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Use of the Glycemic Index and the DASH diet to Lower Blood Pressure in Adolescents with Hypertension and Pre-Hypertension

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ABSTRACT

Purpose: To assess the change in the glycemic index of the diets of teenagers with hypertension or pre-hypertension who received formal nutrition counseling as part of a 3-month intervention to lower blood pressure. Subjects: A sub-set of the participants’ data as reported by Couch et al. (12) was used. Participants were adolescents between the ages of 11-19 years with diagnosed pre-hypertension and Stage 1 hypertension (n=45). Study Design: Participants were randomly assigned to a behavioral nutrition intervention focused on the DASH diet (n=23) or Routine Care (RC) (n=23). Methods: Dietary glycemic index (DGI) and dietary glycemic load (DGL) were calculated for each treatment group for food items obtained from three 24-hour recalls collected at baseline and 3 months (post-intervention). Change scores for dietary glycemic index, dietary glycemic load, systolic blood pressure and diastolic blood pressure were calculated by subtracting baseline values from post-treatment values. Results: Neither the DASH nor the RC treatment significantly reduced DGI or DGL. There was no detectable association between change in DGI or DGL for all participants or by intervention group and change in systolic blood pressure or diastolic blood pressure. Conclusion: Based on these findings, counseling on a DASH type diet did not significantly modify DGI or DGL. Further, change in DGI and DGL was not associated with change in blood pressure in this study. These results suggest that the effects of the DASH intervention on blood pressure in this study were mediated by some component of the DASH diet other than DGI or DGL.
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I. Trends in overweight, obesity, hypertension and other cardiovascular risk factors in adolescents

In the United States, almost one-third of children and adolescents are overweight or obese. In youth, obesity is defined as a body mass index (BMI) for age greater than or equal to the 95th percentile and overweight is defined as a BMI for age greater than or equal to the 85th percentile (1). Recently, the National Health and Nutrition Examination Survey (NHANES) data were used to examine obesity trends by age between the years 1988-1994 and 2003-2006. These data showed that prevalence of obesity in children increased significantly over this time period as follows: 5.2% in children two to five, 5.7% in children six to nine and 7.1% in adolescents between the ages of 12-19 (1). When these age groups were further scrutinized by race and gender, the data showed that the increase in prevalence was higher among non-Hispanic blacks (7.8%) and Mexican-Americans (8.0%) than for non-Hispanic whites (5.7%) (1). For adolescent females, non-Hispanic blacks had the highest increase in prevalence of obesity (14.5%) when compared to non-Hispanic whites (7.1%) and Mexican-Americans (10.7%) (1). This prevailing trend of being obese in adolescence could be tracked into young adulthood, where the trend data projected that 80% of overweight or obese adolescents would be overweight or obese by age 25 (1).

Several lines of evidence support an association between obesity and the presence of cardiovascular disease (CVD) risk factors, namely insulin resistance, dyslipidemia, and hypertension. Li et al. (2) studied the prevalence of pre-diabetes, defined as the presence of impaired fasting glucose (IFG) or impaired glucose tolerance (IGT), and its association with a clustering of cardiometabolic risk factors using data from NHANES 2005-2006. In this study,
the cardiometabolic risk factors measured included: 1) high triglycerides (>150 mg/dl), 2) high blood pressure (systolic ≥130 mmHg or diastolic ≥ 85 mmHg), 3) central obesity (waist circumference ≥ 90\textsuperscript{th} percentile), and 4) low HDL cholesterol (<40 mg/dl). The subjects were 777 boys and girls aged 12-19 years. The results from this study showed that 83.9% of adolescents had neither IFG nor IGT, 12.6% had isolated IFG, 3% had isolated IGT and 0.5% had both IGT and IFG (2). Overweight adolescents had twice the prevalence of IFG as compared to those with a normal weight, and IFG characterized 80% of the adolescents with pre-diabetes. Pre-diabetes was associated with being male and having hyperinsulinemia and was negatively associated with being non-Hispanic black. Those adolescents with two or more cardiometabolic risk factors had a 2.7 fold higher unadjusted rate of pre-diabetes than those with no risk factors and a 2.3 fold higher prevalence than those with one risk factor. Additionally, Li and colleagues (2) found that both IFG and IGT were associated with obesity and the clustering of cardiometabolic risk factors. The findings of this study are profound because pre-diabetes is an intermediate stage in the development of Type 2 diabetes mellitus (DM), which is a CVD risk factor. Thus identification of adolescents with pre-diabetes and subsequent intervention by lifestyle modification should begin early to halt progression to CVD disease.

Lui et al. (3) studied how non high-density HDL cholesterol (non-HDL) was associated with IFG and the clustering of cardiometabolic risk factors in adolescents aged 12-19 years using NHANES data from three time periods: 1999-2000, 2001-2002, and 2003-2004. Results showed that 11% of adolescents had IFG, and that the mean level of non-HDL cholesterol was higher for those adolescents after adjustment for confounding variables. Indeed, the odds of having clustered cardiometabolic risk factors were 1.08 for those with IFG and low non-HDL
cholesterol, 3.55 for those without IFG but with high non-HDL cholesterol, and 10.10 for those with IFG and high non-HDL cholesterol. For this reason, elevated non-HDL cholesterol may be an important indicator of CVD risk in adolescents. Similar to Li et al. (2), these researchers also found that IFG was associated with the clustering of cardiometabolic risk factors.

With regards to the prevalence of hypertension in adolescents, Din-Dzietham et al. (5) used data from NHANES and examined the trends in high blood pressure and mean blood pressure for obese boys and girls ages 8-17 by racial and ethnic group from 1963-2002. This study used the current diagnostic criteria for pre-hypertension and hypertension published in the *Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents* (4), hereafter referred to as the Fourth Report on pediatric blood pressure. According to this report, classification of blood pressure in the pediatric population is defined by age in the following manner:

1) Normal blood pressure is systolic and diastolic blood pressure less than the 90th percentile for age, height and gender;

2) Pre-hypertension is systolic or diastolic blood pressure greater than or equal to the 90th percentile and less than the 95th percentile or above 120/80 mmHg;

3) Stage 1 hypertension is systolic or diastolic blood greater than or equal to the 95th percentile and less than the 99th percentile plus 5 mmHg;

4) Stage 2 hypertension is systolic or diastolic blood pressure greater than the 99th percentile plus 5 mmHg.
Results of the study showed that the greatest increase in pediatric hypertension over the last forty years was observed over the last decade. Also males had a higher mean blood pressure than females overall, which was the same trend seen in adults. In the time period from 1999-2002 the age adjusted prevalence of high blood pressure was 4.2%, 3.3%, and 4.6% for blacks, whites and Mexican-Americans, respectively. During the same time period (1988-2002), the increase in pre-hypertension was significant for blacks and Mexican-Americans. In relation to obesity, Din-Dzietham et al. (5) showed that BMI and waist circumference significantly increased the likelihood of having high blood pressure. From 1999-2002 the odds ratios for high blood pressure among obese adolescents were 2.1, 1.8 and 3.2 for blacks, whites and Mexican-Americans, respectively for each 1kg/m$^2$ increment in BMI (5). Of particular interest was the finding that diastolic blood pressure increased from 1988-1994 and 1998-2002 more rapidly than did systolic blood pressure among lean children, yet their median diastolic blood pressure remained the same. This finding suggested that obesity is only one among many risk factors for high blood pressure. The results of this study provided support for a link between obesity and hypertension and suggested a need for intervention to improve both health conditions in adolescents.

II a. Relationship between obesity and cardiovascular disease

The consequence of obesity is the development of a multiplicity of disease states which together constitute the metabolic syndrome. This syndrome includes dyslipidemia, hypertension, systemic inflammation, a prothrombotic tendency, and dysglycemia (6). According to the National Cholesterol Education Program (need to add this reference), the diagnostic criteria for metabolic syndrome in adults include elevated waist circumference, elevated triglycerides,
Reduced high density lipoprotein cholesterol, elevated blood pressure, and elevated fasting glucose. One must have at least three of these five criteria to be diagnosed with this condition. Diagnosed metabolic syndrome is a risk factor for CVD. This is evidenced by the observation that those with the syndrome have twice the risk for developing CVD than those without the syndrome (6).

Both metabolic syndrome and modified low density lipoprotein (LDL) are synergistic in the process of atherogenesis, which is characterized by two major events: 1) arterial injury and 2) response to injury (6). The injury itself is caused by infiltration of LDL into the arterial intima. The severity of infiltration depends on both the concentration of LDL and the permeability of the arterial wall to LDL. Once LDL is trapped in the extracellular matrix, it becomes susceptible to modification by aggregation, hydrolysis of cholesterol esters, oxidation and glycation. Through this modification LDL becomes pro-inflammatory.

The damage caused by each component of metabolic syndrome is as follows:

- Hypertension is associated with endothelial cell dysfunction and an increase in angiotensin II, which enhances the release of pro-inflammatory cytokines;

- Dysglycemia leads to a formation of advanced glycation products which aid in causing chronic inflammation, thereby increasing vascular permeability and decreasing vascular dilation by reducing the actions of nitric oxide. The result of this glycation is that it promotes the trapping of LDL in the extracellular matrix as well as its modification;
• Dyslipidemia often presents in the form of low amounts of high density lipoprotein (HDL) which decreases the ability to transport excess LDL and prevent its modification;

• A pro-thrombotic state causes abnormalities in thrombus formation and degradation which enhance coagulation, decrease fibrinolysis and alters platelet function mainly by increasing plasminogen activator inhibitor-1, fibrinogen, factors VII, VIII, X and Von Willebrand factor.

To summarize, both metabolic syndrome and LDL synergistically promote endothelial cell dysfunction which is the distinguishing characteristic of atherogenesis and results in the destruction of the extracellular matrix and formation of unstable plaques that can rupture and cause CVD events (6).

II b. Relationship between obesity and hypertension

The link between obesity and hypertension should be cause for concern because obesity rates are increasing among every demographic in the US. Overweight/obesity makes up 65-75% of the associated risk for primary hypertension in adults and children (6). The pathophysiology of hypertension as a consequence of obesity has been well-characterized. Explanations for the relationship between these two risk factors for CVD include: 1) release of angiotensinogen from adipocytes as a substrate for the renin-angiotensin system, and 2) augmented blood volume related to an increased body mass. Obesity also leads to impaired insulin dependent transport of glucose into tissues thereby causing serum glucose levels to rise, stimulating the pancreas to release more insulin. In this way, hyperglycemia leads to hyperinsulinemia in an attempt to force
glucose into the cells. Hyperinsulinemia contributes to hypertension by increasing sympathetic activation and/or by stimulation of vascular smooth muscle cell hypertrophy, resulting in increased vascular resistance. Another proposed mechanism by which obesity is thought to lead to hypertension includes upregulation of Type A endothelin-1 receptors in the kidneys which promote vasoconstriction, impairment of natriuresis and a decrease in the glomerular filtration rate through stimulation of vascular smooth muscle contraction (6).

III. Dietary interventions to treat hypertension and other cardiovascular risk factors

Non-pharmacological means of treatment for hypertension include mainly lifestyle changes, most notably, the Dietary Approaches to Stop Hypertension diet (DASH), which was first shown to lower blood pressure in adults and adolescents. The DASH study in adults enrolled 459 participants with untreated systolic blood pressure between 120-159 mmHg and a diastolic pressure between 80-95 mmHg (8, 9, 10). Participants were assigned to one of three diet groups for this study: 1) a control diet, which was the typical American diet with potassium, calcium and magnesium close to the 25th percentile of the usual intake of US adults, 2) a fruit and vegetable enriched diet, which was similar to the typical American diet, but had more fruits and vegetables, with less snacks and sweets; the potassium, calcium and magnesium content were close to the 75th percentile of the typical US adult intake, 3) a combination or DASH diet, which was rich in fruits and vegetables, low fat dairy foods, and low in fat and cholesterol. The combination diet was also high in fiber and protein. Potassium, calcium, and magnesium were close to the 75th percentile of the typical US adult intake. For all three diet groups, the sodium was held constant at 3g/day. Also, subjects were provided with foods for the diet, including snacks, which increased compliance to the diet. The treatment phase of this study was eight weeks. Findings
from the study showed that the DASH diet resulted in significantly greater decreases in blood pressure as compared to the control diet. The fruit and vegetable diet also lowered blood pressure more than the control diet but less than the DASH diet. A follow-up study to this was the DASH-sodium Trial. In this study, the participants were randomly assigned to a control diet which included foods that were typically found in the American diet (low fruits, vegetables and fiber, high in fat) or the DASH diet, which emphasizes high consumption of fruits, vegetables, low-fat dairy foods, whole grains, lean meats and was low in sodium, sweets and sugar. The participants followed their assigned diets for three consecutive thirty day periods. Both the DASH group and the control group were randomized into three subgroups for sodium intake (high sodium [3.5 g/day], intermediate sodium [2.3 g/day] and low sodium [1.2 g/day] (8, 9). The results of DASH sodium study showed that the DASH diet lowered hypertension in a stepwise fashion according to the sodium subgroups, with the subgroup having the lowest sodium intake experiencing the greatest reduction in blood pressure. For those on the DASH diet, reducing sodium intake from the high to low levels resulted in a decrease in systolic blood pressure of 7.1 mmHg for those without hypertension and 11.5 mmHg for those with hypertension (8, 9). Bray et al. (11) found that age factored heavily into the effects of sodium reduction on blood pressure for the DASH diet participants with reductions of 4.8 mmHg for those aged 23 to 41, 5.9 mmHg for those aged 42 to 47, 7.5 mmHg for those aged 48 to 54, and 8.1 mmHg for those aged 55 to 76. To summarize, the combined effects of the DASH diet coupled with low sodium intake produced better results than the DASH diet or low sodium diet alone. Therefore one needs to consider both the overall dietary composition in addition to sodium levels to produce the greatest results.
In adolescents, Couch and colleagues (12) developed a three month behavioral nutrition intervention program emphasizing the DASH diet versus the routine care offered for adolescents with hypertension in a hospital-based clinic setting. Adolescents aged 11-18 years with a clinical diagnosis of pre-hypertension or stage 1 hypertension were stratified by gender and race and randomly assigned to the DASH intervention (n=28) or routine care (n=29). Post-treatment assessment was scheduled for 3 months after the in-clinic counseling and follow up was scheduled for 6 months after the baseline visit. Results showed that from baseline to post-treatment, reductions in systolic blood pressure were significantly greater for the DASH group compared with routine care. The change in systolic blood pressure among the DASH group was -7.9% versus -1.5% in the routine care group. Also, by post treatment, 50% of the DASH group achieved normalization of blood pressure versus 36% in the routine care group. Fruit intake increased by approximately two servings more per day and high sodium, high fat foods decreased by approximately 0.8 servings more per day among DASH participants compared to those in routine care. From baseline to post treatment, dietary intake of potassium and magnesium increased by 42% and 36%, respectively and total fat decreased by 12% for the DASH group. This change was significantly greater than in the routine care group. The increase in potassium and magnesium was correlated with the increase in fruit and vegetable intake as expected since these minerals are found within these foods. These studies provide evidence that the DASH diet can reduce hypertension in the adult and adolescent population, but whether dietary therapy focusing on a DASH diet can be used to treat other aspects of the metabolic syndrome and lead to a reduced risk of CVD has yet to be ascertained.

IV. Use of glycemic index and glycemic load in the diet to lower CVD risk factors
Recently, the glycemic index (GI) has been the focus of specialized diets designed to lower risk factors for CVD. However, before discussing these pertinent studies, a brief description of the GI is appropriate. The GI categorizes foods containing carbohydrates by the ability of a food to increase glucose levels in terms of velocity and magnitude (13, 14). GI is measured by comparing the increase in glucose levels in the blood induced by an isolated food under standard conditions (usually 50 grams of carbohydrate) with that induced by a reference food, usually glucose or white bread. The GI can then be determined by comparing the area under the curve within two hours of ingestion. The reference food is given the value of 100, and all other foods are compared to this value, which is expressed as a percentage (13, 14). Foods are usually categorized into one of three categories of GI values: high (70 and above), intermediate (56-69), and low (55 and under). Low GI foods include foods generally high in fiber, including unsweetened fruits and vegetables, beans/lentils, grainy breads, and foods very low in carbohydrate such as fish, eggs, meat and some cheeses, while high GI foods include foods such as white potatoes, refined cereals and starches and white rice. The effect of soluble fiber on GI is that it acts on intermediate metabolism by forming a gel in the intestinal track and as a consequence slows the absorption rate of glucose and fat from the small intestine and the release of glucose into the blood stream (15). One of the most important aspects of the GI is that it takes into account several physical and chemical factors associated with different foods and their interaction such as, processing of food, cooking, type of starch, fiber content and type of carbohydrate. Because of these factors, the GI for the same food may have a high degree of variability.
A derivative of the glycemic index, the glycemic load (GL) is calculated by multiplying the GI by the amount of carbohydrate eaten in grams and dividing the product by 100 (13). Each unit of dietary GL represents the equivalent of glycemic effect of one gram of carbohydrate from white bread (15). GL can also be categorized as high, medium or low. The major advantage to the GL versus the GI is that it takes into account the actual amount of carbohydrate eaten and is therefore a better reflection of the ability that GI has to raise the blood glucose level in terms of quantity; in other words, the ability of GI is dependent upon how much of that carbohydrate containing food was eaten.

Several studies have examined the effectiveness of GI and GL on modifying risk factors for CVD. Ebbeling et al. (16) conducted a 12-month randomized control trial to examine the efficacy of a low GL diet consumed ad libitum versus a low fat diet on decreasing CVD risk in obese young adults aged 18-35 years. The researchers also incorporated physical activity and behavioral modification in both the treatment and control groups. After 12 months of the intervention, low GL diet resulted in a greater decline in plasma triglycerides and plasmin activator inhibitor-1 (PAI-1) compared to the low fat diet. There were no significant changes in LDL-cholesterol or blood pressure in either group.

Lui et al. (17) studied the effects of a high GL diet on plasma concentrations of high sensitivity C-reactive protein (hs-CRP) in middle-aged women. The objective for this observational study was to examine whether a high dietary GL was positively associated with hs-CRP concentrations and whether this association was modified by BMI. This study enrolled 244 participants from the Women’s Health Study aged 45 to 82 with a mean BMI of 26 kg/m². These participants had been part of the control group for a cardiovascular disease study within the
initial Women’s Health Study. Diet was assessed using a food frequency questionnaire and the DGL was calculated for these foods. Results of the study showed that both the quality and quantity of carbohydrate were directly related to plasma concentrations of CRP independent of risk factors for ischemic heart disease including BMI and total energy intake. As evidence of this, the adjusted geometric mean plasma CRP in the highest quintile of DGL was nearly 2 fold that in the lowest quintile. Liu et al. (17) found that there was a dose-response relationship between DGL and plasma CRP especially in obese women. The women with the highest CRP included diabetics and overweight women which is consistent with recent literature. Based on findings from this study, the authors projected that for each 1.0 mg/L increase in CRP the risk for CVD was increased by 25%. This study is important because it shows that with a 2.4 mg/L increase in plasma hs-CRP there would be an increase in ischemic heart disease risk by 60% (17). The major weakness of the study was that CRP can be affected by many factors other than DGL and it is mostly stable over long periods of time and less so over short periods of time. Implications for this study are that if one lowers the GL of the diet, plasma CRP would decrease, causing a lowering of CVD risk.

Levitan et al. (18) conducted an observational study that examined the relationships between GI and GL on blood lipids and CRP levels in non-diabetics and whether these relationships varied with BMI. The participants were 18,137 women in Women’s Health study who gave fasting blood samples (8 hrs), were not diabetic (self-report), were not taking lipid lowering medications, and had an energy intake between 600 and 3500 kcal/day. Results showed that DGL was highly correlated with carbohydrate intake while the correlation between DGI and carbohydrate was much lower, possibly because DGL accounts for carbohydrate load.
Women with a high DGI diet tended to be less physically active and to have lower intakes of folate and magnesium compared with women who had a low GI diet. Dietary GL was a stronger predictor of HDL and LDL/HDL ratio than dietary GI. However, dietary GI was more strongly associated with LDL and CRP. The -4.9 mg/dL difference in HDL between extreme quintiles of GL was associated with a 22% increase in risk of coronary heart disease, while the 0.24 unit difference in LDL/HDL was associated with an increase in CVD risk of 14% (18). One key question that was not explained by this study was whether the low GL diet was achieved by consuming less total carbohydrate or by reducing the GI of the carbohydrate that was consumed or both. Some investigators have pointed out that the best health outcomes can be obtained by improving the quality of the carbohydrate first and then focusing on the amount.

Several researchers have found that plant based diets with a low GI are effective in the management of diabetes as a major risk factor for CVD because they improve the lipid profile of those who adhere to the diet. In a study by Barnard et al. (19) diabetics who modified their dietary pattern to a vegan diet were found to reduce their diabetes medications by 43%. Change to this dietary pattern was also accompanied by a significant decrease in HbA1c levels (a long term glucose marker) by nearly one percentage point, which indicates a significant improvement in glycemic control. Similar findings were reported in another study of the vegan diet and insulin resistance, which is one of the trademark characteristics of Type 2 diabetes. Goff et al. (20) found that vegans had a higher insulin sensitivity and a lower systolic blood pressure than omnivores; a major difference between these types of dietary patterns is that the vegan diet consists of greater amounts of low GI foods compared with the omnivorous diet (20). Finally, Ford et al. (21) examined the relationship between increased fruit and vegetable consumption
and the incidence of Type 2 diabetes. They found that those who consumed five or more servings of fruits and vegetables a day had a lower incidence of diabetes than those who consumed less servings, which suggests that it may be beneficial for those who are at risk for diabetes to increase their intake of these foods, without necessarily becoming a vegetarian or a vegan.

Another component of a low GI diet is fiber, which is not only found in fruits and vegetables, but also in whole grains, beans and lentils. Fiber, specifically the soluble type is necessary for optimal glucose control because it reduces urinary glucose losses and delays the absorption of glucose into the blood stream, thereby controlling the rate of insulin release (14). A study by Chandalia et al. (22) found that high fiber diets (50 g/day; 25g soluble and 25 insoluble) reduced total cholesterol by 6.7%, triglyceride by 10.2%, and very low density lipoprotein by 12.5%. Cholesterol, triglyceride and very low density lipoprotein are all indicators of risk for cardiovascular disease, so this study shows that high fiber diets significantly decrease these risk factors. However, they did use 50 g of fiber per day, which is more than two times the average amount that Americans eat. Therefore, the applicability of these findings is uncertain. Nevertheless, based on the preceding discussion, there is adequate evidence to conclude that fiber does positively impact CVD and renal disease (22). It should be noted that examining the role of GI and GL in diabetes is relevant to the purpose of this study as diabetes is a CVD risk equivalent and is likely to co-exist with hypertension, which also contributes to CVD risk.

V. Association between GI and GL in the diet and CVD risk factors in adolescents

Gunther et al. (23) investigated whether adherence to a DASH diet was associated with blood pressure and hypertension status in youth with diabetes from the SEARCH for Diabetes in Youth
Study. These adolescents were aged 10-22 years and had Type 1 and Type 2 diabetes. Dietary assessment was done with a food frequency questionnaire and adherence to the DASH diet was assessed with a dietary index developed for this study consisting of eight DASH food groups including grains, vegetables, fruits, dairy, meat, nuts/seeds/legumes, fats/oils, and sweets. For each food component a maximum score of ten could be achieved if the DASH dietary recommendation was met. Proportional scores were derived when intakes were less than the recommendation. The investigators found that the prevalence of hypertension was markedly different between youth with Type 1 and Type 2 diabetes: 28.2% versus 6.8%, respectively. For those with Type 1 but not Type 2 diabetes, mean diastolic blood pressure significantly decreased across tertiles of DASH adherence, with the lowest blood pressure in those with the highest adherence to the diet. Systolic blood pressure was not different for either Type 1 or Type 2 diabetics by tertile of DASH adherence. These findings suggest that the DASH diet may be useful in lowering blood pressure in adolescents with diabetes.

Slyper et al. (24) studied the relation between dietary GI and GL and blood lipids, specifically HDL-cholesterol in adolescents and young adults aged 11 to 25 years with high cholesterol. The researchers obtained dietary information using 3 day food records and then calculated the GI and GL of the food items eaten. The results showed that HDL-cholesterol level was significantly correlated with GL, GI, total sugars, total carbohydrate and fructose. Of these, the GL was the only independent predictor of HDL-cholesterol levels and accounted for 21% of the variation in this lipoprotein. Therefore, findings from this study support the use of a low GL diet to raise HDL-cholesterol levels.
Grant et al. (25) studied the impact of a vegetable-rich diet (low-GI diet) on key markers of health in 207 Australian adolescents aged 14-15 years old who attended Seventh-day Adventists high schools. Subjects were classified as either vegetarian (n=53) or non-vegetarian (n=160) based on nutrient information provided in the Schools Physical Activity and Nutrition Survey (SPANS). Findings from this study showed that statistically lower scores were observed on average for vegetarians compared to non-vegetarians for cholesterol/HDL ratio, LDL-cholesterol, waist circumference, body weight and BMI. There was no observed difference in iron status or height between vegetarians and non-vegetarians. This suggests that vegetable-rich diets provide sufficient nutrients for adolescents. These findings are consistent with benefits seen in adults and suggest that a vegetarian diet (e.g, low GI) will significantly improve CVD risk factors in adolescents.

Ebbeling et al. (26) compared the effects of a reduced GL diet versus a reduced fat diet in the treatment of adolescent obesity. This randomized control trial consisted of a six month intervention and six month follow-up for sixteen obese adolescents aged 13-21 years. The reduced GL diet targeted a proportion of energy from carbohydrate and fat of 45%-50% and 30-35%, respectively with the remainder coming from protein. The reduced fat diet was based on current recommendations for weight loss and diabetes prevention. Accordingly, meal plans were designed to give a negative energy balance of 250-500 kcal/d, and consisted of 55%-60% of energy coming from carbohydrate, 25%-30% of energy from fat, with the remainder of energy coming from protein. Both groups were allowed to eat ad libitum and received an equivalent amount and type of behavioral therapy and nutrition education. Findings showed that GL decreased for the experimental group and did not change significantly for the conventional
group, whereas dietary fat decreased for the conventional group and tended to increase for the experimental group. BMI and fat mass decreased in the experimental group from 0-12 months while neither outcome changed significantly in the conventional group. At 12 months, BMI and fat mass decreased significantly more in the experimental compared with the conventional group. Insulin resistance, as assessed by HOMA, increased significantly less in the experimental compared with the conventional group during the intervention period and statistical adjustment for BMI did not alter this result. Overall, change in GL was a strong predictor of this study outcome, explaining about half of the variance in both groups combined. The importance of this study is that it showed that reducing dietary GL may have greater benefits than reducing dietary fat when treating adolescent obesity to lower the risk for Type 2 diabetes, and could thereby lower CVD risk. Notably, this study did not see a change in blood pressure when GI and GL were lowered.

Similarly, Spieth et al. (27) compared the effects of a reduced GI diet versus a reduced fat diet in management of pediatric obesity in a retrospective cohort study. A total of 64 patients received the low-GI diet while 43 received the reduced fat diet for approximately four months. The low-GI diet was given ad-libitum and the macronutrient distribution was 45%-50% carbohydrate, 20%-25% protein, and 30%-35% fat. The reduced fat diet had a macronutrient distribution of 55%-60% carbohydrate, 15%-20% protein, and 25%-30% fat and had an energy restriction of 250-500 kcal per day. Behavioral and nutrition therapy was administered to both groups and did not vary between groups. For each BMI tertile, the low-GI group had a larger decrease in BMI than the reduced fat group. Additionally, compared with the reduced fat group, 17.2% of patients showed a decrease in BMI of at least 3kg/m². The overall mean change in BMI
for the low-GI group was -1.53kg/m² compared with -0.06kg/m² for the reduced fat group. This difference remained statistically significant after adjusting for confounders. The results of this study suggest that a low-GI diet is more effective in lowering BMI compared with a reduced fat diet and supports the use of GI in reducing incidence of Type 2 diabetes mellitus, a CVD risk factor.

Taken together, the findings of these studies strongly support the use of a low-GI and low-GL diet in lowering many CVD risk factors in adults and adolescents. However, there is conflicting evidence that lowering DGI and DGL lowers blood pressure in adults or youth. The conflicting literature on GI/GL and blood pressure is the basis for undertaking the present thesis project.

VI. Purpose and Hypotheses

The purpose of this study was to assess the change in the GI of the diets of teenagers with hypertension or pre-hypertension who received formal nutrition counseling as part of a 3-month intervention to lower their blood pressure. In this study, two different interventions were compared for change in GI and GL. One intervention focused on the DASH dietary pattern and included a behavioral nutrition program that involved one dietary counseling session and 10 telephone contacts. The second intervention focused on lowering dietary fat and sodium according to established guidelines to manage pediatric blood pressure and including only one counseling session. The dietary GI and GL were assessed at baseline before the intervention and immediately post-intervention. The change in dietary GI of the adolescent diets was then correlated with change in SBP and DBP.
The major hypotheses for this study were that:

a) Adolescents counseled on a DASH-diet versus a low fat/low sodium diet (routine care, RC) would have a greater reduction in their dietary GI and dietary GL from baseline to post-intervention.

b) Adolescents who achieve a greater reduction in dietary GI and dietary GL would have a greater decrease in SBP and DBP from baseline to post-intervention as compared to those with no or less reduction in dietary GI/GL.

VII. Methods

A. Subjects:

Subjects were recruited from the Cincinnati Children’s Hypertension Center in Cincinnati, OH. Study participants were adolescents between the ages of 11 to 19 years with diagnosed pre-hypertension and stage 1 hypertension. Detailed methods of study inclusion and exclusion criteria are described in detail by Couch et al. (12). The subjects included in this study encompassed a smaller sample than reported in the published trial. This thesis was initiated before complete blood pressure readings and dietary information was available for all randomized participants. For this reason, the total participant number for this thesis is 23 and 22, respectively in the DASH and usual care groups. This compared with 29 and 28, respectively in the published clinical trial. The study protocol was approved by Cincinnati Children’s Hospital Medical Center and the University of Cincinnati Institutional Review Board. All participants gave their written assent and parents signed informed consents prior to participation.
B. Interventions:

This thesis study compares changes in GI and GL in response to 2 different dietary interventions to manage blood pressure in adolescents. These interventions will be described in brief below. Details of the intervention designs have been published elsewhere by Couch et al (12).

DASH intervention: The DASH intervention emphasized the DASH dietary pattern. Teens were expected to achieve a dietary pattern that included 8 servings of fruits and vegetables, 3 servings of low fat dairy products, and less than 2 servings of high fat/high sodium foods per day. Participants received one face-to-face, 60-minute counseling session with a dietitian in the Hypertension Center followed by weekly 15-20 minute phone calls from a dietitian emphasizing behavioral skills to enable dietary change at home and school. Behavioral skills emphasized food tracking, goal setting, action planning, social support and overcoming barriers. Participants kept weekly food trackers where they tallied their DASH food goals each week. Food goals were set each week to progressively work the teen up to the DASH dietary pattern. Incentive money was awarded for meeting weekly DASH food goals set by the telephone counselor.

Routine Care (RC) intervention: Adolescents in the routine care intervention received the usual hospital-based nutrition care provided by the Hypertension Center. This information follows the guidelines established in the Fourth Pediatric Report (4) for managing pre-hypertension and hypertension. The focus of the intervention was on lowering dietary fat by adding more fruits, vegetables, low fat grains and other foods to meet calorie goals. The intervention also focused on lowering sodium by eating low sodium food products and less processed foods. Participants
were provided with one face-to-face counseling session with the dietitian in the Hypertension Center.

C. Measures

All measures were collected at baseline and 3 months (post-intervention). All measurements were made in the Hypertension Center by trained clinic staff blinded to treatment assignment.

Dietary Assessment

Three 24-hour recalls were collected from each participant at baseline and post-treatment. All food recalls were analyzed by Nutrition Data Systems software (2005).

Using the foods lists and serving information generated from the Food Records File of the NDS output, the GI for each individual food item was calculated. The DGI was then calculated by summing the products of the digestible carbohydrate content per serving, multiplied by the number of servings of that food per day, multiplied by the GI of that food, all divided by the total amount of digestible carbohydrate daily intake (17). The equation used for these calculations is shown below:

\[
\text{Dietary GI} = \frac{\sum \text{foods} \times \text{C} \times \text{S} \times \text{GI}}{\sum \text{foods} \times \text{C} \times \text{S}}
\]

where C represents grams of digestible carbohydrate in serving of food, S is the number of servings of the food and GI is the glycemic index using glucose as the reference value (17). Additionally, the DGL was obtained using the following formula (17):
Dietary GL = \( \sum \text{foods} (C \times S \times \frac{[GI/100]}{100}) \)

The values of DGI and DGL were calculated for both the routine care (RC) and DASH intervention at baseline and post treatment. For statistical analysis, input variables were change (\( \Delta \)) in DGI and \( \Delta \) in DGL between baseline and post treatment. The output variables were \( \Delta \) in systolic blood pressure (SBP) and \( \Delta \) in diastolic blood pressure (DBP) between baseline and post treatment. Change for DGI, DGL, SBP and DBP was calculated by subtracting baseline values from post-treatment values.

D. Data analysis: For all analyses, the statistical software used was Statistica 9.0, StatSoft, Inc., Tulsa, OK. The statistical analyses were done in the following manner:

1) To test the first hypothesis that adolescents in the DASH intervention as compared to those in RC would have a greater reduction in their DGI and DGL from baseline to post-intervention, descriptive statistics were calculated for baseline and post-intervention data. Single sample, two sided t-tests were performed to determine whether intercept coefficients were different from zero, and single sample, single sided t-tests were performed to determine whether slope coefficients were greater than zero. Two sample two sided t-tests were performed to determine whether slope coefficients for the DASH and RC treatments were different from one another.

2) To test the second hypothesis that adolescents who achieve a greater reduction in DGI or DGL would have a greater decrease in SBP and DBP from baseline to post-intervention as compared to those with no or less reduction in dietary GI/GL the following statistical analyses were run: Simple linear regression was performed to determine the signed
magnitude of the slope of the hypothesized linear relationship between the output variables, ΔSBP and ΔDBP, and each of the input variables, ΔDGI and ΔDGL, separately. Statistically significant positive regression slopes were interpreted as confirming the hypothesis that a greater reduction in DGI and DGL was associated with a greater decrease in SBP and DBP from baseline to post-intervention. Multiple regression analysis was used to examine the input variables together and their relationships with change in SBP and change in DBP.
VIII. Results

Table 1 shows baseline characteristics of the groups randomly assigned to DASH and RC. Subjects missing data for blood pressure or dietary were excluded. Groups were similar with respect to gender, race and age.

Table 1 Baseline characteristics of groups randomly assigned to the DASH treatment and Routine care. Values are expressed in terms of mean (SD) or frequency.

<table>
<thead>
<tr>
<th>Variable</th>
<th>DASH group (n=23)</th>
<th>RC group (n=22)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, year</td>
<td>14 (2)</td>
<td>14 (2)</td>
</tr>
<tr>
<td>Gender, N</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male:</td>
<td>15</td>
<td>14</td>
</tr>
<tr>
<td>Female:</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Race, N</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black:</td>
<td>7</td>
<td>10</td>
</tr>
<tr>
<td>White:</td>
<td>16</td>
<td>12</td>
</tr>
</tbody>
</table>
Table 2 shows baseline and post-treatment values for BMI, SBP, DBP, calculated DGI and DGL by group. Baseline and post-treatment values for all variables were similar for DASH and RC participants, with the exception of SBP. SBP was significantly greater in the DASH participants as compared to the RC participants at baseline.

Table 2. Baseline and post-treatment BMI, blood pressure and dietary indices. Values are expressed in terms of mean (SD).

<table>
<thead>
<tr>
<th>Variable</th>
<th>DASH</th>
<th>RC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Post-treatment</td>
</tr>
<tr>
<td>BMI, kg/m2</td>
<td>30 (6)</td>
<td>29 (6)</td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>133 (9)</td>
<td>121 (11)</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>79 (7)</td>
<td>73 (9)</td>
</tr>
<tr>
<td>DGI</td>
<td>59 (7)</td>
<td>56 (6)</td>
</tr>
<tr>
<td>DGL</td>
<td>208 (149)</td>
<td>253 (186)</td>
</tr>
</tbody>
</table>

*Indicates significant difference between groups at baseline (p<.01).
Table 3 shows effects of the DASH and RC interventions on change in SBP, DBP, DGI and DGL from baseline to post intervention. The DASH intervention lowered SBP (-12(2) mmHg, p= 0.001) but no effect on the RC intervention was detected (-1(1) mmHg, p=0.19). Thus the two interventions had different effects on SBP (p=0.0007). Both interventions lowered DBP similarly (p=1.0). These results are similar to those previously reported by Couch et al. (10) in the larger clinical trial. Also, as shown in Table 3, neither the DASH treatment nor the RC intervention reduced DGL (DASH: +45 (29), p=0.06; RC: +36 (37), p=0.20). Both treatments showed a non-significant decrease in DGI (DASH: -3 (3), p=.09; RC: -2 (2), p=0.17). Thus the two treatments had similar effects on DGI (p= 0.50) and DGL (p= 0.13). The DGL actually increased from baseline to post-treatment while the DGI showed a slight increase; however, these results are non-significant. Therefore, these findings are contrary to our research hypothesis.

**Table 3** Effects of the DASH and RC treatments on outcome variables from baseline to post intervention. Results are expressed as mean (SE).

<table>
<thead>
<tr>
<th></th>
<th>ΔSBP</th>
<th>ΔDBP</th>
<th>ΔDGI</th>
<th>ΔDGL</th>
</tr>
</thead>
<tbody>
<tr>
<td>DASH (n=23)</td>
<td>-12 (2)</td>
<td>-6 (2)</td>
<td>-3 (3)</td>
<td>45 (29)</td>
</tr>
<tr>
<td>RC (n=22)</td>
<td>-2 (2)</td>
<td>-6 (2)</td>
<td>-2 (2)</td>
<td>36 (37)</td>
</tr>
<tr>
<td>Two sample</td>
<td>-11 (3)</td>
<td>0 (1)</td>
<td>0 (2)</td>
<td>11 (10)</td>
</tr>
<tr>
<td>P value</td>
<td>.0007</td>
<td>1.0</td>
<td>0.50</td>
<td>0.13</td>
</tr>
</tbody>
</table>
Table 4 shows the association by intervention group of change in DGI with change in SBP and DBP using simple linear regression analysis. This relationship is also demonstrated in Figure 1. The effects of the DASH treatment on ΔSBP were positively associated with those on ΔDGI (b=0.3 (0.2), r=0.3, p=0.60). Additionally the effects of the RC treatment on ΔSBP were positively associated with those on ΔDGI (b=0.17 (0.1), r=0.26, p=0.89), but this association was not significant for either treatment. A significant positive association between ΔSBP and ΔDGI was not detected when both treatment groups were considered together. The positive associations of the DASH treatment on ΔDBP with those on ΔDGI were not significant (b=0.10 (0.2), r=0.08, p=0.65) nor were the effects of the RC treatment on ΔDBP positively associated with those on ΔDGI (b=0.017 (0.1), r=0.24, p=0.87). A positive association between ΔDBP and ΔDGI was not detected when both treatments were considered together (b=−0.01 (0.0), r=0.03, p=0.75). These findings are contrary to our hypothesis.
Table 4 Slope (b) and associated correlation coefficient (r) in regression of ΔSBP and ΔDBP on ΔDGI. Model: ΔSBP= b*ΔDGI + constant; ΔDBP= b*ΔDGI + constant. Regression slopes are expressed as mean (SE). Transformed variables are included in the data for the DASH and RC interventions.  *(1-sample, 1-sided>0)

<table>
<thead>
<tr>
<th></th>
<th>ΔSBP</th>
<th>ΔDBP</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DASH</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b</td>
<td>0.3 (0.2)</td>
<td>0.10 (0.2)</td>
</tr>
<tr>
<td>r</td>
<td>0.03</td>
<td>0.08</td>
</tr>
<tr>
<td>P value*</td>
<td>0.60</td>
<td>0.65</td>
</tr>
<tr>
<td><strong>RC</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b</td>
<td>0.17 (0.1)</td>
<td>-0.17 (0.1)</td>
</tr>
<tr>
<td>r</td>
<td>0.26</td>
<td>0.24</td>
</tr>
<tr>
<td>P value*</td>
<td>0.89</td>
<td>0.87</td>
</tr>
<tr>
<td><strong>All subjects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b</td>
<td>0.11 (0.2)</td>
<td>-0.01 (0.0)</td>
</tr>
<tr>
<td>r</td>
<td>0.13</td>
<td>0.03</td>
</tr>
<tr>
<td>P value*</td>
<td>0.80</td>
<td>0.75</td>
</tr>
</tbody>
</table>
**Figure 1** Effects of the DASH diet and RC on ΔDGI and ΔSBP

![Graph showing the effects of DASH diet and RC on ΔDGI and ΔSBP]
Table 5 shows the association by intervention group of change in DGL with change in SBP and DBP using simple linear regression. This relationship is also shown in Figure 2. Contrary to our hypothesis, an increase in DGL was associated with a decrease in SBP and DBP for both treatments. There was no detectable association between the effects of the DASH treatment on ΔSBP and those on ΔDGL (b=-0.01 (0.01), r=0.18 p=0.70). Nor was there any association between the effects of the RC treatment on ΔSBP and those on ΔDGL (b=0.0 (0.01), r=0.20 p=0.50). A positive association between ΔSBP and ΔDGL was not observed when both treatments were considered together (b=-0.0 (0.01), r=-0.03 p=0.60).

The effects of the DASH treatment on ΔDBP were not positively associated with those on ΔDGL (b=-0.0 (0.01), r=0.09 p=0.62). Similar results were obtained for the RC treatment (b=-0.0 (0.01), r=-0.0 p=0.54). A positive association between ΔDBP and ΔDGL was not observed when both treatments were considered together (b=-0.001(0.00), r=-0.03 p=0.61).
Table 5 Slope (b) and associated correlation coefficient (r) in regression of ΔSBP and ΔDBP on ΔDGL. Model: \( \Delta \text{SBP}= b*\Delta \text{DGL} + \text{constant} \); \( \Delta \text{DBP}=b*\Delta \text{DGL} + \text{constant} \). Regression slopes are expressed as mean (SE). * (1-sample, 1-sided>0)

<table>
<thead>
<tr>
<th></th>
<th>ΔSBP</th>
<th>ΔDBP</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DASH</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b</td>
<td>-0.0 (0.01)</td>
<td>-0.00 (0.01)</td>
</tr>
<tr>
<td>r</td>
<td>0.18</td>
<td>0.09</td>
</tr>
<tr>
<td>P value*</td>
<td>0.70</td>
<td>0.62</td>
</tr>
<tr>
<td><strong>RC</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b</td>
<td>0.0 (0.01)</td>
<td>-0.00 (0.01)</td>
</tr>
<tr>
<td>r</td>
<td>0.20</td>
<td>-0.0</td>
</tr>
<tr>
<td>P value*</td>
<td>0.50</td>
<td>0.54</td>
</tr>
<tr>
<td><strong>All subjects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b</td>
<td>-0.00 (0.01)</td>
<td>-0.00 (0.00)</td>
</tr>
<tr>
<td>r</td>
<td>-0.03</td>
<td>-0.03</td>
</tr>
<tr>
<td>P value*</td>
<td>0.60</td>
<td>0.61</td>
</tr>
</tbody>
</table>
Figure 2 Effects of the DASH diet and RC on ΔDGL and ΔSBP
IX. Discussion

To our knowledge this post-hoc analysis of dietary and blood pressure data from hypertensive or pre-hypertensive adolescents participating in either an intervention focused on the DASH diet or one focused on fat and sodium reduction is among the first to examine the relationship between change in DGI and DGL and change in blood pressure. There were two major findings from this study. First, on average, DGI and DGL did not significantly decrease in response to either the DASH or RC interventions. The second major finding was that the mean change observed in SBP and DBP among the DASH and RC participants was not associated with the mean change in DGI and DGL. These findings do not support the idea that DGI or DGL mediate the blood pressure changes associated with the DASH diet, as demonstrated by Couch et al. (12).

The observation that blood pressure decreased in both treatment groups, even though DGI and DGL did not change significantly suggests that the effect of the DASH and RC treatment on blood pressure was independent of DGI and DGL. Previously, Couch and colleagues (12) noted that participants in the DASH intervention increased their fruit intake by two servings from baseline to post-treatment. This may explain the increase in GL in this group, which is very dependent on the amount or number of servings of carbohydrate that is eaten. In addition, the DASH intervention focused on lowering high fat and high sodium food choices. In the food industry, as manufacturers lower fat in their products, fat is replaced with sugar in an effort to improve the palatability of the final food product. In modifying foods to be lower in fat, the GI of many low fat foods is increased. Participants in the DASH group significantly lowered their dietary fat, and may have inadvertently done this at the expense of increasing their intake of refined starches and adding more sucrose into the diet. Our results suggest this to be true. The
question of why then did GI not show a significant increase still remains. This may be explained in part by the large standard deviation in DGI values post-treatment for members of the DASH intervention compared to the RC group. Another plausible explanation is that if DASH participants decreased their dietary fat, as indicated by Couch et al. (12), they likely increased their carbohydrate intake, which may account for a greater increase in DGL. Calculated DGL is dependent not just on GI, but also on the amount of carbohydrate. Changes in the DGI of the RC group were minimal and may reflect the fact that this group as a whole was not able to achieve significant increases in fruit intake, or lower dietary fat as compared to the DASH participants (12). Findings from this study also support a lack of association between change in DGI and DGL and change in blood pressure.

The results of this study are consistent with previous research on this topic. Even though DGI and DGL have been associated with improved CVD risk factors including plasmin activator inhibitor-1 (16), triglycerides (16), C-reactive protein (17), HDL (18, 24), LDL (18, 28), and BMI (25, 26), GI or GL have not been found to have a significant impact on blood pressure (16, 28, 29). Ebbeling et al. (16) found that while the effects of an ad libitum low GL diet led to a significant decrease in several CVD risk factors including triglycerides and PAI-1, no effect was seen on SBP or DBP. Sloth et al. (28) also studied the effects of GI on risk factors for ischemic heart disease and found that a low-GI diet decreased LDL cholesterol by 10%, but saw no significant differences between the low-GI and high-GI groups in SBP or DBP after ten weeks of treatment (28). Additionally, Frost et al. (29) studied the effect of a low-GI diet in patients with CHD and found that although GI decreased by ten and GL decreased by thirty-one in response to low-GI dietary compliance, there were no significant changes in blood pressure as a result. Thus,
although the DASH diet does appear to have beneficial effects on blood pressure as observed in adults by Fung et al. (30), Folsom et al. (31), Azadbakht et al. (32), and in adolescents (12) the effect does not appear to be mediated by the GI or GL value of the diet. This argument is further strengthened when considering that the DASH participants achieved a reduction in SBP and DBP despite the fact the no significant change was detected in DGI or DGL.

This thesis work is not without limitations. One limitation of the present study is the small sample size, which may have minimized the ability to find true associations between diet and blood pressure that may have been present. A second limitation is the fact that DGI and DGL in this study were evaluated in response to a diet education program. They were not evaluated under controlled feeding conditions so true associations between these variables and blood pressure may have been masked by the high degree of variability in daily eating patterns among participants. Third, it should be considered that nutrients are not eaten in isolation nor do they work in isolation. Because fiber, protein and fat intake can affect the absorption of glucose into the blood stream, the DGI can only approximate the blood-glucose-raising effect of a mixed diet. This can be seen by the fact that some studies on GI and GL treat fiber as a confounding variable. Fourth, confounders such as BMI and gender were not controlled in the data analyses which may affect the result. Finally, the results from the initial study done by Couch et al. (12) show poor compliance to treatment in some of the adolescents, which is not uncommon for this age group. Because a true DASH diet was achieved by only a small number of participants, the ability to show change in DGI and DGL in response to a DASH diet may have been underestimated.

Conclusion
On the basis of this study, our results suggest that modifying diet to comply with a DASH-type diet may result in changes in DGI and DGL, which are not associated with a change in blood pressure. However, because of the small sample size and few number of participants who actually achieved a “true” DASH diet in this trial, the DASH diet cannot be definitively characterized as a high or low-GI or GL diet. More research is needed to clarify this issue. These results suggest that the effects of DASH on blood pressure may be mediated by some component of the DASH diet other than GI or GL.

*Implications for future research*

As suggested previously, confidence in the conclusions derived from this study are limited by the fact that neither GI nor GL were controlled in this study. Therefore, the potential causal relationships between GI and GL and blood pressure were not tested directly. The design of an experiment to do so should begin with the question of how large of a decrease in GI or GL would be necessary to see any significant changes in SBP or DBP.

Further research is also needed to study if the DASH diet combined with a low-GI diet has a greater impact on CVD risk factors than the DASH diet or low-GI alone. Finally, research is needed to investigate which component of the DASH diet does reduce blood pressure. Possibilities include potassium (11), low fat dairy (28), amount of saturated fat (28), low sodium, and the combined actions of greater amounts of fruits and vegetables. It is also entirely possible that the all components of the DASH diet must be present together in order to reduce blood pressure.
REFERENCES


