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Vegetable consumption linked to decreased visceral and liver fat and improved insulin resistance in overweight Latino youth

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Abstract

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There is limited data on the impact of vegetable consumption on adiposity and metabolic health, specifically non-starchy vegetables (NSV) and vegetables that are dark green and deep orange/yellow (also known as nutrient-rich vegetables, NRV). This study examines the relationship between vegetable intake and adiposity, liver fat and insulin dynamics in overweight Latino youth. This cross-sectional study of 175 overweight (≥85th percentile BMI) Latino youth (8–18 years), with data collected 2006–2011, included the following: dietary intake via multiple 24-h recalls, total body fat via dual-energy x-ray absorptiometry, adipose tissue distribution and liver fat via magnetic resonance imaging, and insulin dynamics via frequently-sampled intravenous glucose tolerance test. Linear regression and analysis of covariance were used for analysis, with the following a priori covariates: age, sex, energy intake and total body fat. Participants who consumed the most NSV (mean intake = 1.7±1.0 servings/d) compared to the least (mean intake = 0.1±0.1 servings/d) had 44% less liver fat (10.0±8.5 vs. 5.6±8.7%, p=0.01). NRV intake was positively correlated with insulin sensitivity (SI, r=0.19, p=0.03). Consumers of NRV (mean intake = 0.3±0.4 servings/day, n=107), compared to non-consumers (n=68), had 31% increased SI (1.6±1.6 vs. 2.1±1.3 × 10^{-4}·min^{-1}·μU^{-1}·mL^{-1}, p=0.03) and 17% less visceral adipose tissue (2.3±0.9 vs. 1.9±0.7 L, p=0.01). In conclusion, consumption of specific vegetable types by overweight Latino youth is associated with positive metabolic outcomes, including reduced visceral and liver fat and risk factors for type 2 diabetes, even when consumed in small quantities. These may be relevant targets for interventions.

Keywords

obesity; Latino; adipose tissue; diabetes; vegetables

Introduction

Poor dietary habits are implicated in the development of chronic disease, yet dietary recommendations are consistently unmet, especially in children. The United States Department of Agriculture (USDA) My Plate recommends 2–3 servings of vegetables per day based on age and sex, yet data from the National Health and Nutrition Examination Survey indicate that less than 6% of adolescents meet these recommendations [1,2].

The low intake of vegetables is concerning, given that these foods are specifically connected to the prevention of many chronic conditions, including cardiovascular disease and diabetes [3–5]. However, mixed evidence exists for the association between vegetable intake and obesity (a condition strongly associated with many chronic diseases), possibly due to the frequent use of imprecise measurements such as BMI[6]. Micronutrients found in foods, specifically dark green and deep orange/yellow vegetables (also know as nutrient-rich vegetables, or NRV) have been shown to have protective properties against cardiovascular disease and cancer, and special emphasis is placed on these foods in My Plate recommendations [2,7]. However, little research has focused on these high-nutrient foods, especially in children.

Dietary habits in children are especially important, given that many of these habits persist into adulthood [8], and because many chronic conditions are becoming more prevalent in

younger populations. Furthermore, many metabolic diseases, including diabetes, are more widespread in ethnic minorities[9]. Previous work with overweight Latino children has shown prediabetes in 30% of study participants, and high levels of visceral adipose tissue (VAT), which is implicated in insulin resistance.[10] High levels of hepatic adipose tissue were also observed, with 38% of this cohort having clinical signs of non-alcoholic fatty liver disease (NAFLD, liver fat fraction greater than 5.5%) [11].

Given the severity of these conditions in this population of overweight Latino youth, understanding the metabolic impact of lifestyle factors, such as diet, is imperative. The relationship between vegetable intake and metabolic health has not been well examined, especially using rigorous measures of dietary intake and adiposity. This study aims to elucidate the relationship between vegetable consumption, including NRV intake, with insulin dynamics and adipose tissue distribution in high-risk Latino youth. We hypothesize that those who consumed greater quantities of vegetables, specifically nutrient rich vegetables, would have better insulin sensitivity and decreased abdominal adiposity, compared to those who consumed the least.

Methods

Participants

This cross-sectional study utilizes data from five studies at the University of Southern California with consistent data collection methodology and measures, collected from 2006 – 2011[12–15]. Two of these studies were observational, while the other three were lifestyle modification programs (diet and/or physical activity and/or stress reduction). Participants were incentivized to participate via a cash incentive and learning about their personal metabolic risk (observation studies), or the aforementioned plus participation in an obesity-reduction program (intervention studies). All data included in this analysis were collected prior to any intervention participation. Criteria for inclusion in these analyses included a) BMI ≥ 85th percentile for age and sex based on guidelines from the Centers for Disease Control and Prevention [16], b) self-report of Latino descent in all four grandparents, c) no conditions or medications that may influence metabolism or body composition, d) completed at least one study visit with adiposity and metabolic data collected, and at least two valid days of dietary data. These studies were approved by the Institutional Review Board at the University of Southern California, and were compliant with the Health Insurance Portability and Accountability Act. Informed assent and consent were obtained from all children and parents. The final samples included 175 participants.

Physical, Adiposity, and Metabolic Data

Participants completed a comprehensive physical exam and medical history with a licensed health care provider at the University of Southern California General Clinical Research Center (or the Clinical Trials Unit at University Hospital after 2008). This evaluation included weight measured to the nearest 0.1 kg on a medical scale (Health O Meter Professional ProPlus, Beford Heights, OH) and height measured to the nearest 0.1 cm using a wall-mounted stadiometer (Seca 240, Chino, CA). Total body fat was measured using dual-energy X-ray absorptiometry (DXA) using a Hologic QDR 4500W, QDR software

version 12.6 (Beford, MA, CV 2.7% in similar sample)[17]. Abdominal adipose tissue was measured at the Los Angeles County + University of Southern California Imaging Science Center using cross-sectional magnetic resonance imagining (MRI) for subcutaneous abdominal adipose tissue (SAT), visceral adipose tissue (VAT), and hepatic fat fraction (HFF), as described elsewhere [11,18]. Participants were scanned using either a General Electric 1.5-Telsa magnet (n=128, Waukesha, WI) or 3.0 Telsa magnet also by General Electric (n=47), with a correction factor applied based on duplicate measures in a sample of 18 young adults[18] (SAT CV 1.50%, VAT CV 3. 69%, HFF CV 1.18% in healthy adult sample)[19]. Those with HFF ≥ 5.5% were classified as having NAFLD.

Additionally, after receiving a standardized dinner, participants underwent an overnight fast with a frequently sampled intravenous glucose tolerance test in the morning,[20,21] as previously described [22]. All samples were centrifuged (10 minutes, 2500 rpm, 8–10°C), and plasma aliquots were frozen at −70°C until analysis. Glucose samples were assayed using the glucose oxidase method using a Yellow Springs Instrument (model 2700, Yellow Springs, OH, CV <2%). Insulin was assayed using either a specific human insulin enzyme-linked immunoabsorbent assay kit (Linco, St. Charles, MO) or an automated enzyme immunoassay (Tosoh AIA 600 II analyzer, Tosoh Bioscience Inc., S San Francisco, CA); %CV intra-assay 5.96 ± 1.17, inter-assay 10.3 ± 0.9. Glucose and insulin concentrations were analyzed using MINMOD Millennium 2003 software (version 5.16, Richard N. Bergman, Los Angeles, CA) to obtain values for insulin sensitivity (SI), acute insulin response (AIR) and disposition index (DI).[21] Homeostasis model assessment of insulin resistance (HOMA-IR) was calculated as [fasting insulin (μIU/mL)*fasting glucose (mg/dL)]/405.[23]

Dietary intake

Dietary intake was assessed by research staff trained and supervised by an registered dietician, through 24-hour dietary recalls using the multiple pass technique[24,25]. Recalls were done in person or over the phone, using paper format and visual aids (measuring cups and spoons, or booklets for phone recalls). At least 2 recalls were collected from participants within a 4-week period (average time between recalls = 4.8 ± 6.0 days). An effort was made to collect recalls on two weekdays and one weekend day, and an average of 2.0 ± 0.7 recalls were collected on weekdays and 0.8 ± 0.5 recalls were collected on weekends. Recalls were not collected on major holidays, and were omitted if the participant was sick (n= 7), or reported fasting for religious reasons (n=1). An average of 2.8 ± 0.7 recalls were utilized from each participant. Data were collected throughout the calendar year.

Data were analyzed using the NDS-R 2011 software (Nutrition Data System for Research, 2011, University of Minnesota, Minneapolis, MN). This program calculates total energy intake and serving data for various food groups, based on the Dietary Guidelines for Americans 2005 [26] and the USDA Food Guide Pyramid. All fried vegetables, vegetable juice, beans and tomatoes were excluded from this analysis of vegetable consumption. Tomatoes were excluded from this analysis because they are a fruit, and because this serving category also includes tomato sauces and pastes, which may have added sugars. Non-starchy vegetables (NSV) included the following serving categories: dark green, deep yellow/

orange, and other vegetables (a category which includes vegetables such as beets, cabbage and summer squash), and excluded white potatoes and other starchy vegetables (which includes corn, lima beans, and peas, among others). Servings of dark green and deep yellow/orange vegetables were combined to create the NRV serving category, which is a subset of NSV. Dietary data were cleaned to remove participants with improbable caloric intake by performing linear regression of mean caloric intake on body weight. Those with residuals greater than ± 2 SD from the mean were omitted (n=7).

Statistics

Prior to beginning analyses, data were checked for normality and variables were log (VAT, HFF, glucose, SI) or square root transformed (SAT, insulin, HOMA, AIR, DI). Because all participants were overweight with higher risk for metabolic disease, it is not unexpected for data to be skewed. For ease of interpretation, means provided in tables and text are untransformed. Independent variables included total vegetables, NSV, NRV, and dietary fiber (as a potential mechanism). Partial correlations were examined first, and analyses of covariance of intake quartiles with a Tukey adjustment for multiple comparisons were examined next, due to minimal variance in diet variables. Ranked groups were unable to be established for NRV because a large number of participants had no intake of these foods, so this variable was dichotomized into consumers and non-consumers (consumers were defined as > 0.0 servings of NRV, all others were defined as non-consumers). Participants missing data on specific measures (due to scheduling, which was primarily an issue with MRIs; discomfort; or other conflicts) were excluded from relevant models (n=0 missing glucose; n=3 missing insulin and HOMA; n=14 missing SI, DI and AIR; n=32 missing SAT and VAT; n=39 missing HFF). The following a priori covariates were included in all analyses: sex, age, energy intake, and body fat mass. Fiber was also included as a covariate in follow-up analyses. All analyses were performed using SAS (version 9.2, 2008, SAS Institute Inc., Cary, NC) with a two-sided significance level set at $\alpha < 0.05$.

Results and Discussion

One hundred and seventy five overweight Latino children (31% male, 8–18 years, >85th percentile BMI for age and sex) had adiposity and metabolic data, and plausible dietary data (Table 1). Mean vegetable consumption (excluding fried, including starchy) was 1.0 ± 1.1 serving per day, and mean intake of NRV was 0.2 ± 0.4 servings per day.

There was a significant positive partial correlation between SI and NRV consumption (r=0.19, p=0.03), independent of age, sex, energy intake and body fat mass. No other partial correlations were statistically significant, although trends for inverse associations (p<0.10) were observed in HFF and AIR with NSV consumption.

Analysis of covariance found significant differences in HFF among quartiles of both total vegetables and NSV (p=0.03 and p=0.01, respectively, Table 2). Participants who consumed the greatest amount of total vegetables (Q4; mean intake of 2.5 ± 1.2 servings/d, HHF 6.6 ± 8.7%) had a 39% lower HFF compared to those with the lowest intake (Q1; mean intake of 0.1 ±0.1 servings/d, HFF 10.9 ± 8.6%). These findings were strengthened when examining only NSV. After adjusting for a priori covariates, participants who consumed the most NSV

(Q4 mean intake of 1.7 ± 1.0 servings/d) compared to those who consumed the least NSV (Q1 mean intake 0.1 ± 0.1 servings/d) had a 44% less HFF (5.6 ± 8.7 % HFF vs. 10.0 ± 8.5%;, p=0.01; see Table 2 for additional comparisons.). Those who consumed the most NSV had 54% lower prevalence of NAFLD (27.3%), compared to those who consumed the least NSV (59.4%, p<0.01, data not shown).

There were also significant differences in AIR by quartiles of all vegetables and NSV (p=0.01, Table 3). For NSV, those who consumed the least amount (Q1; mean intake of 0.1 ±0.1 servings/d) had significantly higher AIR (1874.1 ± 1001.9 mU/mL × 10 min), compared to those who consumed the most (Q4 mean intake 1.7± 1.0 servings/d, AIR 1200.5 ± 1003.1 mU/mL × 10 min, p=0.04; see Table 3 for additional comparisons).

Consumers of NRV (mean intake 0.3 ± 0.4; n=107), compared to non-consumers (mean intake 0.0 ± 0.0; n=68), had 31% higher SI (p=0.03, Table 3), 17% lower VAT (p=0.01, Table 2), and 25% lower AIR, although not significant (p=0.05, Table 3), after adjusting for covariates. When fiber was added as a covariate, all significant relationships remained ($p < 0.05$). No significant associations were found between fiber and all outcome variables (data not shown).

Consistent with other reports of dietary intake among adolescents [1,27], vegetable intake in this sample of overweight Latino youth was well below the recommended guidelines, at one serving of total vegetables per day. Youth with the highest intake of vegetables, particularly NSV, had significantly lower HFF compared to those who consumed the least amount. Additionally, those who consumed NRV had lower VAT and higher SI compared to those who did not consume NRV. These findings demonstrate that dietary patterns are associated with metabolic disease, which is particularly relevant in a population with a high predisposition for pre-diabetes and fatty liver disease [10,28].

The relationship between fruit and vegetable intake and adiposity is limited, especially in children, and only 1 in 4 longitudinal studies in a recent review found the expected inverse association [29]. However, these studies primarily used BMI as an indicator of adiposity, which does not account for body fat distribution. Conversely, one study with 325 predominantly Caucasian young children (3–8 years) measured whole body fat using DXA, and found a dietary pattern high in NRV was associated with reduced body fat [30]. Findings presented here use fat distribution measured by MRI, and indicate that vegetable consumption is associated with decreased hepatic fat (for NSV) and visceral fat (for NRV), which are implicated in type 2 diabetes (T2D) development [31]. These findings suggest that a difference of approximately 1.5 servings (about ¾ cups) of NSV per day is associated with liver fat near normal levels, although future research is needed to determine if this could be effective for treatment or prevention.

Similar to reports on adiposity, the link between vegetable consumption and diabetes risk is inconsistent. Meta-analyses have shown pooled relative risks between fruit and vegetables consumption and T2D diagnosis are non-significant, but this may be due to the use of food frequency screeners, which provide limited reflections of dietary intake [32,33]. Additionally, diagnosis of T2D may provide another limitation, as this relationship may be more complex

than is evident from a simple clinical test. It may be possible that fasting glucose and HOMA-IR may not be precise enough to measure this relationship, demonstrated by the significant findings with NRV and SI only. However, hazard ratios in a meta-analysis by Carter et al. showed a 1.15 serving increase of green leafy vegetables resulted in a 14% reduced risk for T2D [32], suggesting that even with a simple measure, NRV may be beneficial. Our findings are consistent and demonstrate that NRV consumption is positively associated with SI. We also found that non-consumers of NRV had increased AIR, which would reflect the compensatory insulin response to the decreased SI. Similarly, we saw that low NSV consumers had significantly higher AIR, coupled with lower SI, although not significant ($r=0.14$, $p=0.12$).

The pathway through which vegetables influence health are not well understood. Dietary fiber is a potential mechanism, by limiting insulin response following meals and slowing gastric emptying and thus macronutrient absorption.[34,35] Previous research has shown that increases in dietary fiber in overweight Latino youth contributed to a 4–10% decrease in VAT,[22,36] yet there was no association between fiber and adiposity and metabolic outcomes in this analysis, possibly due to low fiber intake among all participants. Yet, fiber intake was highly correlated with all vegetable serving groups ($p<0.0001$), and all significant relationships remained when fiber was controlled. This supports the whole food hypothesis,[37] in that nutrients in isolation may not be sufficient to achieve positive health effects, and whole food should be consumed instead. It is likely that the combinations of compounds are driving the positive relationships with metabolic health. These can include polyphenols, which may impact sugar digestion by altering sucrose breakdown and glucose and fructose transport,[38] and may also be protective against NAFLD,[39] yet further research is needed on these potential mechanisms. These data may also indicate general healthy living practices.

One major limitation of this study is the cross-sectional design, which prevents conclusions of causal relationships. Furthermore, there may be other dietary patterns or lifestyle factors that may confound this relationship, or work in conjunction with vegetable intake (for example, stress) to affect metabolic health. Another possible lifestyle determinant is physical activity, which we have previously shown to be associated with improved hepatic fat, adiposity and glucose control in minority youth, [41–43] but this was not included in this analysis given systematic missing data. Additionally, a minimum of two 24-h dietary recalls may not be sufficient to explain long-term dietary patterns (especially of NRV, which may be more inconsistent than other variables), and underreporting is a frequent limitation of dietary recall.[40] Also, fruit is not presented in this data and further research is needed to understand its impact on metabolic disease risk.

In lieu of these limitations, there are strengths of these analyses. Because participants were unaware of their metabolic disease status at the first data collection time-point, it is unlikely that dietary intake was determined by this information. Also, the homogeneity of the study sample may minimize between-group differences. Dietary data were collected throughout the year, but the year-round growing season in Southern California makes seasonal variation unlikely. Finally, although generalizability to other populations is limited, understanding the impact of diet on disease risk in this high-risk population is warranted.

Conclusions

Consumption of non-starchy vegetables is associated with lower liver fat deposition, and dark green or bright orange/yellow vegetable intake is associated with lower visceral fat and improved insulin sensitivity. This finding is especially important given the low intake of these foods in this population of Latino youth. The mean difference between consumers and non-consumers of NRV was less than ½ serving, equivalent to just ¼ cup of carrots or broccoli, which may be a very attainable and useful recommendation if shown to have a causal impact on health. These two vegetables (carrots and broccoli) were among the most commonly consumed, along with lettuce, green beans, and cucumbers, and may be simple recommendations for youth. Other less popular nutrient-rich vegetables, such as dark leafy greens and winter squashes, could be important targets in interventions to increase exposure and availability. Programs to decrease metabolic disease risk by targeting these vegetables should be explored, especially those with Latino youth.

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Table 1

Physical, metabolic and dietary characteristics of overweight Latino youth 8–18 years in a cross-sectional sample (n=175) [a]

Physical characteristics	
Sex (M/F)	55/120
Age (y)	15.0 ± 2.4
BMI percentile	96.7 ± 3.5
BMI *z* score	2.0 ± 0.4
Total fat (kg)	31.6 ± 10.8
Total lean (kg)	48.3 ± 11.6
Adiposity and insulin dynamics [b]	
SAT (L)	13.4 ± 7.1
VAT (L)	1.9 ± 1.3
HFF (%) [c]	8.2 ± 7.4
Fasting glucose (mg/dL)	91.1 ± 5.9
Fasting insulin (μIU/mL)	17.6 ± 11.1
HOMA-IR ($\times 10^{-4} \cdot \text{min}^{-1} \cdot \mu\text{U}^{-1} \cdot \text{mL}^{-1}$)	4.0 ± 2.7
SI ($\times 10^{-4} \cdot \text{min}^{-1} \cdot \mu\text{U}^{-1} \cdot \text{mL}^{-1}$)	1.8 ± 1.6
AIR (μU/mL × 10 min)	1344.6 ± 882.2
Disposition index ($\times 10^{-4}$/min)	1977.9 ± 1048.7
Dietary characteristics	
Energy (kcal)	1766.3 ± 566.2
Vegetables (servings/d) [d]	1.0 ± 1.1
Non-starchy vegetables (servings/d)	0.7 ± 0.8
Nutrient-rich vegetables (servings/d)	0.2 ± 0.4
Dietary fiber (g/1000 kcal)	8.4 ± 3.2

[a] Values are mean ± SD except sex.

[b] AIR: Acute insulin response, HFF: Hepatic fat fraction, HOMA-IR: Homeostasis assessment model of insulin resistance, SI: Insulin sensitivity, SAT: Subcutaneous adipose tissue, VAT: Visceral adipose tissue.

[c] 42% of subjects (n=57) with HFF data had non-alcoholic fatty liver disease (HFF ≥ 5.5%)

[d] All vegetable categories exclude fried.

Table 2

Differences in adipose tissue distribution by vegetable intake quartiles in a cross-sectional sample of overweight Latino youth 8–18 years [a, b, c]

All vegetables

Quartiles of intake	**Q1**	**Q2**	**Q3**	**Q4**	**p-value (between group comparisons)**
servings	**0.1 (0.1)**	**0.5 (0.1)**	**0.9 (0.2)**	**2.5 (1.2)**	
n	**43**	**44**	**44**	**44**	
SAT volume (L)	13.7 (5.0)	13.5 (5.3)	13.5 (4.8)	14.2 (5.1)	ns
VAT volume (L)	2.1 (1.1)	2.3 (1.2)	1.8 (1.1)	2.3 (1.2)	ns
HFF (%)	10.9 (8.6)	9.5 (8.9)	8.5 (8.1)	6.6 (8.7)	0.03 [d] (Q1–Q4: p<0.05)

Non-starchy vegetables

Quartiles of intake	**Q1**	**Q2**	**Q3**	**Q4**	**p-value (between group comparisons)**
servings	**0.1 (0.1)**	**0.4 (0.1)**	**0.7 (0.1)**	**1.7 (1.0)**	
n	**43**	**44**	**44**	**44**	
SAT volume (L)	13.5 (5.0)	13.4 (5.0)	13.6 (4.9)	14.2 (5.1)	ns
VAT volume (L)	2.3 (1.2)	2.2 (1.2)	2.0 (1.1)	1.9 (1.2)	ns
HFF (%)	10.0 (8.5)	10.0 (8.3)	9.6 (7.9)	5.6 (8.7)	0.01 [d] (Q1–Q4: p=0.01 Q2–Q4: p=0.03)

Nutrient-rich vegetables

	Non-consumers	**Consumers**	**p-value**
servings	**0.0 (0.00)**	**0.3 (0.4)**	
n	**68**	**107**	
SAT volume (L)	14.4 (4.0)	13.3 (3.2)	ns
VAT volume (L)	2.3 (0.9)	1.9 (0.7)	0.01 [d]
HFF (%)	9.5 (6.6)	8.5 (5.6)	ns

[a] All vegetable categories exclude fried.

[b] Data are mean (SD), p-values were determined using ANCOVA, with a Tukey adjustment for multiple comparisons between groups. All data are adjusted for age, sex, energy intake, and body fat mass.

[c] HFF: Hepatic fat fraction, SAT: Subcutaneous adipose tissue, VAT: Visceral adipose tissue

[d] Significant relationships are preserved when means are further adjusted for fiber.

Table 3

Differences in insulin dynamics by vegetable intake quartiles in a cross-sectional sample of overweight Latino youth 8–18 years [a, b, c]

All vegetables					
Quartiles of intake	**Q1**	**Q2**	**Q3**	**Q4**	**p-value (between group comparisons)**
servings	**0.1 (0.1)**	**0.5 (0.1)**	**0.9 (0.2)**	**2.5 (1.2)**	
n	**43**	**44**	**44**	**44**	
Fasting glucose (mg/dL)	90.1 (6.6)	92.3 (7.0)	90.8 (6.3)	90.0 (6.7)	ns
Fasting insulin (μIU/mL)	16.8 (11.6)	17.7 (12.3)	16.5 (11.3)	16.8 (11.9)	ns
HOMA-IR ($\times 10^{-4} \cdot min^{-1} \cdot \mu U^{-1} \cdot mL^{-1}$)	3.9 (2.9)	4.1 (3.0)	3.6 (2.8)	3.8 (2.9)	ns
SI ($\times 10^{-4} \times min^{-1} \times mU^{-1} \times mL^{-1}$)	1.7 (2.0)	2.2 (2.1)	1.8 (2.0)	1.9 (2.0)	ns
AIR (mU/mL × 10 min)	1810.6 (1001.0)	1104.2 (1061.7)	1022.3 (1015.1)	1354.9 (1019.0)	<0.01 [d] (Q1–Q2: p=0.04 Q1–Q3: p<0.01)
Disposition index ($\times 10^{-4}$/min)	2142.2 (1230.2)	1855.9 (1304.8)	1851.2 (1247.3)	2064.6 (1252.0)	ns

Non-starchy vegetables					
Quartiles of intake	**Q1**	**Q2**	**Q3**	**Q4**	**p-value (between group comparisons)**
servings	**0.1 (0.1)**	**0.4 (0.1)**	**0.7 (0.1)**	**1.7 (1.0)**	
n	**43**	**44**	**44**	**44**	
Fasting glucose (mg/dL)	90.2 (6.7)	91.0 (6.8)	91.8 (6.4)	89.8 (6.7)	ns
Fasting insulin (μIU/mL)	16.3 (11.7)	17.0 (11.9)	16.9 (11.4)	17.4 (11.7)	ns
HOMA-IR ($\times 10^{-4} \cdot min^{-1} \cdot \mu U^{-1} \cdot mL^{-1}$)	3.8 (2.9)	3.9 (2.9)	3.8 (2.8)	3.9 (2.9)	ns
SI ($\times 10^{-4} \times min^{-1} \times mU^{-1} \times mL^{-1}$)	1.6 (2.0)	2.4 (2.0)	1.7 (2.0)	2.0 (2.0)	ns
AIR (mU/mL × 10 min)	1874.1 (1001.9)	1114.6 (1044.7)	1152.5 (1001.9)	1200.5 (1003.1)	0.01 [d] (Q1–Q2: p=0.03 Q1–Q3: p=0.01 Q1–Q4: p=0.04)
Disposition index ($\times 10^{-4}$/min)	2027.6 (1245.4)	1940.3 (1298.2)	1950.1 (1245.3)	2026.1 (1246.7)	ns

Nutrient-rich vegetables			
	Non-consumers	**Consumers**	**p-value**
servings	**0.0 (0.00)**	**0.3 (0.4)**	
n	**68**	**107**	
Fasting glucose (mg/dL)	89.6 (5.4)	91.5 (4.3)	ns
Fasting insulin (μIU/mL)	17.1 (9.4)	16.8 (7.6)	ns
HOMA-IR ($\times 10^{-4} \cdot min^{-1} \cdot \mu U^{-1} \cdot mL^{-1}$)	3.8 (2.3)	3.8 (1.9)	ns
SI ($\times 10^{-4} \times min^{-1} \times mU^{-1} \times mL^{-1}$)	1.6 (1.6)	2.1 (1.3)	0.03 [d]
AIR (mU/mL × 10 min)	1588.0 (854.0)	1191.0 (685.6)	0.05
Disposition index ($\times 10^{-4}$/min)	2001.5 (1015.7)	1978.5 (815.8)	ns

[a] All vegetable categories exclude fried.

[b] Data are mean (SD), p-values were determined using ANCOVA, with a Tukey adjustment for multiple comparisons between groups. All data are adjusted for age, sex, energy intake, and body fat mass.

[c] AIR: Acute insulin response, HOMA-IR: Homeostasis assessment model of insulin resistance, SI: Insulin sensitivity.

[d] Significant relationships are preserved when means are further adjusted for fiber.