

differences between the 2005 DQI and the DQI-R are as follows: (1) Data was collected

through FFQs whereas the DQI-R used repeated 24-hour recalls. FFQs are designed to measure long-term dietary intake but provide less detailed information to compute nutrient values. (2) Iron was removed as an indicator of diet quality because it is not a nutrient of concern in this population of young adults who are more at risk for over- rather than under-nutrition. (3) The scoring for consumption of grains was modified to reflect an emphasis in whole grains as opposed to refined grains. (4) Reduction in the intake of sugars was a key message of the 2005 DGA hence a separate component was created to address this recommendation. (5) The dairy component was modified to include only reduced-fat milk because the 2005 DGA emphasize reduced fat alternatives. **Table 5** shows each of the CARDIA DQI components and how they were scored. More details are discussed on page 50.

IV. NEIGHBORHOOD POVERTY AND FOOD STORE AVAILABILITY

A.   ABSTRACT

Background: The cross-sectional literature suggests reduced access to supermarkets in economically-deprived areas, which has led to policy efforts to increase supermarket access in low income areas. However, this cross-sectional literature has heretofore been unable to control for unmeasured neighborhood- and individual-level factors that could bias estimates.

Objective: To examine the longitudinal association between census block group poverty and race/ethnicity and neighborhood-level availability of supermarkets and grocery stores.

Methods: We used prospective data from the Coronary Artery Risk Development in Young Adults (CARDIA) study, a cohort of Black and White Americans aged 18-30 at baseline (1985-86) from four US cities. Participants' addresses in 1992, 1995, and 2000 were geocoded and then spatially and temporally linked to data from the U.S. census for neighborhood socio-demographics and Dun & Bradstreet for the number of supermarkets or grocery stores per 10,000 people living within 5 miles of each participant's residence at each time point. We tested for confounding by time-varying participants' characteristics (i.e., age, education, income) and used repeated measures, conditional regression modeling to control for time-constant unmeasured characteristics.

Results: We found a significant interaction between neighborhood poverty and minority composition, but no confounding by time-varying, individual-level characteristics.

Controlling for time-constant unmeasured characteristics, we observed higher access to supermarkets and grocery stores in high minority, high poverty neighborhoods. Conclusion: Our finding of lack of confirmation of food deserts is in contrast with the U.S. cross-sectional literature, suggesting that complex social and economic processes underlying the relationship between neighborhood socio-demographic composition and the food environment are not adequately captured by cross-sectional data.

B. INTRODUCTION

Areas that have limited supply of foods that can be used to meet recommended guidelines for a healthy diet are known as food deserts (Clarke 2002, Cummins 2002). A food desert does not imply that there is a shortage of food in an area, but rather that the availability of healthy foods is limited. A recent focus has been on the distribution of larger supermarkets, which in contrast to grocery stores, have considerably higher sales volume, larger square footage, more employees and more on-site services (Powell, et al., 2007). In general, larger stores such as supermarkets have superior selection, quality, and affordability of healthy foods compared to smaller food stores (i.e, grocery stores) (D. Block & Kouba, 2006; Cheadle, et al., 1991; Chung & Myers, 1999; Drewnowski & Specter, 2004; Glanz, et al., 1998; Jetter & Cassady, 2006). The larger physical size of supermarkets contributes to lower store margins compared with smaller sized stores and it also allows for greater product variety, including many lower cost private-label and generic items (Kaufman, Cooper, & McGee, 1997).

These differences between food stores are critical because the spatial distribution of supermarkets in an area may differ according to ethnic composition and socio-economic

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status (SES). Several cross-sectional studies have found that supermarkets tend to be more common in high SES and predominantly White census tracts or zip codes, while grocery stores are more common in low SES and predominantly Black areas (Baker, et al., 2006; L. V. Moore & Diez Roux, 2006; Morland & Filomena, 2007; Morland, Wing, Diez Roux, et al., 2002; Powell, et al., 2007; Zenk, et al., 2005). Moreover, Zenk et al. found that among the most impoverished Detroit neighborhoods, distance to the nearest supermarket averaged 1.15 miles farther in predominantly Black census tracts than in predominantly White areas (Zenk, et al., 2005).

Hence, people in low-income areas, who have a greater disease burden, may also have the worst access to healthy foods. Since studies have found a correlation between what people eat and the foods available in their communities (Cheadle, et al., 1991; L. V. Moore, et al., 2008; Morland, Wing, & Diez Roux, 2002), lack of access to healthy foods has been hypothesized to be a determinant of diet, and initiatives to encourage the development of grocery retail investments in low-income communities have been called for (Pothukuchi, 2005; Story, et al., 2008). However, most of these studies are cross-sectional and do not include modeling strategies to control for unmeasured neighborhood- and individual-level factors, which could potentially bias estimates of the neighborhood SES and food environment association. In contrast, longitudinal studies with repeated measures on individuals can be used to estimate changes in a person's environment while controlling for observed and unobserved time-constant characteristics by using each person as his own control (Boone-Heinonen, Gordon-Larsen, Guilkey, Jacobs Jr, & Popkin, 2009).

To this end, we use a large, geographically-diverse cohort followed over 8 years to examine the longitudinal association between neighborhood poverty and the availability of

supermarkets and grocery stores within 5 miles of each respondent at each time period. We hypothesized that high poverty neighborhoods would have lower availability of supermarkets but higher availability of grocery stores. Further, we hypothesized that those relationships would vary depending on the ethnic composition of the neighborhood.

C.   SUBJECTS AND METHODS

Data source and sample

We used data from the Coronary Artery Risk Development in Young Adults (CARDIA) study, a prospective epidemiologic study of the determinants and evolution of cardiovascular disease risk factors among young adults. The initial cohort consisted of 5,115 young adults recruited from Birmingham, Alabama; Chicago, Illinois; Minneapolis, Minnesota; and Oakland, California in 1985-86 and followed over a 25-year period. The sample was balanced with regard to age (18-24, 25-30), gender, race (Black and White), and educational status (high school graduate or less, more than a high school education). Specific recruitment procedures have been described elsewhere (Hughes, et al., 1987). After excluding participants for whom census data was missing, our sample size was 5,108 at year 7, 5,105 at year 10, and 5,101 at year 15 (total of 15,314 observations). This secondary data analysis was approved by the CARDIA Steering committee and the Institutional Review Board of University of North Carolina at Chapel Hill.

At each exam year, the current residential street address for each respondent was recorded; we then geocoded the street addresses for each respondent at each exam period. Using a Geographic Information System (GIS), residential street addresses of CARDIA participants for exam years 7 (1992), 10 (1995), and 15 (2000) were linked to time-varying

neighborhood information from federal and commercial databases. We did not use the

baseline examination (1985) because the external food store database was not available at the

same level of detail for that year, this causing a discrepancy in measurement of food stores

between 1985 and all other exam years. Over this time period the CARDIA respondents

moved from baseline in four U.S. metropolitan areas (700 census tracts) to 48 states, 1

federal district, 1 territory, 529 Counties and 3,805 Census Tracts (48% moving residential

locations between 0 and 7, 69% between 7 and 10, and 33% between 10 and 15).

Analysis variables

Census data

Data from the 1990 U.S. census was used for the 1992 and 1995 exam years; data

from the 2000 census was used for the 2000 exam year. Census block groups (national

administrative boundaries containing approximately 1,500 individuals) were used as proxies

for neighborhoods because they are smaller than the commonly used census tract (or zip

code) and thus more likely to fit individually-perceived neighborhood boundaries (O'Campo,

2003). Block groups were classified as urban/non-urban based on the census definition of

urbanized areas, which in 2000 were defined as having a population density of at least 1,000

people per square mile and surrounding census blocks that have an overall density of at least

500 people per square mile. Neighborhood poverty was defined as the percent of people in a

block group living under the U.S. federal poverty level. This measure of economic

deprivation seems to be the most robust for a variety of health outcomes (Krieger, Chen,

Waterman, Rehkopf, & Subramanian, 2003) and it is comparable over time. It was

categorized into low, medium and high poverty, corresponding to <10%, 10-20%, and ≥20%

of census block group residents living under the poverty line, based on the federal definition

of high poverty area (Subramanian, Chen, Rehkopf, Waterman, & Krieger, 2005). We defined neighborhood racial and ethnic minority population as the percent of the population in a census block group who self-classify as other than non-Hispanic White.

 Food stores

Retrospective commercial databases from Dun & Bradstreet (DNB) were used to locate food stores near participants' residences for the time periods of interest. DNB's 8-digit SIC codes were used to differentiate between types of food stores. We examined supermarkets, large stores (mostly chains) including places like Albertson's, Kroger, Harris Teeter, and Whole Foods Market (SIC codes 54110100, 54110101, 54110102, 54110103, 54110104, and 54110105) and grocery stores, which are smaller than supermarkets and include both chain and independent grocers, but not convenience stores (SIC codes 54110000, 54119900, 54119903, 54119904, and 54119905). We did not include smaller specialty stores (e.g., fruit and vegetable markets, meat markets) because there were too few to make into a separate category. Convenience stores (with or without a gas station) were excluded because we aimed to quantify availability of 'healthy' foods (i.e., foods necessary to meet dietary recommendations), which are not often found at these types of stores (Sloane, et al., 2003).

We operationalized availability of food stores as the number of either supermarkets or grocery stores per 10,000 people living within an 8 km (5 mile) Euclidean distance from each participant's residence at each exam period. Hence, each participant had time-varying, unique estimates of supermarket and grocery store availability. Food stores were assessed on a per capita basis to allow comparability across different geographic areas with varying population density. Previous work guided our use of the 8 km Euclidean radius for

availability of food stores within this buffer (relative to 1-, 3-, and 5-k buffers). This work

showed the 8 km buffer size was associated with significantly improved diet quality (Boone-

Heinonen, et al., 2010). Moreover, surveys from the U.S. Department of Transportation

showed that in 1995, a standard shopping trip for the average U.S. family involved a six-mile

drive (USDT, 2003), but low-income consumers may not travel more than 3 to 5 miles to

purchase food (Ohls, et al., 1999).

<u>Individual-level characteristics</u>

Standard questionnaires were used at each exam year to assess socio-demographic

characteristics. In the case of missing data, data from previous years was imputed.

Education was defined as the number of years in school completed. Household income was

categorized into <$25,000, $25,000-$50,000, and >$50,000. We categorized participants

into four categories of "household structure": married without children, single without

children, married with children, and single with children.

Statistical analysis

All analyses were conducted with Stata version 10 statistical software (College

Station, TX). Characteristics of participants and their environment were compared across

categories of neighborhood poverty (significant differences were determined using ANOVA,

$\alpha=0.05$). We estimated associations between neighborhood poverty and minority

composition on food store density with longitudinal, repeated measures conditional

regression linear models. These models, conditioned on the subject, do not estimate

parameters for variables constant within subject (i.e., race, sex, and study center), but have

the advantage of adjusting for potential confounding by all measured *and* unmeasured

characteristics of individuals (or within-person effects). These models effectively subtract the

within-subject mean value of each variable (Boone-Heinonen, et al., 2009), thereby controlling for time-constant characteristics of each individual (e.g., race, sex, health-related attitudes and behaviors); even those that are unobserved and hence omitted from analysis. In addition, these models adjust for the correlation between repeated observations taken in the same subject and have the advantage of handling longitudinal data on subjects with varying number and unequally spaced observations, thereby allowing for inclusion of the maximum number of data points, in this case 8 years of follow-up (Baltagi, 2001; Greene, 2003; Hsiao, 2003).

Studies that use a combination of measurements from individuals and their environments as independent variables often use multilevel modeling methods (Diez-Roux, 2000; Diez Roux, 2002; Pickett & Pearl, 2001). Such models are appropriate for data with nested sources of variability—that is, involving units at a lower level (e.g., individuals) nested within units at a higher level (e.g., neighborhoods) because it allows the simultaneous examination of effects from variables at multiple levels on individual-level outcomes while accounting for the non-independence of individuals within a group. CARDIA does not have a nested study design. For example, at year 0, participants lived in 799 block groups while at year 15, participants lived in 3,461 different block groups, with an average of 1.5 participants (range: 1-15) per block group. Thus, we did not use a nested approach because our data are sparse (few individuals on average within block groups) and unbalanced (variable number of individuals within block groups) at the beginning of the study and become more so over time as participants move residences (Clarke, 2008). Although our primary source of clustering is on the individual (repeated observations over time), our longitudinal, repeated measures

conditional regression models also adjust for higher level clustering by neighborhood (Diez-Roux, 2000).

Variables were considered confounders if they changed any of the main exposure coefficients by at least 10%. We tested for confounding by census block urbanicity and time-varying characteristics of the participants (age, education, household income, and family structure) using a backward elimination approach. None of the individual-level factors met our criteria for confounding; thus, only urbanicity was included in our models as a confounder. Effect modification by neighborhood minority composition was assessed through the inclusion of interaction terms and included in final models if the likelihood ratio test was significant at $\alpha = 0.10$. To address non-linearity (assessed graphically), neighborhood minority composition was categorized into low, medium, and high minority, corresponding to $\geq 75\%$, 50-75%, and <50% of census block group residents who are non-Hispanic White. Food store densities were natural log-transformed to address skewness, hence, model coefficients were interpreted as the percent change in food store density expected from a change in the categories of the corresponding independent variable.

D. RESULTS

Participants in low-poverty neighborhoods were generally older, more educated, and were more likely to report household income greater than $50,000 compared to participants in higher poverty neighborhoods (**Table 2**). High poverty neighborhoods had the highest proportion of ethnic minority population (**Table 3**). Population density, supermarket and grocery store raw counts, and grocery store density were higher with higher neighborhood poverty. Supermarket density did not vary across categories of neighborhood poverty.

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Using longitudinal, repeated measures conditional regression models, we found a significant interaction between neighborhood poverty and minority composition (likelihood ratio test p<0.01 for supermarkets, p<0.01 for grocery stores). Hypothesized disparities by neighborhood poverty of the availability of supermarkets were only evident in low-minority areas (**Table 4**; medium vs. low poverty p<0.01, high vs. low poverty p=0.06). Overall, living in high minority, high poverty, neighborhoods was associated with slightly higher supermarket density. The relationship between neighborhood poverty and grocery store density was more stable across strata of neighborhood minority composition. Overall, higher poverty or minority population of the neighborhood was associated with higher grocery store density.

E.   DISCUSSION

Following a large sample of young adults as they move across America, we found that neighborhoods with higher poverty and ethnic minority composition had higher availability of supermarkets and grocery stores (controlling for time-constant individual characteristics). Our findings for supermarkets are in contrast with the U.S. cross-sectional literature showing reduced access to supermarkets in economically-deprived areas (Beaulac, et al., 2009). For example, Moore et al. found that among selected census tracts in North Carolina, Maryland, and New York, low-income areas had half as many supermarkets compared to the wealthiest areas, and predominately minority areas had half as many supermarkets compared to predominately-white areas (L. V. Moore & Diez Roux, 2006). Whereas our findings for grocery stores, which suggest higher grocery store density with

higher poverty and minority population, confirm the cross-sectional literature (L. V. Moore & Diez Roux, 2006; Morland, Wing, Diez Roux, et al., 2002).

While living in an economically-deprived area has been linked with the development of obesity, metabolic syndrome, and cardiovascular disease, independently of individual-level factors (Diez-Roux, et al., 2001; Diez-Roux, et al., 2000; Diez Roux, et al., 2002; Diez Roux, et al., 2001), relatively little is known about the mechanism by which the socioeconomic environment affects health (Diez Roux, 2001; S. Macintyre, et al., 2002). One hypothesis is that these relationships are in part caused by the lower availability of healthy foods in low-income neighborhoods (Glanz, et al., 1998; Morland, Wing, & Diez Roux, 2002; Papas, et al., 2007).

The idea that food deserts have contributed to the obesity epidemic has led to recommendations to bring grocers to underserved areas (Khan, et al., 2009). These initiatives are based on two important assumptions: 1) that poor and minority populations are disproportionately affected by food deserts; and 2) that the observed correlations between the food environment and the diet of people who live there are causal in one direction (the food environment determines diet, rather than diet of people in an area determines the what foods are available). An alternative hypothesis is that individual choice taking place within the neighborhood context drives demand for certain food items, which then become more available in those environments. For example, healthy foods are generally more expensive (e.g., lean meats, fish, fresh vegetables and fruits) or may require more preparation (e.g., legumes, whole grains) than energy-dense, processed foods (Drewnowski & Specter, 2004). In addition, for a variety of reasons, what are conceived of as unhealthy foods (e.g., fatty meats, fried foods, sugary-fatty foods) by public health researchers, might be considered

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desirable by the poor (Popkin, et al., 1996; Popkin, et al., 2003). Hence, people living in poor neighborhoods (i.e., the working poor) may be less likely to purchase these foods if they perceive the price or preparation requirements to be too high or desire for other reasons these "unhealthy" foods. (Blisard, Stewart, & Jolliffe, 2004), regardless of what foods are actually available, and in the process create a higher demand for less healthy foods (Cheadle, et al., 1991; Gittelsohn, et al., 2008). Indeed, two experiments in England showed that placement of supermarkets in low income areas did not change purchasing behavior of the lower income individuals (Steven Cummins & Macintyre, 2002; S. Cummins, M. Petticrew, C. Higgins, A. Findlay, & L. Sparks, 2005; S Macintyre, A Ellaway, & S Cummins, 2002). Lack of appropriate methods to address these complex choices can provide biased estimates of the effect of neighborhood poverty on the food environment(National Research Council, 2009). Thus, if differences in demand caused an imbalance in the distribution of food stores, opening new food stores in underserved areas will not solve the problem of poor dietary quality. In fact, there is little evidence that people improve their diets in response to increased availability of healthy foods without a price incentive to do so (Seymour, Lazarus Yaroch, Serdula, Blanck, & Khan, 2004; M. C. Wang, et al., 2008).

Our longitudinal, repeated measures conditional regression modeling strategy, in which we take advantage of variation within person, over time, to control for time invariant unmeasured characteristics, suggest that individual characteristics played a role in residential selection related to food stores. These types of factors might be related to both living in an economically-deprived neighborhood (e.g., attitudes toward health, social resources, race) as well as living in an area with a given supermarket density (Boone-Heinonen, et al., 2009).

Yet, it is important to note that our analysis does not explicitly model residential choice and is thus a first step in understanding these complex relationships.

There are several limitations to our study: (1) While we observed an association between neighborhood socio-demographic characteristics and availability of food stores, we cannot be certain of the direction of this relationship; (2) Use of retrospective contextual data. The accuracy of our food store data is dependent on obtaining accurate information from archived commercial datasets. Some research suggests that there are errors within national business databases (such as those from Dun & Bradstreet) when measurements of these spatial datasets were validated against actual visual measurements in specific areas, but the errors seem to be random (Boone, et al., 2008). Boone et al. compared a commercial database of physical activity facilities to a field census in two communities and found that main sources of error resulted from facilities not included or misclassified as physical activity facilities in the commercial database. Since there is little evidence that disagreement between the two sources is correlated with the socioeconomic or demographic characteristics of neighborhoods (Bader, et al., 2010), our estimates are not likely to be biased (but they may be attenuated). More research is needed to validate the food store environment measures obtained from commercial databases. (3) We use the presence of supermarkets as proxies for the availability of healthy foods, without detailed information about the products sold in these supermarkets. Also, Standard Industry Classification codes that we used in this study may not provide a precise categorization of supermarkets and grocery stores (L. V. Moore, et al., 2008); however, neighborhood audits are unfeasible for large studies spanning the US with retrospective data, such as ours. (4) We assume that the residential address is the central point of reference for food shopping, yet some people shop after work or in combination with

other places they frequent during their day. (5) It is possible that our buffer size (8 km) may be too large, especially for poor people who do not own a car and may do most of their shopping closer to home. However, in our preliminary analysis we found an association between food store availability within the 8 km buffer size (relative to 1, 3, and 5 km Euclidean buffers) and overall dietary intake (Boone-Heinonen, et al., 2010).

One key strength of our study is our use of a longitudinal, geographically-diverse dataset with time-varying individual data geographically linked to the residential neighborhood locations of all study participants. Further, we captured availability of supermarket and grocery stores within a 5-mile area surrounding each individual respondent's residence at each time period. Thus, we are able to explicitly capture the neighborhood of each respondent (most previous studies have defined availability using census tract or zip code boundaries). To our knowledge, this is the first longitudinal analysis assessing the association between poverty and supermarket availability.

In summary, our longitudinal results suggest that the problem of food deserts in low-income areas may not be generalizable to all urban areas in the U.S. as previous studies have suggested. Our findings suggest that there may be complex social and economic processes underlying the relation between neighborhood composition and the food environment that are not captured by cross-sectional data. Thus, we view our analyses as an exploratory first step in better understanding how demographic considerations of the spatial area could be factored into empirical research to examine correlates of food deserts. Much more attention must be given to the demand side of the food consumption equation, namely, what will it take to get low income and minority populations to purchase and consume healthier foods.

Table 2. Characteristics of CARDIA participants at exam year 7 (1992-93)[1]

	Neighborhood Poverty[2]		
	Low Poverty	Medium Poverty	High Poverty
	n= 2172	n= 1225	n= 1711
Age, years	32.2 (3.4)	31.8 (3.6)	31.4 (3.8)[3]
Education, years	15.0 (2.5)	14.5 (2.4)	13.4 (2.2)[3]
Income, %			
<$25,000	20.1	32.4	46.2
$25,000-$50,000	34.6	36.6	28.6
>$50,000	39.2	21.3	13.3[3]
Married, %	60.4	49.4	41.2[3]
Children at home, %	42.5	44.6	47.5[3]

[1]Values are means (SD) or %.
[2]Low, medium, and high poverty areas: <10%, 10-19%, and ≥20% of census block group population living under poverty line, respectively.
[3]Indicates significant difference across poverty categories (ANOVA or Chi^2, α=0.05).

Table 3. Characteristics of the residential environments of CARDIA participants at exam years 7 (1992-93), 10 (1995-96), and 15 (2000-01)[1]

	Neighborhood Poverty		
	Low Poverty	Medium Poverty	High Poverty
Year: 1992	n= 2172	n= 1225	n= 1711
Population density[3,4]	2251 (2196)	2461 (1880)	2631 (1906)[2]
% ethnic minority in neighborhood	22.3 (24.3)	50.1 (32.0)	75.9 (28.4)[2]
Supermarkets, raw count[3]	17.6 (19.6)	21.2 (17.7)	22.6 (16.1)[2]
Supermarket density [3,5]	0.54 (0.26)	0.57 (0.30)	0.55 (0.26)
Grocery store, raw count[3]	129.2 (153.7)	156.2 (149.2)	190.8 (170.8)[2]
Grocery store density[3,5]	2.79 (1.35)	3.14 (1.12)	3.43 (1.10)[2]
Living in non-urban area, %	1.9	2.0	1.2[2]
Moved since recruitment into study, %	58.0	46.5	36.9[2]
Year: 1995	n= 2644	n= 1168	n= 1289
Population density[3,4]	1521 (1654)	1924 (1717)	2404 (1929)[2]
% ethnic minority in neighborhood	19.5 (22.5)	43.9 (32.6)	72.2 (30.2)[2]
Supermarkets, raw count[3]	12.5 (18.4)	14.9 (14.0)	16.4 (15.5)[2]
Supermarket density [3,5]	0.52 (0.33)	0.51 (0.33)	0.46 (0.31)[2]
Grocery store, raw count[3]	66.8 (106.8)	93.4 (106.3)	132.5 (133.2)[2]
Grocery store density[3,5]	1.99 (0.97)	2.54 (1.60)	2.73 (0.93)[2]
Living in non-urban area, %	3.5	6.1	2.9
Moved since recruitment into study, %	89.3	80.7	70.4[2]
Year: 2000	n= 2653	n= 1165	n= 1287
Population density[3,4]	1525 (1698)	1967 (1775)	2371 (2071)[2]
% ethnic minority in neighborhood	26.2 (23.7)	54.9 (29.9)	80.9 (24.1)[2]
Supermarkets, raw count[3]	13.9 (20.0)	17.2 (15.0)	19.77 (19.71)[2]
Supermarket density [3,5]	0.54 (0.34)	0.54 (0.34)	0.53 (0.27)
Grocery store, raw count[3]	85.1 (129.3)	112.6 (116.1)	144.7 (151.4)[2]
Grocery store density[3,5]	2.72 (1.16)	3.13 (1.27)	3.31 (1.17)[2]
Living in non-urban area, %	3.4	5.1	2.0[2]
Moved since recruitment into study, %	91.8	83.6	73.7[2]

[1]Values are means (SD) or %
[2]Indicates significant difference across poverty categories (ANOVA or Chi[2], $\alpha=0.05$)
[3]Based on 8 km Euclidian radius from participant's residence.
[4]Population per km^2.
[5]Food store density= # of stores per 10,000 people living within 8km buffer zone

Table 4. Association between neighborhood poverty, ethnic minority composition, and food store density[1] [coefficient (95% CI)]

Ethnic minority composition[2]	Low Poverty[3]	Medium Poverty	High Poverty
Supermarkets			
Low	Reference	-0.017 (-0.029, -0.006)	-0.018 (-0.038, 0.001)
Medium	-0.001 (-0.011, 0.011)	0.008 (-0.006, 0.021)	-0.008 (-0.024, 0.008)
High	-0.009 (-0.023, 0.004)	0.009 (-0.003, 0.021)	0.011 (0.003, 0.022)
Grocery stores			
Low	Reference	0.119 (0.094, 0.143)	0.015 (-0.025, 0.055)
Medium	0.065 (0.042, 0.088)	0.147 (0.119, 0.176)	0.162 (0.162, 0.229)
High	0.124 (0.096, 0.152)	0.192 (0.167, 0.216)	0.239 (0.217, 0.262)

[1]Food store density= natural log [# of supermarkets or grocery stores per 10,000 people living within 8km of participants' residence]. Based on longitudinal, repeated measures conditional regression models with interactions between poverty and ethnic composition of neighborhood and adjusted for exam year and urbanicity.

[2]Low, medium, and high minority areas: >75%, 50-75%, and <50% of census block group population classified as non-Hispanic White, respectively.

[3]Low, medium, and high poverty areas: <10%, 10-19%, and ≥20% of census block group population living under poverty line, respectively.

IV. DIET QUALITY AND WEIGHT GAIN

A.   ABSTRACT

Background: Little is known about the long-term health consequences of following the 2005 Dietary Guidelines for Americans (DGA).

Objective: To examine the longitudinal association between diets consistent with the 2005 DGA and subsequent weight gain.

Design: We used data from the CARDIA Study, a cohort of Black and White men and women, aged 18-30 years at baseline, who attended up to seven examinations from 1985-86 to 2005-06 (n= 4,913). We created a 100-point Diet Quality Index (2005 DQI) to rate participants' diets based on meeting the 2005 DGA key recommendations. Longitudinal models of weight gain were adjusted for physical activity, smoking, energy intake, age, education, gender, and initial BMI, and included interaction terms of DQI by race and initial BMI (if statistically significant).

Results: We found effect modification by race (likelihood ratio test $p<0.03$ in all models). The mean adjusted 20-year weight change (kg) was +19.4 for Blacks and +11.2 for Whites with high diet quality (DQI>70), +17.8 for Blacks and +13.9 for Whites with DQI<50 ($p<0.05$). In race-specific Cox models (with interaction terms for DQI* initial BMI, $p<0.05$), a 10-point increase in DQI score was associated with 10% lower risk of gaining 10 kg in Whites with initial BMI <25 kg/m^2 but with 15% higher risk in Blacks with baseline obesity ($p<0.001$).

Conclusions: Our findings do not support the hypothesis that a diet consistent with the 2005 DGA benefits long-term weight maintenance among American young adults. Greater need for attention to obesity prevention in future DGA is warranted.

B.   INTRODUCTION

Many different ways to characterize a healthy diet have been used, yet there is no consensus as to what the best definition is. One commonly used definition of a healthy diet is adherence to the Dietary Guidelines for Americans (DGA). Since the DGA are intended to promote health and reduce risk of chronic disease (2005), it is often assumed that they can help prevent weight gain. Indeed, people who adhere to the 2005 version of the guidelines may have lower energy intakes (X. Gao, Wilde, Lichtenstein, & Tucker, 2006). Yet, there is little evidence that people who have followed the DGA (or similar dietary patterns) actually gained less weight over a long period of time. One reason may be that past dietary guidelines were not intended to prevent weight gain the population. Another reason may be that the knowledge base used in creating the guidelines was limited. For example, the 2005 DGA were mostly based on studies that reduce diets to individual components (e.g., grams of fiber, percentage energy from fat) (King, 2007). This poses a challenge because, due to the complex interactions among known and unknown food components (Togo, Osler, S›rensen, et al., 2001), the relationship between a single dietary component and disease may differ when all aspects of the diet are considered (Jacobs & Steffen, 2003).

For the most part, the studies that found inverse associations between adherence to the DGA and body weight or obesity have been short-term or cross-sectional (Berg, et al., 2008; S. K. Gao, et al., 2008; Guo, et al., 2004; Kant & Graubard, 2005; Togo, Osler,

S›rensen, et al., 2001). We found seven longitudinal studies of the association between diets consistent with the DGA and subsequent changes in body weight. While two studies found that diets consistent with the 2005 DGA were associated with lower weight gain in Whites (Barbara V. Howard, et al., 2006; Paula A. Quatromoni, et al., 2006), the rest produced inconsistent results where DGA-like dietary patterns were not clearly better at preventing weight gain compared to other patterns (Burke, et al., 2003; Newby, et al., 2003; P. A. Quatromoni, et al., 2002; Thomson, et al., 2005; Togo, et al., 2004). An important limitation of this literature is that most of these studies were performed in subjects who were White and thus findings may not be generalizable to African Americans, who are at highest risk for obesity (Ogden, et al., 2006). Further, findings from studies performed in subjects who are older (past the age of highest risk for weight gain) or who are already overweight or obese, may not be applicable to young and normal-weight adults (Bes-Rastrollo, et al., 2006; Barbara V. Howard, et al., 2006; Mendez, et al., 2006; Miller & Parsonage, 1975; Segal, Gutin, Nyman, & Pi-Sunyer, 1985; Wang, et al., 2008).

We used longitudinal data from a cohort of Black and White young adults to (1) create an index of overall diet quality based on the key messages conveyed by the 2005 DGA, (2) examine the association between diet quality and 20-year risk of weight gain, and (3) determine whether diet quality had the same association with body weight regardless of participants' race or initial body mass index (BMI). We hypothesized that higher diet quality would be associated with less weight gain, and that this association would be stronger in Whites and in those with normal-weight (BMI <25 kg/m^2) at baseline.

C.   SUBJECTS AND METHODS

Data source and sample

We used data from the Coronary Artery Risk Development in Young Adults (CARDIA) study, a prospective epidemiologic study of the determinants and evolution of cardiovascular disease risk factors among young adults. The baseline examination was conducted in 1985-86 and follow-up exams were conducted 2, 5, 7, 10, 15, and 20 years later. The initial cohort consisted of 5,115 young adults recruited from Birmingham, Alabama; Chicago, Illinois; Minneapolis, Minnesota; and Oakland, California and was balanced as to age (18-24, 25-30), gender, race (Black and White), and educational status (high school graduate or less, more than a high school education). In addition, eligibility criteria included freedom from chronic disease or disability that would interfere with any part of the examination. The retention rate at year 7 was 81% and 72% at year 20. Details of study design and participants have been reported elsewhere (Friedman, et al., 1988; Hughes, et al., 1987). In the present analysis, we excluded subjects missing data for key variables or pregnant at time of interview (n=74). We also excluded subjects who had unusually high or low average daily caloric intake (<800 or >8000 kcal for men, <600 or >6000 kcal for women; n=128). Baseline sample size was 4,913 after exclusions. All analyses were in accordance with the ethical standards of the University of North Carolina at Chapel Hill; the study was approved by the Institutional Review Board at the University of North Carolina at Chapel Hill.

The CARDIA diet history (available for 1985-86, 1992-93, and 2005-06) is an interviewer-administered instrument that consists of a questionnaire regarding usual dietary practices and a quantitative diet history questionnaire that assessed consumption of foods over the past month. One hundred header questions, such as "do you eat meat", "how much

do you eat", and "how was it prepared" elicited foods eaten in an open-ended fashion. Questions were asked about brand names, preparation methods, and frequency of consumption. The open-ended aspect elicited information about special ethnic foods and unusual dietary preferences. Because of the diversity in socioeconomic status and literacy among the CARDIA population, the dietary history was designed to place the responsibility for documentation on the nutritionist, who was trained to probe for specific information in a standard way.

Nutrient and energy intakes were computed using the nutrient table developed by the Nutrition Coordinating Center (NCC) at the University of Minnesota, based on the 1609 distinct NCC food codes referenced at either year 0 (NCC tape 10) or 7 (NCC tape 20) and several thousand codes at year 20 (NDS-R in 2005). Food groups developed by the NCC were used. Additional details about the quality control and validation of the CARDIA Diet History are available elsewhere (Liu, et al., 1994; McDonald, et al., 1991; Slattery, et al., 1992).

Creation of the 2005 Diet Quality Index

The original Diet Quality Index (DQI) was designed in 1994 to evaluate the overall quality of diet based on the 1989 recommendations by the National Academy of Sciences Food and Nutrition Board (R. E. Patterson, et al., 1994). It has since been revised in adaptation to different populations and to reflect changes in nutrition knowledge (Haines, et al., 1999; Kim, et al., 2003; Laraia, et al., 2004; Popkin, Haines, & Siega-riz, 1999; Popkin, et al., 1996). Our dietary assessment tool is based on one of these revisions, the DQI-R, which quantifies adherence to the 1995 Dietary Guidelines for Americans (Haines, et al., 1999). This new index, the 2005 DQI, reflects the key messages conveyed by the 2005

Dietary Guidelines for Americans (USDA, 2005). Since there is no 'gold standard' for measuring adherence to the DGA, our index, as well as others that are also based on the 2005 DGA (Guenther, Reedy, & Krebs-Smith, 2008), represents its authors' interpretation of the dietary recommendations.

Table 5 shows each of the ten 2005 DQI components and how they were scored. Scores were based on the percentage of dietary recommendations met, specific cut points for nutrient intake, or distribution of values in our sample. Consumption of vitamin supplements did not contribute to estimates of nutrient intakes. Three of the ten DQI components include intake of key nutrients addressed by the DGA; i.e., fat (between 20 and 35% of total energy), saturated fat (≤10% total energy), and cholesterol (≤300 mg). Four components quantify adequate intake of dairy (reduced-fat), fruits, vegetables, and grains. The specific serving recommendations for different levels of energy intake were obtained from appendix A-2 of the 2005 DGA (2005), which lists sample eating patterns from the USDA Food Guide. As a general rule, foods were excluded only when they were specifically mentioned in the DGA (e.g., whole milk consumption was not counted for the dairy intake recommendation because the DGA specifies reduced-fat milk). Points were neither added nor subtracted for servings in excess of recommended intakes. The last three components relate to broader health messages, including an emphasis in 2005 on consumption of a variety of foods within and among the basic food groups in order to achieve the recommended nutrient intakes as well as reduced intake of "empty" calories and sodium. The diversity component reflects consumption of foods from 17 broad food group categories. Eight of the groups include different types of fruits and vegetables (e.g., dark-green vegetables, deep-yellow vegetables, tomatoes, potatoes), three represent reduced-fat dairy products, and six represent meat and

meat alternatives (e.g., eggs, fish, legumes, nuts/seeds, lean meats). The moderation component reflects "discretionary" behavior on the part of consumers and is based on limiting the consumption of alcohol and reducing sodium intake. The 2005 DGA gives different recommendations for sodium intake for Blacks and Whites (≤1500mg for Blacks, ≤2300mg for Whites). For Blacks, we assigned 5 points for sodium intakes ≤1500mg, 2.5 points for 1500-3200mg, and 0 points for >3200mg; for Whites, the respective cut-points were ≤2300mg, 2300-4000mg, and >4000mg. Similarly, the 2005 DGA gives different recommendations for alcohol consumption for men and women (2 servings/day for men, 1 serving/day for women). We assigned 5 points for alcohol servings ≤1 (women) or ≤2 (men), 2.5 points for 1-1.5 (women) or 2-3 (men), and 0 points for >1.5 (women) or >3 (men). The added sugars component was based on the 2005 DGA's recommendation to limit added sugars and caloric sweeteners. We ranked participants into quintiles based on their intake of added sugars from foods (sugars not naturally occurring in foods, e.g., honey, table sugar, candy) and sugar-sweetened beverages. Our scoring was based on the assumption was that people in the lowest quintiles of intake were limiting their intake. Points were summed across the 10 components for a maximum score of 100, with low values reflecting a poor diet and high values reflecting a healthy diet. We categorized the continuous score into three categories: low (DQI<50), mid (DQI 50-70), and high (DQI >70) based on the distributions of total score.

Analysis variables

Body weight and height were measured at each examination by trained staff. Body weight was measured on a calibrated balance scale with participants dressed in light clothing and without shoes, and was recorded to the nearest 0.2 kg. Height (without shoes) was

assessed using a vertical ruler and recorded to the nearest 0.5 cm. Body mass index (BMI) was calculated by dividing weight in kg by height in meters squared. Participants were categorized as normal (BMI <25 kg/m^2), overweight ($25 \leq$ BMI <30 kg/m^2), or obese (BMI ≥ 30 kg/m^2) (NHLBI, 1998). We calculated weight gain of 10 kg or more by subtracting baseline weight from weight at each subsequent examination. Weight gain was chosen as the outcome instead of obesity because all people are at risk of weight gain, regardless of current weight, while the likelihood of becoming obese depends importantly on the starting BMI and excludes those who are already obese. Further, a 10 kg weight gain has been linked to adverse changes in serum cholesterol, triglycerides, fasting insulin, and blood pressure (Norman, Bild, Lewis, Liu, & West, 2003).

Physical activity was assessed using the CARDIA Physical Activity History questionnaire, a self-report of frequency of participation in leisure, occupational, and household physical activities over the past 12 months (Jacobs Jr, Hahn, Haskell, Pirie, & Sidney, 1989). Physical activity level was expressed in units of total activity based on frequency and intensity of each activity and is available at each examination. Standard questionnaires were used at each visit to assess socio-demographic variables. Participants were classified as smokers or non-smokers at each examination.

Statistical analysis

We used survival analysis methodology (Cleves, Gould, & Gutierrez, 2008) in Stata software version 10 (College Station, TX) to examine the associations between diet quality and risk of major weight gain from 1985 to 2005. To avoid the issue of reverse causality (weight change affecting diet, rather than diet affecting weight change), our models were set up so that diet at year 0 predicted weight gain from baseline to years 2, 5, and 7, and diet at

year 7 predicted weight gain from baseline to years 10, 15, and 20. Hazard ratios (HR) were estimated using Cox proportional hazards regression models (Cox, 1972). Models were adjusted for baseline BMI, age, gender, race, education, clinic of recruitment, and time-varying physical activity score, energy intake, and smoking status. Effect modification by race and initial BMI classification was assessed in separate models through the inclusion of interaction terms and significance was determined using likelihood ratio tests with $\alpha = 0.05$. We used race-specific models if both race and BMI interaction sets were significant, then tested for BMI*DQI interactions within each race-specific model. Interactions with time were included for variables that did not meet the proportional hazards assumption. We compared models with and without energy intake, as well as models coding DQI as a continuous or categorical variable (DQI <50 was used as the reference for the categorical exposure).

The longitudinal association between participants' continuous weight change from baseline and diet quality was examined using generalized estimating equation (GEE) models adjusted for baseline BMI, age, gender, race, education, clinic of recruitment, and time-varying physical activity score, energy intake, and smoking status. Effect modification by race and initial BMI was assessed as described above. To allow flexibility in the shape of the distribution of weight at each follow-up year, indicator variables for exam year and interaction terms for DQI*exam year were added to the statistical models. Multivariate analyses were repeated using different cut-points for excluding 'implausible' energy intakes, with the most stringent cut-points excluding men with <1,000 or >4,000 kcal and women with <800 or >3,500 kcal.

D.   RESULTS

Overall DQI scores increased over time, although not all DQI components increased (**Table 5**). The largest increases were driven by reduced intake of fat, saturated fat, and cholesterol. In both 1985 and 1992, a greater proportion of Whites met the 2005 DGA recommendations for all DQI components except for fruit intake, for which more Blacks reported eating at least 90% of the recommended servings. Relatively few participants met the recommended intakes of dairy, whole grains, or were in the lowest quintiles for both added sugars from foods and beverages.

At baseline, most participants were classified as having a low DQI score (**Table 6**). Blacks with high DQI scores had higher BMI ($p<0.01$) and a higher proportion were obese compared to Blacks with low scores ($p<0.01$). In contrast, Whites with higher DQI scores had lower BMI ($p<0.01$). There was a trend for increased physical activity and education with higher DQI ($p<0.01$) in both Blacks and Whites.

For the most part, baseline mean total food and nutrient intakes differed significantly between Blacks and Whites. For example, compared to Blacks, Whites reported lower intakes of total calories, fat, cholesterol, and sweetened beverages, but higher intakes of dairy, whole grains, and vegetables. Among participants with high diet quality, Blacks reported consuming a higher percentage of energy from carbohydrates, higher intake of sweetened beverages, fruits, and 100% fruit juice, and lower intakes of reduced fat dairy, sodium, and saturated fat compared to Whites.

Diet Quality Index Score and Weight Gain

Most participants gained weight over the 20-year study period, regardless of diet quality, with an average weight gain of 17.9 kg (SD= 14.5) in Blacks and 12.5 kg (SD=

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11.7) in Whites. In multivariable-adjusted GEE models of continuous weight change (**Figure 2** and **Table 8**) we found significant effect modification by race (likelihood ratio test p <0.01) but not by baseline BMI. On average, Blacks with high diet quality (DQI>70) gained significantly more weight than Blacks with low diet quality (DQI<50) over 20 years (19.4 kg vs. 17.8 kg). In contrast, Whites with high diet quality gained significantly less weight than Whites with low diet quality (11.2 vs. 13.9 kg).

Results of multivariable Cox regressions for risk of major weight gain (≥10 kg) are presented in **Table 7**. Overall, having a high (vs. low) diet quality was associated with 25% lower risk of major weight gain (HR: 0.75; 95% CI: 0.65, 0.87). However, we found significant (p<0.05) effect modification by race and baseline BMI and therefore present effect estimates for each subgroup. After adjustment for potential confounders, a10-point increase in DQI score was associated with a 10% risk reduction in normal-weight (BMI <25 kg/m^2) Whites. In contrast, Blacks who were obese at baseline had 15% higher risk of gaining 10 kg for each 10-point increase in DQI score. Further adjustment for energy intake caused almost no change to estimates. Similar results were obtained from models using DQI score as a categorical variable.

In Blacks, even though the sample was reduced by 549 participants, results were robust to more stringent exclusion criteria for implausible energy intakes (<1,000 or >4,000 kcal for men and <800 or >3,500 kcal for women, compared to our original exclusion of men with <800 or >8,000 kcal and women with <600 or >6,000 kcal). In Whites, the only appreciable difference seen with more stringent exclusion cut-points (sample reduced by 239 participants) was that the effect modification by initial BMI became attenuated (results not shown). In models with continuous DQI as the exposure, effect modification by initial BMI

was no longer significant (likelihood ratio test p= 0.13; overall HR for 10-point increase in DQI score among Whites: 0.92, 95% CI: 0.88, 0.97). In models with categorical DQI, the hazard ratios for obese Whites with high (vs. low) DQI scores reached statistical significance (HR: 0.48, 95% CI: 0.23, 0.98) but there was still an overall significant effect modification by initial BMI (likelihood ratio test p=0.05).

E.   DISCUSSION

Despite higher risk for obesity and weight gain among Blacks, few longitudinal studies of diet and weight change have examined racial differences. The intent of this study was to examine the association between having a diet consistent with the 2005 Dietary Guidelines for Americans (as operationalized by the 2005 DQI) and subsequent weight gain in Black and White young adults. Our findings suggest that a diet consistent with the Guidelines was associated with more weight gain in Blacks (particularly if obese), but with less weight gain in Whites, after adjusting for participants' physical activity, caloric intake, smoking, and other socio-demographic characteristics. However, even Whites with high DQI scores experienced an average weight gain of over 10 kg over a 20-year period.

Both Blacks and Whites with higher DQI scores ate more whole grains, fruits, low-fat dairy, and non-fried vegetables, and had lower intakes of total fat, saturated fat, cholesterol, and sugar-sweetened beverages. However, due to ambiguity in the 2005 DGA, a high DQI score does not represent a single dietary pattern. The reason is that many different food options can be used interchangeably to meet a dietary recommendation. For example, people can meet the DGA recommendations for fruits by eating either fresh raw or processed fruits, yet the differences in nutritional quality and glycemic effects can be large (J. C. Brand-

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Miller, et al., 2003; Mateljan, 2006; Oettle, Emmett, & Heaton, 1987; Price, 1979). Thus, it is possible that the observed differential associations for diet quality by race could be partly explained by differences in the nutritional quality of foods consumed. A few differences between the diets of Blacks and Whites in the United States have been found across studies, including a higher intake of refined grains and sweet beverages (Buzby, Lin, Wells, Lucier, & Perez, 2008; Deshmukh-Taskar, et al., 2007; B. H. Patterson, Harlan, Block, & Kahle, 1995; Ritchie, et al., 2007; Swanson, et al., 1993). Our own results show that in relation to Whites with high diet quality, Blacks with high diet quality report a higher percentage of energy from carbohydrates, higher intake of sugars, fruits, and 100% fruit juice. Such differences are indicative of a higher glycemic load among Blacks compared to Whites (Janette C. Brand-Miller, Holt, Pawlak, & McMillan, 2002; J. C. Brand-Miller, et al., 2003; David S. Ludwig, 2002; D. S. Ludwig, et al., 1999). However, the diets of Blacks with high DQI scores are still closer to the DGA than the diets of Blacks with low DQI scores. Moreover, our analyses are race-specific and thus Blacks with high diet quality are compared to Blacks with low diet quality, not to Whites with high diet quality.

Another possibility is that metabolic and/or physiologic differences between Blacks and Whites underlie differential responses to diet. Racial differences in several metabolic aspects of weight regulation have been observed, including fuel oxidation, resting energy expenditure, and the metabolic response to weight loss (Berk, Kovera, Boozer, Pi-Sunyer, & Albu, 2006; Chitwood, Brown, Lundy, & Dupper, 1996; Cortright, et al., 2006; Gannon, DiPietro, & Poehlman, 2000; Kaplan, Zemel, & Stallings, 1996; Nicklas, Berman, Davis, Dobrovolny, & Dennis, 1999; Sharp, et al., 2002; Weinsier, Hunter, Schutz, Zuckerman, & Darnell, 2002; Weyer, Snitker, Bogardus, & Ravussin, 1999). This might help explain why

in weight-loss trials Black participants tend to lose less weight and regain weight faster than their White counterparts (Barbara V. Howard, et al., 2006; Kumanyika, Obarzanek, Stevens, Hebert, & Whelton, 1991; Wing & Anglin, 1996). Further, Blacks of all ages tend to have higher plasma insulin levels and lower insulin sensitivity compared to Whites, independently of adiposity (Arslanian, Suprasongsin, & Janosky, 1997; Bacha, Saad, Gungor, & Arslanian, 2005; Gower, Fernández, Beasley, Shriver, & Goran, 2003; Ku, Gower, Hunter, & Goran, 2000; Osei & Schuster, 1994; Ryan, Nicklas, & Berman, 2002). Since insulin resistance and insulin secretion play a role in body weight regulation (Mosca, Marshall, Grunwald, Cornier, & Baxter, 2004; Odeleye, de Courten, Pettitt, & Ravussin, 1997; Sigal, et al., 1997; Torbay, et al., 2002; Wedick, Mayer-Davis, Wingard, Addy, & Barrett-Connor, 2001) and diet composition can affect these parameters (Janette C. Brand-Miller, et al., 2002; Gropper, Groff, Smith, & Combs, 2005), a person's insulin response may modify the association between diet and body weight (David S. Ludwig, 2002). Results from trials comparing weight change among individuals of varying levels of insulin secretion (Cornier, et al., 2005; Ebbeling, Leidig, Feldman, Lovesky, & Ludwig, 2007; Pittas, et al., 2005; Torbay, et al., 2002) suggest that the glycemic load of meals is more relevant for people who have higher insulin secretion. The 2005 DGA emphasize a high-carbohydrate dietary pattern, but were not designed to have a low glycemic load. Hence, whether due to genetic or environmental factors, the higher insulin secretion documented in African Americans (Gower, et al., 2003) may make them more susceptible to the glycemic effects of a high-carbohydrate dietary pattern.

Adjusting for energy intake did not attenuate the association between DQI scores and weight gain. Thus, the possibility that the observed associations are the result of miss-

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reporting of dietary intake may not be ruled out. A common concern with self-reported dietary data is that heavier participants may under-report food intake to a greater extent (Johansson, et al., 2001). Moreover, differential reporting of diet by race is likely. Despite concerted effort to include foods relevant to dietary preferences of both Blacks and Whites, the CARDIA dietary history questionnaire did not yield estimates of caloric intakes at baseline that were as reasonable for Blacks compared with other populations (McDonald, et al., 1991). Also, results from a validation study suggest that there was more random variation in the diet histories of Blacks compared to Whites (Liu, et al., 1994). However, sensitivity analysis limiting the range of allowable energy intakes did not attenuate effect estimates in Blacks, and the effect modification by initial BMI was attenuated only in Whites (although effect estimates remained largely unchanged).

Using a comprehensive assessment of the whole diet is less subject to measurement error compared to energy intake alone (Schatzkin, et al., 2003). That is because even when people under- or over-report the total amount they consume, the ratios of the foods they do report is likely still be reflective of actual consumption. Several components of the 2005 DQI are designed to account for misreporting by scoring based on each subject's *reported* intake, rather than their predicted intake based on weight, gender, age, etc. For example, a person with a predicted energy requirement of 2000 kcal who actually eats (or reports eating) 6,000 kcal will not get a high DQI score simply by meeting the food and nutrient recommendations for a 2,000 kcal intake level. To get a high score, subjects have to meet the dietary recommendations for the amount and type of food they reported eating.

Limitations of this study relate mainly to its observational nature. Although we adjusted analyses for several characteristics of participants, the possibility of residual

confounding precludes definitive conclusions about causality. Also, our statistical models

relied on the assumption that dietary patterns were applicable several years (up to 13) after

they were reported. Although we cannot know for sure how those assumptions affected our

results, some research suggests that the dietary patterns of adults are relatively stable over

time (Dunn, Liu, Greenland, Hilner, & Jacobs, 2000; Millen, et al., 2005). Moreover, due to

collinearity among foods and nutrients, we are not able to accurately determine which

specific components of the DQI score are driving the observed associations.

The 2005 DQI was designed to assess how well participant's usual diets agreed with

the dietary recommendations provided by the 2005 DGA (2005) and great care was taken not

to allow our preconceptions about optimal nutrition influence decisions regarding the scoring

of the index. Hence, our results do not suggest that a healthy diet is ineffective in the

prevention of weight gain, but rather that a different definition of a healthy diet may be

needed to achieve better results (McCullough, et al., 2002). It is important to note that

creating the DQI entailed making some subjective decisions based on our interpretation of

the 2005 DGA (e.g. which components to include). Thus, the possibility exists that a

different interpretation of the DGA would yield different results. Nevertheless, the 2005

DQI is similar to the 2005 Healthy Eating Index (HEI-2005), an index that has also been

revised to meet the changing dietary guidelines (Guenther, et al., 2008). For example, both

our DQI and the HEI-2005 were designed to uncouple diet quality from diet quantity and

they both include components for fruits, vegetables, grains, dairy, fats, sugars, alcohol,

sodium, and account for dietary variety. The main difference between the two indices is that

the HEI-2005 includes a component for 'meat and beans' but not for cholesterol, while the

reverse is true for our DQI.

Our findings do not support the hypothesis that a diet consistent with the 2005 DGA benefits long-term weight maintenance among American young adults. Greater need for attention to obesity prevention in future DGA is warranted. More research is needed to determine whether the observed differential associations by race are due to differences in diet, dietary reporting, or physiological mechanisms.

Table 5. Scoring of the 2005 Diet Quality Index (DQI) components and distribution by CARDIA study year [1]

Recommendation	Scoring Criteria	Points	Percentage meeting respective scoring criteria					
			Baseline (1985)			Year 7 (1992)		
			Black n=2786	White n=2427	Total n=4913	Black n=1725	White n=2014	Total n=3739
Keep total fat intake	>40% or <15%	0	40	31	36	30	18	24
between 20 and 35% of	36-40% or 15-19%	5	34	36	35	34	33	33
total energy	≤35% and ≥20%	10	26	33	29	36	49	43
Reduce saturated fat	>13%	0	68	64	66	42	35	39
intake to less than 10%	11–13%	5	26	28	27	40	39	39
of total energy	≤10%	10	7	8	7	18	26	22
Reduce cholesterol	>400mg	0	57	42	50	41	21	30
intake to less than 300	300–400mg	5	16	20	18	19	18	19
mg daily	≤300mg	10	27	38	33	40	61	51
Choose foods and	Based on population	0-5	73	51	62	72	55	63
beverages that limit	distribution	6-8	22	32	27	20	31	26
intake of added sugars[2]		9-10	5	17	11	8	14	11
2-3 servings of reduced-	% of rec. servings							
fat milk or milk	≤50	0	88	64	76	86	61	72
alternatives[3]	60–80	5	7	18	13	9	23	17
	≥90	10	5	18	11	5	16	11
2-5 servings of fruits[3]	% of rec. servings							
	≤50	0-5	37	38	38	41	38	39
	60–80	6-8	26	30	28	26	32	30
	≥90	9-10	37	32	34	33	30	31
2-8 servings of	% of rec. servings							
vegetables[3]	≤50	0-5	53	33	43	43	23	32
	60–80	6-8	34	41	38	38	46	42
	≥90	9-10	13	26	19	19	31	26
1.5-5 servings of whole grains[3]; at least half the grains should come from whole grains[4]	% of rec. servings % whole grains							
	≤50 ≤25	0-5	66	50	58	58	42	50
	60–80 30–40	6-8	25	32	29	29	35	32
	≥90 ≥45	9-10	9	18	13	13	23	18
Consume a variety of	Dietary Diversity score	0-10	5.6	6.5	6.0	5.9	7.1	6.5
nutrient-dense foods[5]	(means (SD))		(1.8)	(1.8)	(1.9)	(1.9)	(1.8)	(1.9)
Limit sodium and	Dietary Moderation	0-10	5.6	6.1	5.9	5.5	6.0	5.7
alcohol consumption[6]	(means (SD))		(2.1)	(2.5)	(2.3)	(2.2)	(2.3)	(2.3)

[1] The 2005 DQI is based on the 2005 Dietary Guidelines for Americans (DGA).
[2] Calculated quintiles of intake of added sugars from foods and quintiles of intake of calorically-sweetened beverages. We assigned 5 points to the 1st quintile, 3 and 2 points to the 2nd and 3rd quintiles, respectively, and 0 points to the 4th and 5th quintiles.
[3] The specific serving recommendations for different levels of energy intake were obtained from appendix A-2 of the 2005 DGA (2005).

[4] Five points assigned for meeting 100% of the recommended whole grain servings (lower scores were prorated) and 5 points for consuming ≥50% of all grain servings as whole grains (whole grain servings divided by all grain servings, multiplied by 10, prorated).

[5] For each of four food groups, participants got up to 4 points if they were consumers (≥1.75 servings/week) of each of the respective subgroups. Vegetable subgroups (4pts): dark-green, deep-yellow, orange, starchy vegetables, legumes, and other vegetables. Fruit subgroups (2pts): citrus fruits and non-citrus fruits. Meat subgroups (2pts): Lean beef/pork/poultry, eggs, fish, nuts/seeds. Dairy subgroups (2pts): reduced-fat milk, cheese, and yogurt.

[6] Higher scores represent lower intake of sodium (for Blacks, we assigned 5 points for sodium intakes ≤1500mg, 2.5 points for 1500-3200mg, and 0 points for >3200mg; for Whites, the respective cut-points were ≤2300mg, 2300-4000mg, and >4000mg) and moderation in consumption of alcohol. We assigned 5 points for alcohol servings ≤1 (women) or ≤2 (men), 2.5 points for 1-1.5 (women) or 2-3 (men), and 0 points for >1.5 (women) or >3 (men).

Table 6. Baseline characteristics of CARDIA participants presented by race and Diet Quality Index (DQI) score, 1985-86[1]

Characteristic	Blacks				Whites			
	DQI <50	DQI 50-70	DQI >70	Total	DQI <50	DQI 50-70	DQI >70	Total
Number (%)	1859 (75)	553 (22)	74 (3)	2486[2]	1130 (47)	938 (39)	359 (15)	2427[2]
Age, years	24.1 (3.7)	24.9 (3.8)	25.3 (3.7)	24.3 (3.8)[2]	25.2 (3.4)	25.5 (3.2)	25.7 (3.2)	25.4 (3.4)[2]
DQI score	35.9 (8.2)	57.6 (5.3)	76.1 (4.7)	41.9 (13.2)[2]	38.9 (7.6)	58.8 (5.7)	77.4 (5.6)	52.3 (15.4)[2]
Education, years	12.9 (1.8)	13.4 (1.9)	14.1 (1.8)	13.1 (1.8)[2]	14.2 (2.4)	15.0 (2.3)	15.3 (2.2)	14.6 (2.4)[2]
Physical activity score	383 (303)	358 (256)	450 (319)	380 (301)[2]	428 (277)	448 (281)	522 (298)	450 (283)[2]
BMI, kg/m^2	25.1 (5.7)	26.5 (5.8)	25.9 (6.1)	25.3 (5.8)[2]	23.8 (4.2)	23.7 (4.0)	23.1 (3.7)	23.7 (4.1)[2]
Obese, %	15.6	20.2	20.3	16.8[2]	7.1	7.0	5.0	6.8
Male, %	47.9	29.8	21.6	43.2[2]	59.7	40.7	24.8	47.2[2]
Daily intake[3]								
Total energy, kcal	3205 (1426)	2368 (1289)	1788 (1010)	2977 (1444)[2]	3076 (1214)	2383 (1053)	1981 (846)	2646 (1182)[2]
Total fat, % of Energy	39.8 (5.1)	34.2 (5.4)	28.9 (4.7)	38.2 (5.9)[2]	40.1 (4.9)	36.1 (4.8)	30.0 (5.2)	37.0 (6.0)[2]
Saturated fat, % of energy	15.0 (2.6)	12.2 (2.4)	9.6 (2.1)	14.0 (2.9)[2]	15.6 (2.6)	13.5 (2.4)	10.6 (2.5)[4]	14.0 (3.0)[2]
Carbohydrates, % of energy	44.4 (6.5)	51.4 (7.7)	57.6 (6.8)	46.3 (7.6)[2]	42.2 (6.1)	47.0 (5.8)	53.5 (6.9)[4]	45.7 (7.3)[2]
Cholesterol, mg[5]	189 (70)	144 (56)	130 (45)	177 (69)[2]	172 (57)	147 (52)	119 (50)	155 (57)[2]
Sodium, g[5]	1.5 (0.3)	1.3 (0.3)	1.3 (0.3)	1.4 (0.3)[2]	1.6 (0.3)	1.5 (0.3)	1.5 (0.3)[4]	1.5 (0.3)
Fiber, g[5]	1.6 (0.6)	2.3 (1.0)	3.9 (1.5)	1.8 (0.9)[2]	1.8 (0.6)	2.5 (1.0)	3.7 (1.5)[4]	2.4 (1.6)
Dairy, total srv[5]	1.0 (0.6)	1.0 (0.9)	1.0 (0.7)	1.0 (0.7)	1.3 (0.9)	1.4 (0.7)	1.4 (0.8)[4]	1.4 (0.8)
Dairy, reduced-fat	0.2 (0.3)	0.4 (0.5)	0.6 (0.5)	0.2 (0.4)[2]	0.4 (0.5)	0.7 (0.6)	0.9 (0.7)[4]	0.6 (0.6)[2]
Vegetables, total srv[5]	1.1 (0.6)	1.5 (1.0)	2.5 (1.7)	1.2 (0.8)[2]	1.3 (0.6)	1.8 (1.2)	2.8 (2.0)	1.7 (1.3)[2]
Dark-green	0.1 (0.1)	0.2 (0.2)	0.4 (0.5)	0.1 (0.2)[2]	0.1 (0.2)	0.2 (0.4)	0.5 (0.9)	0.2 (0.5)[2]
Deep-yellow	0.0 (0.1)	0.1 (0.2)	0.4 (1.0)	0.1 (0.2)[2]	0.1 (0.1)	0.2 (0.3)	0.4 (0.6)	0.1 (0.3)[2]
Tomatoes	0.1 (0.1)	0.2 (0.3)	0.3 (0.3)	0.1 (0.2)[2]	0.2 (0.1)	0.2 (0.2)	0.3 (0.3)	0.2 (0.2)[2]
Starchy veg.	0.2 (0.2)	0.2 (0.2)	0.2 (0.2)	0.2 (0.2)	0.2 (0.2)	0.2 (0.2)	0.2 (0.2)	0.2 (0.2)
Other veg.	0.3 (0.3)	0.6 (0.5)	0.9 (0.8)	0.4 (0.4)[2]	0.5 (0.4)	0.7 (0.7)	1.2 (0.9)	0.7 (0.7)[2]
Fried veg.	0.3 (0.2)	0.2 (0.2)	0.2 (0.3)	0.3 (0.2)[2]	0.2 (0.2)	0.2 (0.2)	0.1 (0.2)	0.2 (0.2)[2]
Whole grains, srv[5]	0.4 (0.4)	0.6 (0.5)	1.1 (0.7)	0.5 (0.5)[2]	0.5 (0.4)	0.8 (0.6)	1.2 (0.7)	0.7 (0.6)[2]
Alcohol, srv[5]	0.2 (0.4)	0.2 (0.4)	0.1 (0.3)	0.2 (0.4)[2]	0.4 (0.4)	0.3 (0.4)	0.3 (0.4)[4]	0.3 (0.4)[2]
Fruit, total srv[5]	1.0 (0.7)	2.0 (1.2)	3.5 (1.5)	1.3 (1.1)[2]	0.7 (0.6)	1.5 (0.9)	2.1 (1.1)[4]	1.2 (1.0)[2]
100% fruit juice	0.6 (0.6)	1.2 (1.0)	1.8 (1.4)	0.8 (0.8)[2]	0.4 (0.4)	0.7 (0.7)	0.9 (0.9)[4]	0.6 (0.6)[2]
Sweetened drinks, srv[5]	0.7 (0.6)	0.8 (0.9)	0.4 (0.6)	0.7 (0.7)[2]	0.5 (0.6)	0.4 (0.6)	0.2 (0.3)[4]	0.4 (0.6)[2]

[1] The DQI is based on the 2005 Dietary Guidelines for Americans. Scores range from 0-100, higher scores indicate a diet more consistent with the guidelines. DQI categories: Low= DQI<50, Mid= DQI 50-70, High= DQI >70. Values are means (SD) or percent.
[2] Indicates statistically significant difference (Chi^2 or ANOVA, α=0.05) across DQI categories within each race group.
[3] Serving (srv) sizes based on the 2005 Dietary Guidelines for Americans. Mean daily intake is significantly different (Wilcoxon rank-sum test p<0.03) between Blacks and Whites for all nutrients/foods shown except saturated fat and starchy vegetables.

Table 7. Associations between DQI scores and risk of 10kg weight gain from 1985 to 2005[1]

	DQI score as continuous variable (per increment of 10 points)			**DQI score as categorical variable** (reference: DQI<50)		
	Blacks	**Whites**			**Blacks**	**Whites**
Model 1[2]	0.98 (0.95, 1.01)		DQI 50-70		0.98 (0.89, 1.08)	
			DQI >70		0.75 (0.65, 0.87)	
Model 2[3] Normal-weight	1.00 (0.95, 1.06)	0.90 (0.86, 0.95)	DQI 50-70	0.97 (0.81, 1.16)	0.77 (0.65, 0.91)	
			DQI >70	0.83 (0.59, 1.17)	0.61 (0.49, 0.75)	
Overweight	1.02 (0.96, 1.10)	1.04 (0.96, 1.12)	DQI 50-70	1.09 (0.87, 1.38)	1.11 (0.85, 1.44)	
			DQI >70	0.78 (0.48, 1.28)	1.10 (0.77, 1.57)	
Obese	1.15 (1.05, 1.23)	0.92 (0.81, 1.04)	DQI 50-70	1.36 (1.03, 1.79)	1.12 (0.75, 1.68)	
			DQI >70	1.68 (1.01, 2.82)	0.54 (0.27, 1.09)	

[1] Cox proportional hazards regression models adjusted for baseline age, education, BMI, clinic of recruitment, race, gender, and time-varying physical activity, energy intake, and smoking status (baseline n=4913). Estimates are hazard ratios (95% CI).
[2] Model without interaction terms.
[3] Race-specific models include DQI interactions with baseline BMI.

Figure 2. Adjusted 20-year mean weight change in CARDIA participants with low (<50) or high (>70) DQI scores. Weight change estimates based on a generalized estimating equation model that includes DQI score interactions with year and race, and is adjusted for baseline BMI, age, gender, education, clinic of recruitment, and time-varying physical activity score, energy intake, and smoking status. Baseline n= 4913, year 20 n= 2735 (average of 5 observations per person). Mean weight change is significantly different (p<0.05) at years 15 and 20 for all subgroups shown.
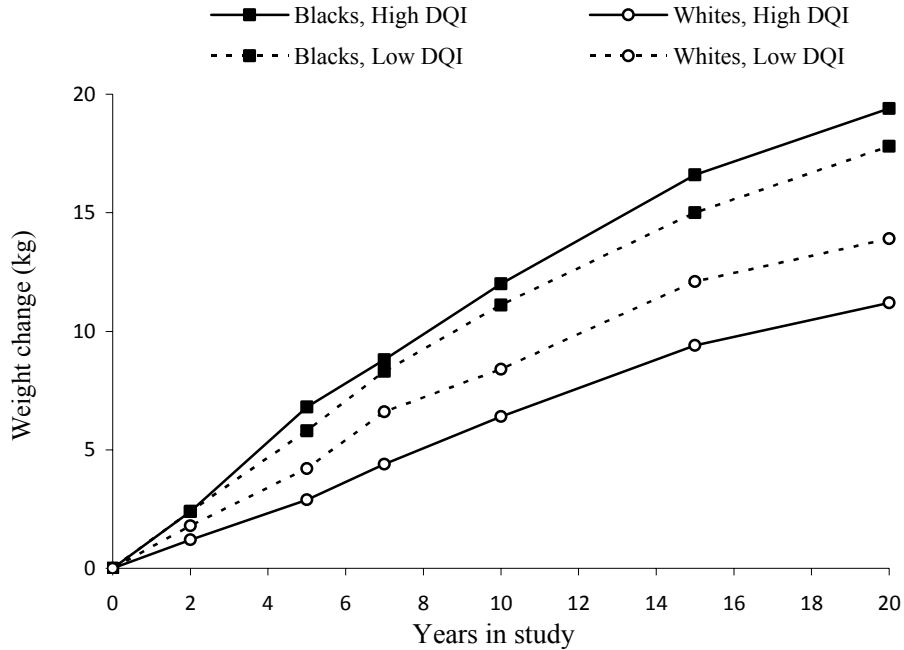
Table 8. Adjusted mean weight change (kg) from baseline in CARDIA participants with low (<50) or high (>70) DQI scores[1]

Years in study	Blacks		Whites	
	DQI <50	DQI >70	DQI <50	DQI >70
5	5.82 (5.41, 6.23)	6.82 (6.06, 7.57)	4.18 (3.73, 4.63)	2.93 (2.20, 3.65)
7	8.34 (7.90, 8.77)	8.76 (8.04, 9.47)	6.64 (6.15, 7.13)	4.44 (3.75, 5.13)
10	11.07 (10.63, 11.52)	12.02 (11.28, 12.76)	8.40 (7.90, 8.90)	6.40 (5.70, 7.10)
15	14.97 (14.51, 15.42)	16.56 (15.80, 17.32)	12.07 (11.56, 12.58)	9.36 (8.65, 10.06)
20	17.79 (17.32, 18.27)	19.35 (18.55, 20.15)	13.87 (13.34, 14.39)	11.18 (10.46, 11.89)

[1] Means (95% confidence intervals). Weight change estimates based on a generalized estimating equation model that includes DQI score interactions with year and race, and is adjusted for baseline BMI, age, gender, education, clinic of recruitment, and time-varying physical activity score, energy intake, and smoking status. Baseline n=4913, year 20 n=2735 (average of 5 observations per person).

VI. DIET QUALITY, DIABETES, AND CARDIO-METABOLIC RISK FACTORS

A.   ABSTRACT

Background: Little is known about the long-term health consequences of following the 2005 Dietary Guidelines for Americans (DGA).

Objective: To prospectively examine the associations between level of accordance with the 2005 DGA and subsequent incidence of diabetes and progression of cardio-metabolic risk factors.

Design: We used data from the Coronary Artery Risk Development in Young Adults (CARDIA) study, a cohort of Black and White Americans, aged 18-30 y at baseline, who attended up to seven examinations from 1985-86 to 2005-06 (n = 4,381). We created a 100-point Diet Quality Index (DQI) to rate participants' diets based on meeting the 2005 DGA key recommendations (higher scores = more consistent with DGA).

Results: Participants with higher DQI scores during the first seven years of follow-up subsequently experienced more favorable changes in HDL cholesterol (4th vs. 1st quartile mean difference: 4.9 mg/dL) and blood pressure (-3.4 mm Hg systolic, -3.8 mm Hg diastolic), but not in waist circumference, plasma triglycerides, nor insulin resistance (among Blacks, DQI scores were positively associated with changes in insulin resistance). We also found effect modification by race using Cox regression models for 20-year diabetes incidence (328 cases; adjusted for lifestyle and socio-demographic confounders). There was no association between DQI score in the whole sample, although Blacks in the 3rd (vs. 1st)

DQI quartile were 55% more likely to develop diabetes.

Conclusion: Participants whose diets reflected accordance with the 2005 DGA experienced more favorable changes in some intermediate risk factors, but were not at lower risk of developing diabetes.

B.   INTRODUCTION

It has been recognized that diet plays an important role in disease prevention (2003). Nevertheless, dietary advice regarding chronic disease prevention is complex in part due to the heterogeneity of the population. The Dietary Guidelines for Americans (DGA) are issued every five years by the U.S. Departments of Agriculture and Health and Human Services (2005). Although the overall goal of the DGA is "to promote health and to reduce risk for chronic diseases" (2005), they are limited by the data available at the time they were created.

The knowledge of diet-disease associations used to develop the 2005 DGA was largely based on observational cross-sectional and longitudinal studies of middle-aged Caucasian populations (King, 2007). Most studies that have attempted to evaluate the association between diets consistent with the DGA and incidence of chronic disease have not examined minorities. Hence, one potential limitation of this literature is that the results may not be generalizable to people of other races, socio-economic status, or who are younger. This is important because the burden of disease disproportionately affects minorities and the poor. Moreover, many metabolic and physiologic differences exist between Blacks and Whites (Berk, et al., 2006; Chitwood, et al., 1996; Cortright, et al., 2006; Gannon, et al., 2000; Kaplan, et al., 1996; Nicklas, et al., 1999; Sharp, et al., 2002; Weinsier, et al., 2002; Weyer, et al., 1999), making it plausible that the associations between diet patterns and

health outcomes differ depending on a person's race. Because the age of onset of chronic diseases, such as type 2 diabetes, has decreased over the last few decades (Duncan, 2006), it is also essential to examine these associations in younger populations.

While some studies have raised questions regarding the applicability of DGA recommendations to cardio-metabolic concerns (Folsom, et al., 2007; Fung, et al., 2005; B. V. Howard, et al., 2006; McCullough, et al., 2002), others have found that diets consistent with them may be associated with lower risk of cardiovascular disease (CVD) (Fung, et al., 2008; Imamura, et al., 2009; McCullough, et al., 2000). Still, there remains a lack of information on the relation between a diet consistent with the 2005 DGA and prevention of cardio-metabolic risk factors for CVD among Blacks and among young adults.

We examined the prospective association between a diet consistent with the 2005 DGA, as assessed by the 2005 Diet Quality Index (DQI), and cardio-metabolic risk in a cohort of Black and White American young adults. Specifically, we compared the associations between DQI score and 1) 20-year incidence of diabetes, and 2) 13-year changes in high-density lipoprotein (HDL) cholesterol, plasma glucose, homeostasis model insulin resistance (HOMA-IR), systolic and diastolic blood pressure, waist circumference, and triglycerides.

C. SUBJECTS AND METHODS

Data source and sample

We used data from the Coronary Artery Risk Development in Young Adults (CARDIA) study, a prospective epidemiologic study of the determinants and evolution of CVD risk factors among young adults. The baseline examination was conducted in 1985-86

and follow-up exams were conducted 2, 5, 7, 10, 15, and 20 years later. The initial cohort consisted of 5,115 young adults recruited from Birmingham, Alabama; Chicago, Illinois; Minneapolis, Minnesota; and Oakland, California and was balanced as to age (18-24, 25-30), gender, race (Black and White), and educational status (high school graduate or less, more than a high school education). Exclusions from the baseline CARDIA exam were done for the following reasons: blindness, deafness, muteness, inability to communicate, and permanent inability to walk. The retention rate among survivors at year 7 was 81% and 72% at year 20. Details of study design and participants have been reported elsewhere (Friedman, et al., 1988; Hughes, et al., 1987). In the present analysis, we excluded subjects who had unusually high or low average daily energy intake at baseline (<800 or >8000 kcal for men, <600 or >6000 kcal for women; n=129). We also excluded subjects for whom outcome measures were not available for at least one follow-up examination (n=526), who were diabetic at baseline (n=35), or had missing data for key variables (n=44). Baseline sample size was 4,381 after exclusions. All clinic procedures were conducted by trained and certified staff according to standardized procedures.

The CARDIA Diet History (collected at exam years 0, 7, and 20) is an interviewer-administered instrument that consists of a questionnaire regarding usual dietary practices and a quantitative diet history questionnaire that assessed consumption of foods over the past month. One hundred header questions, such as "do you eat meat", "how much do you eat", and "how was it prepared" elicited foods eaten in an open-ended fashion. Questions were asked about brand names, preparation methods, and frequency of consumption. The open-ended aspect elicited information about special ethnic foods and unusual dietary preferences. Because of the diversity in socioeconomic status and literacy among the CARDIA

population, the dietary history was designed to place the responsibility for documentation on the nutritionist, who was trained to probe for specific information in a standard way. Nutrient and energy intakes were computed using the nutrient table developed by the Nutrition Coordinating Center (NCC) at the University of Minnesota, based on the 1609 distinct NCC food codes referenced at either year 0 (NCC tape 10) or 7 (NCC tape 20). Food groups developed by the NCC were used. Additional details about the quality control and validation of the CARDIA Diet History are reported elsewhere (Liu, Slattery, Jacobs, Cutter, McDonald, Van Horn, Hilner, Caan, Bragg, & Dyer, 1994; A. McDonald, et al., 1991; Slattery, et al., 1992).

Analysis variables

Level of accordance with the 2005 DGA was assessed using the 2005 DQI. Table 2 shows each of the ten 2005 DQI components and how they were scored based on either the percentage of recommended servings met, specific cut points for nutrient intake, or distribution of values in our sample.

Body weight and height were measured at each examination by trained staff. Body weight was measured on a calibrated balance scale with participants dressed in light clothing and without shoes, and was recorded to the nearest 0.2 kg. For this study, body weight was set to missing if a woman was pregnant at time of interview. Height (without shoes) was assessed using a vertical ruler and recorded to the nearest 0.5 cm. Body mass index (BMI) was calculated by dividing weight in kg by height in meters squared. Participants were categorized as normal (BMI <25 kg/m^2), overweight (25≤ BMI <30 kg/m^2), or obese (BMI ≥30 kg/m^2) (NHLBI, 1998). Waist circumference was measured as the average of two waist circumference measures at the minimum abdominal girth (nearest 0.5 cm) from participants

standing upright.

Cardio-metabolic outcomes were measured at exam years 0, 7, 10, 15, and 20. All participants were asked to fast at least 12 hours and to avoid smoking and heavy physical activity at least 2 hours before the examination. Blood pressure was measured on the right arm in the sitting position after a 5 minute sitting rest. First and fifth-phase Korotkoff sounds were recorded three times at 1-min intervals, using a random zero sphygmomanometer (WA Baum Company). The mean of the second and third measurements was used in the analyses. Vacuum tubes containing no preservative were used to draw blood for insulin and glucose. Serum was separated by centrifugation at 4 °C within 60 minutes, stored in cryovials and frozen at −70 °C within 90 minutes until laboratory analysis. Fasting glucose was measured by the hexokinase-ultraviolet method. Fasting insulin was measured by radioimmunoassay. Lipids were measured by the University of Washington Northwest Lipid Research Clinic Laboratory. Total triglycerides and total HDL cholesterol were estimated using enzymatic methods. HDL cholesterol was measured after dextran sulfate–magnesium precipitation. Diabetes was defined as fasting plasma glucose ≥126 mg/dL, non-fasting glucose >=200 mg/dL, postprandial 2-hour glucose >=200 mg/dL from an oral glucose tolerance test, or current drug treatment for elevated glucose. HOMA-IR was calculated as (fasting glucose/fasting insulin)/22.5.

Physical activity was assessed using the CARDIA Physical Activity History, a self-report of frequency of participation in leisure, occupational, and household physical activities over the past 12 months (Jacobs Jr, et al., 1989). Physical activity level was expressed in units of total activity based on frequency and intensity of each activity and was collected at each examination. Standard questionnaires were used at each exam year to assess socio-

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demographic variables (income information is not available for exam year 0, data from year 5 was used instead). Participants were classified as smokers or non-smokers at each examination.

Statistical analysis

All analyses were conducted with Stata version 10 statistical software (College Station, TX). We examined tracking of participants' DQI scores over time by calculating Spearman rank-order correlation coefficients for DQI scores at baseline, year 7, and year 20 (Willett, 1998). To avoid the issue of reverse causality (e.g., diabetes diagnosis affecting diet, rather than diet predicting diabetes development), we only used diet data collected before participants developed the outcome of interest. We categorized DQI scores based on quartiles of the cumulative average of DQI scores at years 0 and 7. We used survival analysis methodology (Cleves, et al., 2008) (st commands in Stata) to examine the associations between diet quality and risk of incident diabetes. Our statistical models were set up so that diet at year 0 predicted risk from baseline to year 7, and the average of year 0 and year 7 diet predicted risk from year 7 to years 10, 15, and 20. Hazard ratios (HR) were estimated using Cox proportional hazards regression models (Cox, 1972). Potential confounders included gender, race, physical activity, smoking, total energy intake, years of education, income, study recruitment clinic, family history of diabetes, and age. Effect modification by race was assessed through the inclusion of interaction terms and included in final models if the likelihood ratio test was significant at $\alpha = 0.10$.

Cardio-metabolic risk factors. We examined the prospective association between DQI scores and 13-year change (from year 7 to year 20) in continuous HDL cholesterol, plasma glucose, HOMA-IR, systolic and diastolic blood pressure, waist circumference, and

triglycerides. Regression models were adjusted for change in smoking from year 7 to year 20 and for the following trait values at the year 7 examination: age, gender, race, years of education, income, clinic of recruitment, physical activity score, energy intake, and family history of diabetes or high blood pressure. The average of year 0 and year 7 diet data was used to calculate DQI scores and energy intake values (only year 0 diet was used if year 7 was missing). Each model was also adjusted for the corresponding year 7 value of the outcome variable, and for use of medications that could affect changes in the outcome variable.

D.   RESULTS

The mean ± SD DQI score increased from baseline (47.8 ± 15.3) to year 7 (54.8 ± 16.3) and year 20 (57.2 ± 15.6). Spearman correlation coefficients for DQI scores were as follows: year 0 and year 7 = 0.52, year 7 and year 20 = 0.47, year 0 and year 20 = 0.40. Baseline DQI score was associated with many of the participant characteristics (**Table 9**). Participants with higher DQI scores were generally white, older, female, more educated, non-smokers, had higher income, and reported lower energy intakes. Nutrient and food group intake related as expected with DQI scores in both Black and White participants (i.e., higher DQI score was associated with intake of more whole grains, vegetables, fruits, reduced-fat dairy, and less fat, saturated fat, cholesterol, and added sugars).

Incidence of diabetes (1985-86 to 2005-06)

A total of 328 incident cases of diabetes were identified during the 20 years of follow-up. The crude incidence rates shown in **Table 10** suggest a non-linear association with diabetes (in Blacks only), with highest incidence seen for participants in the second and

third DQI quartiles. We found race to be an effect modifier of the association between DQI score and risk of diabetes in adjusted Cox models. Blacks in the third quartile had 55% higher risk of developing diabetes compared to those in the lowest quartile after adjusting for several lifestyle and socio-demographic characteristics. This association was attenuated (and no longer significant) after further adjusting for baseline BMI and HOMA-IR. In models that only adjusted for age and gender, White participants with higher DQI scores were significantly less likely to develop diabetes, but these associations became attenuated and non-significant with further adjustment by other confounding variables.

Change in cardio-metabolic risk factors (1992-93 to 2005-06)

Participants with high DQI scores had significantly less increase in blood pressure (systolic and diastolic) and fasting plasma glucose, and greater increase in HDL cholesterol compared to those with lower DQI scores (**Table 11**).   Compared to participants in the lowest DQI quartile, those in the middle quartiles experienced greater increases in waist circumference and plasma triglycerides; there was no difference between those in the lowest and the highest DQI category. We found significant effect modification by race only for HOMA-IR (likelihood ratio test p = 0.03): Blacks with higher DQI scores experienced a greater increase in HOMA-IR, but there was no significant association in Whites.

E.   DISCUSSION

In this longitudinal examination of Black and White American young adults, we found that the relationship between level of accordance with the 2005 DGA (as operationalized by the 2005 DQI) and cardio-metabolic risk factors differed depending on the specific outcome being examined. For example, participants whose self-reported dietary

intake reflected accordance with the 2005 DGA during the first seven years of follow-up

subsequently experienced more favorable changes in HDL cholesterol and less increase in

blood pressure over a 13-year period (compared to subjects in the lowest DQI quartile), but

waist circumference and triglycerides increased in an inconsistent way, that is, most for

subjects in the middle DQI quartiles. In addition, the associations of DQI scores to diabetes

and HOMA-IR were modified by the participants' race. In Whites, higher DQI score was

weakly associated with lower risk of developing diabetes. In Blacks, participants in the third

DQI quartile had significantly higher risk of developing diabetes compared to participants in

the lowest quartile. This association persisted after adjusting for several potential

confounders, including income, education, level of physical activity, and smoking habits, but

was attenuated after further adjustment for baseline BMI and HOMA-IR.

It is important to note that no one in our sample received a DQI score of 100, which

represents adherence to all the key dietary recommendations set forth by the 2005 DGA

(recognizing that the measured dietary intake occurred before 2005). Hence, our results say

nothing specifically about the effects of strictly following the 2005 DGA. The 2005 DGA

executive summary states that although the DGA are integrated messages that should be

implemented as a whole, "even following some of the recommendations can have health

benefits" (2005). Our results for diabetes and HOMA-IR in Blacks do not support this

statement, although findings for blood pressure and HDL-cholesterol do support it.

Moreover, the DQI does not identify specific dietary patterns; instead, it is analogous to a

numbered check list that is tallied up at the end. For example, participants can get a score of

70 by meeting 7 out of the 10 DQI components, regardless of which they are. Moreover,

each DGA recommendation can be met through many different combinations of dietary

intake. For example, a person may meet the recommended intake of vegetables by eating spinach, carrots, lentils, and squash, or by eating mainly processed or nutrient-poor vegetables (e.g., potatoes, iceberg lettuce, tomato sauce, vegetable juice), yet the differences in nutritional value are likely to be large (Price, 1979). Similarly, the degree to which a food is processed may change its physiologic effects (Oettle, et al., 1987), but the DGA (and thus the 2005 DQI) do not take this into account.

Few studies have prospectively investigated dietary patterns congruent with the DGA and risk of developing diabetes independently of lifestyle modifications. Results from these studies are inconsistent, with some showing inverse associations between dietary patterns consistent with the DGA and others showing no association (T. T. Fung, Schulze, Manson, Willett, & Hu, 2004; Nettleton, Steffen, Ni, Liu, & Jacobs, 2008; Tinker, et al., 2008; van Dam, Rimm, Willett, Stampfer, & Hu, 2002). One study of 862 White, Black, and Hispanic American adults found no association between adherence to the Dietary Approaches to Stop Hypertension (DASH) diet (which is a sample dietary pattern recommended by the 2005 DGA) and 5-year diabetes incidence in their overall sample, but found race/ethnicity to be a significant effect modifier (Liese, Nichols, Sun, D'Agostino, & Haffner, 2009). Stratified analyses revealed a strong significant inverse association among White subjects only (n = 347), but no association among Blacks/Hispanics (n = 517).

One potential explanation for the racially-divergent results for diabetes and insulin resistance is that they are driven by physiologic/metabolic differences between Blacks and Whites. For example, studies have found that regardless of age or adiposity, Blacks have higher insulin secretion than Whites (Arslanian, et al., 1997; Bacha, et al., 2005; Gower, et al., 2003; Ku, et al., 2000; Osei & Schuster, 1994; Ryan, et al., 2002). The higher insulin

secretion in Blacks may make them more susceptible to the glycemic effects of a high-carbohydrate diet (David S. Ludwig, 2002). However, it is not clear to what extent racial differences in insulin metabolism are driven by genetic or environmental factors (or an interaction of both). Several studies have reported a higher intake of refined grains and sugar-sweetened beverages among Blacks (Buzby, et al., 2008; Deshmukh-Taskar, et al., 2007; B. H. Patterson, et al., 1995; Ritchie, et al., 2007; Swanson, et al., 1993), suggesting that dietary factors may contribute to racial differences in insulin metabolism. Therefore, another potential explanation for our findings is that race may be a proxy for a different dietary pattern. Our own results show that in relation to Whites with high diet quality (DQI scores >60), Blacks with high diet quality report a higher percentage of energy from carbohydrates, higher intake of sugars, fruits, and 100% fruit juice and lower levels of reduced fat dairy, vegetables, and whole grains. Such differences are indicative of a higher glycemic load among Blacks compared to Whites (J. C. Brand-Miller, et al., 2003; David S. Ludwig, 2002) and are otherwise suggestive of lower nutritional quality among some blacks even when meeting many of the 2005 DGA guidelines.

Compared to the amount of evidence supporting that a diet congruent with the 2005 DGA helps prevents diabetes and CVD, there seems to be more evidence supporting alternative definitions of a healthy diet (Chiuve & Willett, 2007; de Lorgeril, et al., 1999; Teresa T. Fung, et al., 2005; McCullough & Willett, 2006; Sofi, Cesari, Abbate, Gensini, & Casini, 2008). For example, McCullough et al. examined the association between the Healthy Eating Index (HEI), an index based on the DGA (Kennedy, Ohls, Carlson, & Fleming, 1995), and risk of major chronic disease in 51,826 men (McCullough, et al., 2000) and 67,272 women (McCullough, Feskanich, Stampfer, et al., 2000), followed for 8-12 years.

They found that the HEI was not significantly associated with risk of overall major chronic disease (in men and women) and CVD in women, but was associated with moderately lower risk of CVD in men. However, when researchers repeated the analysis using an alternate version of the HEI (AHEI), they found a significant 11-20% reduction in overall major chronic disease (highest vs. lowest quintiles) and 28-39% reduction in CVD risk. This increase in predictive ability of the index was due to inclusion of items that are not part of the DGA (e.g., nuts and soy, multivitamin use, ratio of white to red meat). Moreover, studies of empirically derived dietary patterns suggest that the avoidance of 'westernized' foods (e.g., refined grains, processed meats, regular and artificially-sweetened sodas) may be more important for risk reduction than the consumption of foods commonly perceived as healthy (T. T. Fung, et al., 2004; Lutsey, Steffen, & Stevens, 2008; Qi, Cornelis, Zhang, van Dam, & Hu, 2009; Matthias B. Schulze, et al., 2005).

The limitations of this study relate mainly to its observational nature and to reliance on self-reported data for diet and other lifestyle factors. For example, the CARDIA Diet History instrument may not have measured intake as well for Blacks as for Whites, at least at the baseline examination (McDonald, et al., 1991). Also, results from a validation study suggest that there was more random variation in the diet histories of Blacks compared to Whites (Liu, et al., 1994). Creating the DQI entailed making some subjective decisions based on our interpretation of the 2005 DGA. One example is the number of points assigned for meeting (or partially meeting) each recommendation. Thus, the possibility exists that using a different scoring system to reflect the 2005 DGA would yield different results. However, the intake of several nutrients and foods increased or decreased in the expected direction across DQI categories, indicating that the index captured recommendations of the

dietary guidelines. By using diet measured at baseline and year 7 only, we made the assumption that dietary patterns were representative of usual intake several years after they were reported. The correlation coefficients for DQI scores over time suggest that dietary patterns were relatively stable from baseline to year 7, and from year 7 to year 20 (Dunn, et al., 2000).

The strengths of our study include: long-term prospective study design with high rates of follow-up over 20 years (72% retention); standardized, repeated, and in-person assessments of dietary practices, anthropometrics, and disease outcomes; and the demographics of the cohort—young black and white men and women from four U.S. metropolitan areas. We used repeated measurements of diet to obtain a better assessment of long-term overall diet. The prospective nature of this analysis reduces the probability of dietary recall bias.

Our findings suggest that a higher level of concordance with the 2005 DGA may help prevent adverse changes in blood pressure and HDL cholesterol, but it may not prevent the development of diabetes or increases in triglycerides, waist circumference, and insulin resistance in young adults. The adverse association for insulin resistance observed among Blacks with higher DQI scores merits further study.

Table 9. Baseline characteristics of CARDIA participants according to Diet Quality Index score, 1985-86[1]

Characteristic	Blacks				Whites			
	<40	40-50	50-60	>60	<40	40-50	50-60	>60
Number (%)	1007 (47)	591 (27)	328 (15)	229 (11)	490 (22)	517 (23)	504 (23)	715 (32)
DQI score	31.4 (5.9)	44.8 (2.8)	54.7 (2.8)	67.9 (6.7)	32.9 (5.4)	45.1 (2.8)	54.8 (3.0)	70.9 (7.9)
Age, years	24.0 (3.7)	24.6 (3.7)	24.4 (3.8)	25.3 (3.7)	24.9 (3.5)	25.5 (3.4)	25.4 (3.3)	25.8 (3.2)
Education, years	12.8 (1.7)	13.2 (1.8)	13.4 (1.9)	13.9 (1.9)	13.8 (2.4)	14.5 (2.4)	14.8 (2.3)	15.3 (2.1)
Physical activity score	378 (297)	379 (306)	357 (294)	390 (285)	416 (274)	446 (280)	458 (287)	473 (281)
BMI, kg/m^2	24.8 (5.6)	25.5 (5.8)	26.2 (5.7)	26.4 (5.9)	24.1 (4.4)	23.7 (4.0)	23.8 (4.2)	23.3 (3.7)
Male, %	50.4	41.8	30.5	25.3	65.7	56.1	45.4	28.9
Smokers, %	36.4	32.8	23.5	16.6	41.0	29.2	25.2	13.1
Average daily intake[3]								
Total energy, kcal	3319 (1367)	2915 (1463)	2400 (1298)	2091 (1151)	3228 (1153)	2948 (1234)	2508 (1118)	2081 (833)
Total fat, % of energy	41.2 (4.5)	37.1 (5.3)	35.3 (5.4)	31.5 (5.0)[4]	41.0 (4.5)	39.0 (5.2)	37.1 (4.6)	32.7 (5.4)
Saturated fat, % of energy	15.7 (2.4)	13.8 (2.4)	12.9 (2.3)	10.7 (2.1)[4]	16.1 (2.5)	15.0 (2.5)	14.1 (2.2)	11.8 (2.7)
Carbohydrates, % of energy	42.8 (5.8)	47.1 (6.8)	49.8 (7.4)	55.0 (7.0)[4]	41.3 (5.8)	43.3 (6.5)	45.7 (5.6)	50.8 (6.6)
Cholesterol, mg/1000 kcal	201 (74)	168 (57)	154 (58)	131 (50)	185 (61)	162 (55)	152 (46)	131 (55)
Dairy, reduced-fat[5]	0.1 (0.2)	0.2 (0.3)	0.4 (0.5)	0.5 (0.6)[4]	0.3 (0.4)	0.5 (0.5)	0.7 (0.6)	0.8 (0.7)
Vegetables, total[5]	1.0 (0.5)	1.2 (0.6)	1.4 (0.9)	2.0 (1.4)[4]	1.2 (0.6)	1.4 (0.7)	1.7 (1.0)	2.4 (1.7)
Sugar-sweetened beverages[5]	0.6 (0.6)	0.8 (0.8)	0.7 (0.8)	0.6 (0.7)[4]	0.6 (0.6)	0.5 (0.7)	0.4 (0.5)	0.2 (0.5)
Candies, added sweeteners[5]	0.8 (0.7)	0.8 (0.8)	0.8 (0.9)	0.6 (0.7)[4]	0.9 (1.0)	0.7 (0.8)	0.6 (0.7)	0.5 (0.6)
Fruit, total[5]	0.8 (0.6)	1.3 (0.9)	1.8 (1.0)	2.7 (1.4)[4]	0.6 (0.4)	0.9 (0.6)	1.3 (0.8)	1.9 (1.1)
100% fruit juice[5]	0.5 (0.5)	0.8 (0.7)	1.1 (0.9)	1.6 (1.3)[4]	0.3 (0.3)	0.5 (0.5)	0.6 (0.6)	0.8 (0.8)
Grains, total[5]	2.6 (0.8)	2.6 (0.8)	2.5 (0.8)	2.5 (1.0)[4]	2.5 (0.7)	2.6 (0.8)	2.7 (0.8)	2.8 (0.9)
Whole grains[5]	0.3 (0.4)	0.5 (0.5)	0.6 (0.5)	0.8 (0.7)[4]	0.4 (0.3)	0.5 (0.5)	0.7 (0.6)	1.1 (0.7)
Sodium, g/1000 kcal	1.5 (0.3)	1.4 (0.3)	1.4 (0.3)	1.3 (0.3)[4]	1.6 (0.3)	1.6 (0.3)	1.5 (0.3)	1.5 (0.3)

[1] The DQI is based on the 2005 Dietary Guidelines for Americans. Scores range from 0-100, higher scores indicate a diet more consistent with the guidelines. DQI categories: Low= DQI<50, Mid= DQI 50-70, High= DQI >70. Values are means (SD) or percent.

[2] Indicates statistically significant difference (Chi^2 or ANOVA, α=0.05) across DQI categories within each race group.

[3] Mean daily intake is significantly different between Blacks and Whites for all nutrients/foods shown except total fruit servings.

[4] Indicates significant difference in food intake between Blacks with high DQI score and Whites with high DQI score.

[5] Servings/1000 kcal.

Table 10. Results of multivariable Cox regressions for 20-year incidence of diabetes[1]

		Diet Quality Index Quartiles			
		1st	2nd	3rd	4th
Mean DQI score (SD)		32.1 (5.1)	43.8 (2.8)	54.2 (3.3)	69.3 (6.8)
Overall	Incidence rate[2]	0.0042	0.0045	0.0042	0.0030
	Model 1[3]	1.00	1.04 (0.77, 1.39)	1.05 (0.77, 1.43)	0.82 (0.58, 1.18)
	Model 2	1.00	1.15 (0.85, 1.56)	1.22 (0.88, 1.69)	1.08 (0.73, 1.59)
	Model 3	1.00	1.14 (0.84, 1.56)	1.15 (0.83, 1.59)	1.16 (0.79, 1.71)
Blacks[4]	Incidence rate[2]	0.0045	0.0058	0.0070	0.0046
	Model 1[3]	1.00	1.13 (0.81, 1.59)	1.38 (0.97, 1.97)	0.87 (0.53, 1.43)
	Model 2	1.00	1.22 (0.87, 1.73)	1.55 (1.08, 2.24)	1.06 (0.63, 1.78)
	Model 3	1.00	1.23 (0.86, 1.75)	1.40 (0.97, 2.03)	0.96 (0.57, 1.62)
Whites[4]	Incidence rate[2]	0.0035	0.0029	0.0022	0.0025
	Model 1[3]	1.00	0.71 (0.40, 1.26)	0.50 (0.28, 0.90)	0.56 (0.32, 0.95)
	Model 2	1.00	0.89 (0.50, 1.61)	0.66 (0.37, 1.20)	0.83 (0.48, 1.46)
	Model 3	1.00	0.90 (0.49, 1.65)	0.73 (0.41, 1.32)	1.14 (0.65, 2.00)

[1] Statistical analyses were set up so that diet at baseline predicted incidence from baseline to year 7, and the average of baseline and year 7 diet predicted incidence from year 7 to years 10, 15, and 20. Based on 328 incident cases of diabetes (n=4381).

[2] Incidence rates= number of cases divided by person-years.

[3] Values are hazard ratios (95% confidence intervals). Model 1: adjusted for age, gender, and race. Model 2: further adjusted model 1 for education, income, smoking, physical activity, energy intake, family history of diabetes, and clinic. Model 3: further adjusted model 2 for baseline BMI and HOMA insulin resistance.

[4] Included interaction terms for race*DQI score. Model 1 likelihood ratio test p= 0.03, model 2 likelihood ratio test p= 0.08, model 3 likelihood ratio test p= 0.09.

Table 11. Adjusted mean change in cardio-metabolic risk factors from 1992-93 to 2005-06 according to DQI score categories[1].

	Diet Quality Index Quartiles				P for trend
	1st	2nd	3rd	4th	
Mean DQI score (SD)	34.0 (5.0)	45.4 (2.8)	55.7 (3.1)	69.8 (6.2)	
Diastolic blood pressure (mm HG)[2]	5.15 (4.51, 5.78)	4.42 (3.79, 5.05)	2.51 (1.89, 3.13)	1.29 (0.67, 1.91)	0.00
Systolic blood pressure (mm HG)[2]	7.91 (6.74, 8.33)	7.54 (6.74, 8.33)	6.50 (5.71, 7.28)	4.42 (3.63, 5.21)	0.00
Plasma HDL cholesterol (mg/dL)[3]	-0.62 (-1.32, 0.08)	0.29 (-0.40, 0.98)	0.89 (0.21, 1.57)	3.86 (3.19, 4.54)	0.00
Plasma triglycerides (mg/dL)[3]	15.4 (11.1, 19.8)	26.1 (21.8, 30.4)	25.4 (21.1, 29.6)	20.3 (16.1, 24.5)	0.00
Fasting plasma glucose (mg/dL)[4]	14.6 (13.1, 16.1)	14.5 (13.0, 15.9)	14.7 (13.2, 16.1)	11.5 (10.0, 12.9)	0.00
Waist circumference (cm)[5]	6.63 (6.08, 7.18)	7.78 (7.23, 8.32)	7.80 (7.27, 8.34)	7.53 (6.99, 8.06)	0.00
HOMA-Insulin Resistance[6] Blacks	0.61 (0.38, 0.83)	0.78 (0.53, 1.04)	1.18 (0.88, 1.49)	1.14 (0.70, 1.58)	0.00
Whites	0.56 (0.23, 0.89)	0.43 (0.15, 0.71)	0.48 (0.25, 0.72)	0.47 (0.27, 0.68)	0.13

[1] Values are means (95% confidence intervals). Estimates are from regression models adjusted for change in smoking from 1992-93 (year 7) to 2005-06 (year 20) and for the following trait values at the year 7 examination: age, gender, race, years of education, income, clinic of recruitment, physical activity score, and energy intake. The average of year 0 and year 7 diet data was used to calculated DQI scores and energy intake values.

[2] On the basis of 3,700 participants. Models further adjusted for blood pressure at year 7, family history of high blood pressure, and use of medications to control blood pressure.

[3] On the basis of 3,627 participants. Models further adjusted for year 7 values of triglycerides or HDL cholesterol and use of medications to control cholesterol.

[4] On the basis of 3,320 participants. Models further adjusted for glucose at year 7, family history of diabetes, and use of medications to control blood glucose.

[5] On the basis of 3,604 participants. Model further adjusted for waist circumference at year 7.

[6] On the basis of 3,116 participants. Models further adjusted for HOMA insulin resistance at year 7, family history of diabetes, and use of medications to control blood glucose. Likelihood ratio test for interaction between race and DQI categories p=0.03.

VII. SYNTHESIS

A.   SUMMARY OF AIMS AND RESULTS

The overarching goal of this research was to investigate environmental correlates of eating a healthy diet as well as its association with subsequent development of chronic disease. We defined a healthy diet as one congruent with the 2005 Dietary Guidelines for Americans (DGA). Our aims were:

3) To examine the longitudinal association between neighborhood poverty and the number of supermarkets and grocery stores during the years 1992, 1995, and 2000.

4) To examine the associations between accordance with the 2005 DGA from 1985 to 1992 and subsequent risk of diabetes, major weight gain, and progression of other cardio-metabolic risk factors from 1985 to 2005.

To address our research aims, we used data from the Coronary Artery Risk Development in Young Adults (CARDIA) study, a prospective study of the determinants and evolution of CVD risk factors among young adults. The cohort consists of 5,115 Black and White young adults recruited in 1985 and re-examined up to 7 times until 2005. We linked residential street addresses of participants for exam years 7 (1992), 10 (1995), and 15 (2000) to contemporaneous neighborhood information from federal and commercial databases. Over this time period the CARDIA respondents moved from baseline in four U.S. metropolitan areas (799 census tracts) to 48 states, 1 federal district, 1 territory, 529 Counties and 3,805 census tracts.

For the first aim, we hypothesized that high poverty, high minority neighborhoods would have lower availability of supermarkets but higher availability of grocery stores. The rationale for this was that most studies to date have found these associations.. We found that neighborhoods with higher poverty and higher ethnic minority composition had a greater number of supermarkets and grocery stores.

For the second aim, we hypothesized that higher DQI scores would be predictive of lower disease risk, and that race would modify this association. We developed an index of diet quality (2005 DQI) to rate participants' diets based on meeting the 2005 DGA key recommendations (higher scores = more consistent with DGA) and examined subsequent changes in body weight, blood pressure, lipids, and glucose, and incidence of diabetes. With the exception of blood pressure and HDL-cholesterol, we found little evidence that people who consumed a diet congruent with the DGA had better health in the long run. Specifically, we found that participants with higher DQI scores during the first seven years of follow-up subsequently experienced more favorable changes in HDL cholesterol and less increase in blood pressure over a 13-year period, but the association of DQI scores with waist circumference and triglycerides was not linear; the participants in the middle ranges of DQI scores had the largest increase in both. In Whites, higher DQI scores were associated with *less* 20-year weight gain, but unrelated to diabetes incidence or changes in HOMA insulin resistance. However, in Blacks, higher DQI scores were associated with *more* 20-year weight gain, and with higher incidence of diabetes (only 3rd vs. 1st DQI quartile) and increase in HOMA insulin resistance.

B.   SYNTHESIS OF FINDINGS

The growing recognition of the public health and economic consequences of nutrition-related chronic diseases has prompted actions by policymakers, the public health

community, and other organizations. Many of these actions have focused on improving the consumption of 'healthy' foods by populations at highest risk for obesity and other chronic diseases: people with low SES and African Americans (Harris, et al., 2006; Krieger, 1993). In many cases, however, the evidence base to demonstrate the effectiveness of these interventions in promoting long-term health is weak. It may not be surprising, then, that little progress has been made in halting the obesity epidemic even after decades of trying to understand and modify its causes at the individual level (Carleton, et al., 1995; Farquhar, et al., 1990; J. O. Hill, et al., 2005; Hyman, et al., 2007; Luepker, et al., 1996). Recently, the focus has shifted to role of the residential built environment in the development of nutrition-related chronic diseases (Papas, et al., 2007). For example, in an effort to alleviate SES-linked disparities in diet quality, initiatives to encourage the development of grocery retail investments in low-income communities have been called for (Pothukuchi, 2005; Story, et al., 2008). The problem with these initiatives is that the relation between neighborhood SES and access to healthy foods may not be sufficiently understood to develop a successful intervention at this time. However, even if people had access to foods that allowed them to meet the DGA (and they did so), it is not clear that their risk of chronic disease would be reduced. While specific components of the DGA have been studied and linked to reduced risk of chronic disease (e.g., whole grains, vegetables), there is surprisingly little evidence that adopting a diet congruent with the DGA is an effective way to reduce risk of chronic disease. Because the DGA are used in the implementation of several government programs and public health campaigns are conducted to educate Americans about what they should be eating, having proof that the DGA actually work is paramount. In the following sections we discuss our main findings and how they address important gaps in the literature.

Do people in poor neighborhoods have lower availability of food stores where 'healthy' foods are sold?

Supermarkets are often used as proxies for availability of healthy foods because compared to smaller stores (such as grocery or convenience stores) they tend to have more variety, better prices, and higher quality of foods (D. Block & Kouba, 2006; Cheadle, et al., 1991; Chung & Myers, 1999; Drewnowski & Specter, 2004; Glanz, et al., 1998; Jetter & Cassady, 2006). Overall, our findings suggest that poor and high minority areas may have availability of a higher number of supermarkets and grocery stores, which implies that, in general, the stores to satisfy demand for 'healthy' foods could be in place and available to most Americans in urban areas. This may mean that the SES-linked disparities in diet quality observed in the U.S. are not driven by lower physical availability to high quality foods, implying that factors other than availability (e.g., cost, preparation time) are more important determinants of diet. In the past, the finding of more access to grocery stores in low SES areas has been interpreted as evidence of disparities in access to healthy foods; however, it is unclear whether this is true because of the large diversity in stores that this category encompasses.

While we observed an association between neighborhood socio-demographic characteristics and availability of food stores, we do not know the causal direction of this relationship. However, our findings suggest that there may be complex social and economic processes underlying the relation between neighborhood composition and the food environment, which are not captured by cross-sectional data. Our results imply that residents' characteristics/behaviors may play a role in the association between neighborhood socio-demographics and food deserts. For example, a person's time constant characteristics related to both living in an economically-deprived neighborhood (e.g., attitudes toward health, social resources, race) as well as living in an area with a

given supermarket density may be associated with aspects of residential location that are related to food store availability.

To our knowledge, this is the first longitudinal analysis assessing the association between poverty and supermarket availability. By exploiting the structure of our data (repeated measures on individuals over time and place) and using statistical methods to take advantage of the variation within person, we were able to adjust for time-constant unmeasured characteristics. Furthermore, because by the end of follow-up participants were living in 48 states, our study provides more generalizable results for urban areas than previous studies that have been limited to small geographic ranges (e.g., inner city Detroit).

The study of the built environment is a relatively new field, thus, the limitations of this research pertain mainly to the lack of established methodology. For example, the accuracy of our food store data is dependent on obtaining accurate information from archived commercial datasets. If the error inherent in commercial databases is random as suggested by recent research (Boone, et al., 2008) (Bader, et al., 2010), our estimates are not likely to be biased. For example, if the database is missing or misclassifies 10% of active supermarkets, but such error is not related to the SES of an area (or other variables of interest), then our estimates could be attenuated but not biased by these errors. Moreover, it is possible that our buffer size (8 km) may be too large, especially for poor people who do not own a car and may do most of their shopping closer to home. However, in our preliminary analysis we found an association between food store availability within the 8 km buffer size (relative to 1, 3, and 5 km Euclidean buffers) and overall dietary intake.

Is following the 2005 DGA associated with reduced risk of weight gain, diabetes, and other CVD risk factors?

Taken together, our results suggest that a diet congruent with the 2005 DGA may have different consequences for Blacks and Whites, and that glucose metabolism may be at the heart of those differences. It may be that the higher insulin levels and lower insulin sensitivity documented in African Americans (Arslanian, et al., 1997; Bacha, et al., 2005; Gower, et al., 2003; Ku, et al., 2000; Osei & Schuster, 1994; Ryan, et al., 2002) makes them more susceptible to the glycemic effects of a high-carbohydrate diet, such as that emphasized by the DGA. For example, since insulin resistance and insulin secretion play a role in body weight regulation (Mosca, et al., 2004; Odeleye, et al., 1997; Sigal, et al., 1997; Torbay, et al., 2002; Wedick, et al., 2001) and diet composition can affect these parameters (Janette C. Brand-Miller, et al., 2002; Gropper, et al., 2005), it is plausible that a person's insulin response may modify the association between diet and body weight (David S. Ludwig, 2002). Although it is not known to which extent differences in glucose metabolism are driven by genetic or environmental factors (or interaction of both), it seems that eating a diet defined as healthy according to the 2005 DGA could have some unintended consequences for Blacks.

Other possible explanations for our results could be classified into three categories, specifically, (1) problems with the 2005 DGA, (2) problems with the dietary data, or (3) problems with the 2005 DQI.

First, our results could be explained by problems with the 2005 DGA that would either make it an unhealthy diet for Blacks but not for Whites, or that would allow some people to eat an 'unhealthy' diet while still meeting the recommendations (making race a proxy for two different dietary patterns). Due to ambiguity in the DGA, many different foods can be used interchangeably to meet the guidelines, sometimes even foods with little nutritional value. Hence, a high DQI score does not represent a single, identifiable, dietary pattern. For example, people can meet the DGA recommendations for fruits and

vegetables by eating only canned fruits and vegetables, and they would get the same DQI score as a person who eats mainly fresh produce, yet the differences in nutritional quality can be large (Price 1979; Mateljan 2006). For this reason, it is possible that the observed racially-divergent effects of a high DQI score are not caused by the same dietary pattern, but may reflect different choices in factors such as the degree of processing of foods (J. C. Brand-Miller, et al., 2003; Deshmukh-Taskar, et al., 2007; Mateljan, 2006; Oettle, et al., 1987; B. H. Patterson, et al., 1995; Price, 1979; Ritchie, et al., 2007; Swanson, et al., 1993). In addition, the 2005 DGA were not designed to have a low glycemic load and allow half of the recommended grain servings to be refined grains and for fruit juice to count for meeting the fruit recommendation.

Second, it is possible that racial differences in reporting of dietary intake could have produced the observed associations (i.e., if Blacks with 'unhealthy' diets differentially reported 'healthier' diets). Despite concerted effort to include foods relevant to dietary preferences of both Blacks and Whites, the CARDIA dietary history questionnaire did not yield estimates of caloric intakes that were as reasonable for Blacks compared to the appropriate Recommended Dietary Allowances (McDonald, et al., 1991). Moreover, according to a validation study by Liu and colleagues, the CARDIA Diet History instrument may not have measured intake as well for Blacks as for Whites, at least at the baseline examination (Liu, et al., 1994). However, this validation study showed that Blacks had more *random* measurement error in diet histories compared to Whites, but we are not aware of reporting bias by race. Our finding that obese Blacks were found to have a higher risk of major weight gain (table 7) could be attributed to the known bias in energy intake reporting by obese individuals; however, this argument is not supported by the results in obese Whites, who seem to have the same association with DQI and weight gain risk as normal-weight Whites. As for differences based on ethnic-

specific foods not included on the diet history questionnaire, the open-ended format of the questions asked by the interviewer elicited information about special ethnic foods and unusual dietary preferences, and the history form provided space to record any other foods regularly consumed or common dietary behaviors not discussed during the interview. It is also worth mentioning that an advantage of using a comprehensive assessment of the whole diet is that it is less subject to measurement error compared to energy intake alone (Schatzkin, et al., 2003). That is because even when people under- or over-report the total amount they consume, the ratios of the foods they do report may still be reflective of actual consumption. Hence, Blacks would not have received high DQI scores simply by omitting foods that were not in the questionnaire, they would have had to meet the dietary recommendations for the amount and type of foods they reported eating.

Third, it is possible that the 2005 DQI did not adequately capture the essence of the DGA. However, intake of several nutrients and foods increased or decreased in the expected direction across DQI categories, which suggests that the index captured recommendations of the dietary guidelines. Moreover, the components and scoring of the 2005 DQI are similar to the 2005 Healthy Eating Index (HEI-2005), an index that has also been revised to meet the changing dietary guidelines (35). Although interpretation of the guidelines is necessarily subjective, we strived for an unbiased representation of the DGA that was independent of our own knowledge about nutrition and disease prevention.

Our results are even more striking when we consider that healthy behaviors tend to cluster, hence, even though we control in our analyses for physical activity, smoking, etc., residual confounding by unobserved factors is likely. For example, a person with a high diet quality may also engage in some other health-conscious attitudes or behaviors that would reduce their risk of chronic disease. Because we did not adjust for these

unobserved characteristics, our estimates of the 'effect' of having a high diet quality may be biased to show a benefit, even if there was none. Moreover, the placebo effect cannot be overemphasized. Randomized controlled trials have shown that people who 'adhere' to a treatment are inherently different than those who do not (and are more likely to receive a benefit from the treatment regardless of whether the treatment is real or placebo). It is reasonable to expect that people who eat a diet they think is good for them will experience some health benefit just from the expectation, which could also bias our estimates to show a benefit. Hence, observing a weak association between high DQI scores and lower risk of chronic disease could be seen as the expected association driven by processes other than a true effect of diet.

One key strength of our analyses on diet quality and health is that, to avoid the possibility of reverse causality in our estimates, we examined the association of each outcome modeled as either incidence or change from baseline as a function of diet measured at an earlier time. Also, we used repeated measurements of diet over time to obtain a better assessment of long-term overall diet. This is important because chronic diseases develop over a long period of time, thus long-term diet is conceptually our exposure of interest. However, even though we have relatively high rates of follow-up over 20 years (72% retention), selective loss to follow-up of participants is of concern in all longitudinal studies and thus the generalizability of our results may be limited. In this context, if those remaining in the sample experienced a different relationship between diet quality and health, bias may have resulted.

Self-reported dietary data is notoriously imprecise. Although this is a concern in all observational studies, a comprehensive assessment of the whole diet (such as our DQI) is less subject to measurement error compared to energy intake alone (Schatzkin, et al., 2003). Recall bias is also a problem often faced by observational studies, where

development of disease can interfere with a participant's recall of dietary practices, but this is not an issue in a prospective study. However, from the present study, it is not possible to determine whether the effect modification by race observed for several of the outcomes reflects a physiological process or if it is a proxy for differences in diet or reporting thereof. Although unlikely, the possibility cannot be excluded of racially-linked reporting bias driving the observed associations. An important limitation of our interpretation is that, due to how the data were collected (food-frequency questionnaire) it is not possible to accurately calculate the glycemic load of the participants' diets. This would have been useful to determine if Blacks with high DQI scores had higher glycemic index than Blacks with low DQI scores.

Finally, no one in our sample got a DQI score of 100 (meaning that they met all the key dietary recommendations set forth by the 2005 DGA), hence, our findings are silent as to the possible effect of following a diet that completely embodies the DGA. However, the DGA state that "even following some of the recommendations can have health benefits" (2005) and thus meeting a few of the guidelines should provide some health benefit compared to not meeting any.

C.   IMPLICATIONS FOR PUBLIC HEALTH POLICY AND RESEARCH

The overall contribution of this research was to better understand commonly-held assumptions about the determinants and consequences of diet quality. We were able to accomplish this using high-quality data from a large prospective cohort study and analytic methods that exploited the longitudinal structure of the data. The underlying motive behind examining relations believed to be well-established was to determine whether using methods that more closely approximate causal inference would make a difference in our understanding of these relations.

The goal of public health research is often to inform policies and interventions intended to promote health in a variety of ways. To avoid spending time, money, and effort in ill-designed campaigns, these initiatives should be based on research that recognizes (and hopefully addresses) threats to causal inference. In other words, understanding *how* things are is not enough to develop successful policy, we must strive to understand *why* things are the way they are. While cross-sectional studies should be valued for their contribution to our understanding of how things are, it should be clear that they provide no help in discerning why things are the way they are. Although randomized controlled trials remain the gold standard of proof, they are unfeasible for most questions due to ethical, monetary, or time constraints. Hence, well-designed longitudinal studies should be encouraged for providing the evidence base needed to develop successful strategies to combat public health challenges.

In some regards, it seems that the standard of evidence for public health action is lower than for other matters of equal importance. The rationale may be that if there is a possible benefit to be derived (and no perceived harm), then it is worth taking action. For example, the Dietary Guidelines for Americans were first implemented without evidence that they would improve the public's health. Thirty years later, there is still little proof of their efficacy. A low standard of evidence is only a problem, however, if such guidelines have unintended long-term consequences. Our findings imply that they do for African Americans, although the reasons why need further study. Ironically, intervention efforts to educate people about the DGA are often targeted at this group. Although our findings may not apply to the 2010 DGA, it will be necessary to formally evaluate their effectiveness to understand whether they indeed lead to reduced risk of chronic disease in the population. As for food store environment, our research suggests (but does not prove)

that lower supermarket availability may be a minor determinant of SES-linked disparities in diet quality.

D.   FUTURE RESEARCH DIRECTIONS

Researching and understanding the relationship between the food environment and area socio-demographic characteristics might be facilitated with the development of a conceptual framework. The implicit assumption in most current research is that food store access influences eating behavior (perhaps by influencing perceived barriers or facilitators to food purchases). One problem with this assumption is that people's perception of their food environment is not analogous to their actual environment (L. Moore, Diez Roux, & Brines, 2008). Moreover, it discounts the possibility that residents of an area shape their own food store environment. To date, there has been little interest in determining why some low-income areas indeed are food deserts; however, understanding the mechanisms by which such scarcity arose is needed to develop successful policies that will help low income and minority populations to purchase and consume 'healthier' food choices. In this context, for the few areas of the U.S. where food deserts have been confirmed to exist, attention should be given to understanding the economic or social processes that gave rise to the food desert (for example, do poor people have lower demand for healthy foods?).

Also important is research validating the food store environment measures obtained from commercial databases. Until we have a good understanding of what is being measured, research from large-scale studies, such as ours, depends on assumptions about the type of errors inherent in these databases.

As for the effectiveness of the DGA, randomized controlled trials are necessary to understand whether race is an effect modifier. The Women's Health Initiative Dietary

Modification Trial is the only randomized trial to have evaluated (albeit indirectly) the effect of following a diet consistent with the 2005 DGA. After an average follow-up of eight years, there was little evidence that women in the intervention group were at lower risk of chronic disease. A longer follow-up on these subjects would be very informative. However, a new trial specifically designed to evaluate the 2010 DGA would also be needed, preferably double-blind design, as this would be the only way to account for a placebo effect.

Several studies have now evaluated the efficacy of different popular diets on a variety of disease biomarkers, and the evidence supporting definitions of a healthy diet unlike the 2005 DGA seems robust (Chiuve & Willett, 2007; de Lorgeril, et al., 1999; Teresa T. Fung, et al., 2005; McCullough & Willett, 2006; Sofi, et al., 2008). These studies, along with our present research, leads us to the conclusion that there may not be one ideal dietary pattern suitable for everyone, and thus diets that are not consistent with the DGA deserve further study.

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