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Alexandra Nutaitis March 22, 2016

Diet may be a Modifiable Risk Factor for Cognitive Decline in African Americans and Caucasians at High Risk for Developing Alzheimer's Disease: A Pilot Study of the Southern and Prudent Diets.

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An abstract of
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Abstract

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Importance: African Americans are 64% more likely to develop Alzheimer's disease than Caucasians, though the reason for this difference is not clear. Investigating dietary patterns as a modifiable risk factor applicable to African Americans and Caucasians has the potential to reduce their risk of developing Alzheimer's disease.

Objective: To determine the influence of the Southern dietary pattern and the prudent dietary pattern on cognitive performance in individuals at high risk for developing Alzheimer's disease due to age, parental history of AD, and race.

Design/Setting: Observational pilot study.

Participants: 66 cognitively normal, healthy, African American and Caucasian individuals aged 46-77 years with a parental history of Alzheimer's disease.

Main Outcome and Measure: Food Frequency questionnaire, comprehensive vascular risk assessments, and cognitive assessments.

Results: Caucasians performed significantly better on 3 of the 8 cognitive assessments, the remaining 5 cognitive assessments did not display any significant racial differences. We report a correlation between foods characteristic of the Southern diet and worse cognitive performance in African Americans. Individuals who reported diets that aligned with the prudent dietary pattern performed better on cognitive testing than those who did not align with the prudent dietary pattern.

Conclusion and Relevance: Dietary patterns are easily modified and current research suggests that even late life dietary pattern modification can result in favorable health outcomes. It is possible that the increased prevalence of Alzheimer's disease in African Americans could be lessened through diet modification.

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Introduction

Estimates suggest that over 5 million people in the U.S. are currently living with Alzheimer's disease (AD), and that in the next thirty years the prevalence will increase to over 16 million Americans (alz.org, 2014). Between 2000 and 2010, deaths from AD increased by 68%, and AD is the only cause of death in America's top six causes of death that cannot be cured (alz.org, 2014; Association, 2015). In 2014, the direct costs for caring for those with AD summated to roughly $214 billion, including $150 billion in costs to Medicare and Medicaid (alz.org, 2014). In the absence of a disease-modifying treatment, from both a fiscal and humanistic standpoint, it is of utmost importance that we fully understand the influence of modifiable risk factors on cognitive health. For individuals at high risk for AD, by virtue of race or parental history, current preventative efforts focus on lifestyle interventions including diet, exercise, and cognitive training (Baumgart et al., 2015; Rege et al., 2016).

Researchers have spent significant resources investigating the potential health benefits of nutrients on cognitive function. Flavonoids (Scholey et al., 2010; Vislocky and Fernandez, 2010; Whyte and Williams, 2015), long chain polyunsaturated fatty acids(Baierle et al., 2014; Cederholm et al., 2013; Oster and Pillot, 2010; Samieri et al., 2012), phosphatidylserine(Glade and Smith, 2015), and dietary nitrate(Wightman et al., 2015) have been implicated as having potentially neuroprotective properties. Although these studies are informative, it is difficult to assess the direct influence of specific nutrients on cognitive health in humans because humans do not eat nutrients in isolation; rather we acquire nutrients synergistically throughout various meals. Assessing dietary patterns allows researchers to take into account the complexity of human diet, as well as the possible synergistic influence of various food components on cognitive function (Granic et al., 2016).

We used the guidelines from the *Reasons for Geographic and Racial Differences in Stroke (REGARDS)* study to determine the foods classified as Southern pattern (characterized by foods such as fried food, added fats, eggs, organ and processed meats and sugar- sweetened beverages)(Shikany et al., 2015). In order to capture dietary pattern behaviors on the opposite side of the spectrum, we contrasted the Southern diet with foods characteristic of the prudent diet. The prudent diet is globally considered a healthy diet, and is characterized by dietary patterns with more frequent fruit, vegetable, whole grain, and fish consumption (Shakersain et al., 2016).

Dietary patterns are modifiable and transferable into daily routine for most people. In addition, adhering to health-conscious dietary patterns would reduce the monetary burdens of cardiovascular disease in the U.S. (Abdullah et al., 2015). Furthermore, contrary to popular belief, healthy eating is not synonymous with spending large amounts of money on food (Primavesi et al., 2015), and many low-income minority populations have been able to overcome financial barriers to acquire positive dietary changes (Cason-Wilkerson et al., 2015).

African Americans (AAs) are 64% more likely to develop AD than Caucasians (CCs) (Steenland et al., 2015) and individuals with a parental history of AD are ten times more likely to become afflicted with AD (alz.org, 2015). It is possible that in addition to potential genetic contributions, a large component of the increased prevalence of AD in AAs may be a result of modifiable risk factors including diet (Biessels, 2014; Chin et al., 2011; Norton et al., 2014). Current literature suggests that geographic and racial differences in stroke are associated with the Southern dietary pattern and thus it is possible that this Southern dietary pattern may also contribute to cognitive decline (Shikany et al., 2015). While researchers have reported the impact

of specific nutrients on cardiovascular health and cognitive function, prospective studies addressing dietary patterns in diverse populations in the U.S. are limited (Harmon et al., 2015).

In an attempt to increase evidence of dietary patterns as modifiable risk factors for cognitive decline, we investigated the relationship between dietary patterns, vascular function, and cognition, in AAs and CCs with a parental history of AD. The goal of this project was to evaluate the influence of the Southern dietary pattern, as well as the prudent dietary pattern, on vascular risk factors and cognitive function in AA and CC participants at high risk for AD.

Methods

Study Sample: Sixty-six subjects (M = 58.7 ± 6.4 years), who are enrolled in an ongoing NIH funded study and who have a parental history of AD took part in this prospective pilot study. Subjects participating in the larger NIH funded study were obtained through various community events, conferences, and the Emory Alzheimer's Disease Research Center (ADRC) database. Individuals who expressed interest and met the inclusion criteria were enrolled in the ASCEND (Association between Cardiovascular Risk and Preclinical Alzheimer's Disease Pathology) study (PI: Wharton). Subjects enrolled in ASCEND received vascular and cognitive assessments under the ASCEND IRB approved protocol. Independent funding for the present dietary pattern pilot sub study was obtained through Emory University's Scholarly Inquiry Research Grant for undergraduate students (PI: Nutaitis). Subjects, who elected to participate in the sub study, completed the food frequency questionnaire and were given a $15.00 Amazon electronic gift card for reimbursement for their time completing the survey.

Dietary Pattern Assessment: Diet was assessed via the Jackson Heart Study's shortened version of the Lower Mississippi Delta Nutrition Intervention Research Initiative Food Frequency Questionnaire (Carithers et al., 2009). This questionnaire consists of 160 questions and takes approximately 20 minutes to complete. Participants completed the survey at home via a secure individual link through REDCap, an online HIPAA approved database used to store and protect research data. Participants responded by scaled score, how frequently and at what quantity each food and drink item was consumed. Carithers et al. compared the shortened version to both the full-length FFQ and a 24-hour food recall test, and demonstrated that this shortened version is a both a comprehensive and culturally sensitive instrument (Carithers et al., 2009).

Cardiovascular Risk Factor Assessment: To assess cardiovascular risk factors, participants underwent a one hour comprehensive assessment of peripheral vascular function including microvascular endothelial function (assessed by peripheral arterial tone signal, augmentation and reactive hyperemia index, and flow mediated dilation) and macrovascular endothelial function (assessed by pulse wave velocity). In addition, a comprehensive blood pressure (BP) assessment was obtained via 24-hour ambulatory BP monitoring. Clinically validated ambulatory BP monitors (Spacelabs Healthcare©) automatically recorded nocturnal BP once per hour, and recorded BP three times per hour during the day. Participants recorded their activity during daytime reads. We examined average systolic, average diastolic, and nocturnal dipping percent, all of which have been linked to cognition and AD (Tarumi et al., 2015).

Peripheral vascular function assessments included 1) EndoPAT, an instrument which measures changes in digit pulse waveforms known as peripheral arterial tone signal, to assess microvascular endothelial function (Axtell et al., 2010). 2) Augmentation index (AI), a ratio calculated from the BP waveform, as an indirect measure of arterial stiffness, and reactive hyperemia index (RHI) as a measure for endothelial function (M. Moerland, 2012). 3) Flow-mediated dilation (FMD), a validated method to induce reactive hyperemia, and 4) pulse wave velocity (PWV) to assess macrovascular endothelial function. FMD is a measure of endothelial dysfunction and represents the endothelium-dependent process facilitating the relaxation of an artery in response to increased sheer stress that produces a nitric oxide dependent response (Stout). FMD was measured by inflating a forearm pressure cuff to 250 mmHg for 4.5 minutes. At 60 and 90 seconds after cuff deflation, repeat imaging was obtained. Our laboratory's prior work has shown that these measures are related to cognition in individuals with a family history of AD (Wharton et al., 2014b). Pulse wave velocity (PWV) is a measure of arterial stiffness, or

the rate at which pressure waves move down a vessel. We utilized an AtCor SphymoCor Px tonometry system to measure PWV. A pressure transducer was placed on the skin at the arterial pulsation of the right common carotid and right radial arteries. A Millar micromanometer was in the tip of the probe. Using a generalized transfer function, the distance between these pressure points and the peripheral arterial waveforms, a central aortic pressure signal was derived, from which aortic augmentation index (AI) and PWV were determined (Gottdiener et al., 2004).

Neuropsychological testing: Cognitive function was evaluated by a 1.5 hour battery of thirteen neuropsychological tests in domains reportedly affected in early AD and particularly susceptible to the effects of hypertension (Asthana et al., 1999). The tests included: MOCA, Benson, Buschke, Trails A, Trails B, Digit Span Backwards, Mental Rotation (MRT), Flanker, Set Shifting, N Back, and MINT naming. These tests targeted specific AD related cognitive domains including: working memory and executive function (Trail-Making Test B (Dodrill, 1978; Stroop, 1935)), language (verbal fluency and MINT) (Spreen and Strauss, 1998)), verbal memory (list learning task (Buschke, 1973)), visuospatial ability (Mental Rotation Test (Vandenberg and Kuse, 1978)), and global cognition (MOCA(Nasreddine et al., 2005)). These cognitive tests have also been utilized in our previous studies, as executive function is very vulnerable to vascular dysfunction, and thus these tests were selected to evaluate aspects of cognition selectively impaired in early AD (Asthana et al., 1999) (Saxby et al., 2003).

Data Analysis: The primary study objective was to evaluate the potential relationship between cognitive function and foods characteristic of the Southern diet and foods characteristic of the prudent diet in AAs and CCs at high-risk for developing AD. Subject food frequency questionnaire responses, cognitive test results, and vascular risk factor data were stored in the secure REDCap online database. Researchers utilized IBM SPSS Statistics Version 22 to analyze

the data. All tests were two-tailed and used a significance level of 0.05. To test for group differences between AAs and CCs in demographics, vascular risk factors, and cognitive performance, we conducted independent-samples T-test for equality of means with equal variance assumed. Correlations between cognitive performance and foods were assessed using Pearson's r partial correlations controlling for education and age. Correlations were performed for those cognitive tests in which we found racial differences.

Results

Table 1 reports the demographic characteristics for 21 AA and 45 CC participants. Results show our participants are middle aged (M=58.66 years), mostly female (67.6%), and highly educated (83.8% received graduate or postgraduate education). While AAs and CCs did not differ on measures including age, education, exercise, smoking status, or self-reported depression, significant racial differences were present for gender and income, such that a larger percent of AA females than CC females participated in the study, and AAs reported significantly less income compared to CCs.

Tables 2 shows the results of the vascular risk assessment by race. AAs and CCs did not differ on any vascular measure including BP, arterial stiffness, and endothelial function. Table 3 shows cognitive performance by race. Our results demonstrate that racial differences on cognitive testing exist, such that CCs significantly outperformed AAs on the MOCA, MINT, and Trails B cognitive tests (all p values <0.05). In addition, results revealed a trend for CCs to outperform AAs on the Buschke Delay ($p= 0.073$).

Table 4 and Figure 1 show Pearson's r partial correlations between foods characteristic of the Southern diet and cognitive performance, by race. Pies, mashed potatoes, tea, and sugar drinks showed a significant relationship in AAs with cognitive performance ($p <0.05$), such that AAs performed worse on cognitive tests with consumption of these foods. In addition gravy ($p=0.06$) and added fat or oil while cooking ($p=0.06$) showed a negative relationship in AAs with cognitive performance. Furthermore, this table demonstrates that AAs were more negatively impacted than CCs by foods characteristic of the Southern diet. Our results do not reveal the

same negative relationship in CCs, as CCs who consumed mashed potatoes (p=0.01) and sugar drinks (p<0.1) performed better on cognitive assessments.

Table 5 and Figure 2 show Pearson's r partial correlations between foods characteristic of the more healthy prudent diet and cognitive performance, by race. Whole grain breads (p=0.04), baked fish (p=0.034), and 100% grape juice (p<0.01) showed a significantly positive relationship with cognitive performance. In addition, 100% OJ showed a trend (p<0.1) of better performance on cognitive assessment. The most pronounced relationship was seen with 100% grape juice, such that AAs consuming 100% grape juice performed significantly better on cognitive assessments (p<0.01). All other relationships between foods characteristic of the prudent diet and cognitive performance were seen only in CCs for the four cognitive tests that differed between AAs and CCs.

Discussion

To our knowledge, this is the only study to investigate a relationship between dietary patterns, cardiovascular risk factors, and a comprehensive battery of AD related cognitive tests in healthy, racially diverse middle-aged U.S. adults with a parental history of AD. Moreover, this is the first study to examine racial differences in dietary patterns as a possible preventative tool against cognitive decline in this high-risk population. Our results showed that racial differences on cognitive testing exist in individuals with a parental history of AD, such that CCs outperformed AAs in cognitive domains including global cognition, language retrieval, and executive function. However, these racial differences in cognitive performance could not be explained for by age, education, vascular risk factors, exercise, smoking, or depression in our healthy, middle-aged cohort. As we reported a relationship between cognition and nutrition, this link may be partially explained by dietary patterns specific to the Southern and prudent diets.

The Southern diet is characterized by foods such as fried chicken and fried fish, added fats, eggs, organ and processed meats, pies, and sugar-sweetened beverages(Shikany et al., 2015). In this pilot study of healthy, educated individuals our results show a significant relationship between the four cognitive tests that showed racial differences, and food groups including pies, mashed potatoes, tea, sugar drinks, gravy, and added fat/oil. These results are in line with current literature that suggests a dietary pattern high in gravy or butter is associated with poor cognition in older adults (Granic et al., 2016). Not only do our results demonstrate a relationship between cognition and these foods, which are characteristic of the Southern diet, but also our results show racial differences in dietary patterns such that AAs display a stronger alliance with the Southern diet than CCs in their reported food choices, which may explain some of the increased risk for AD in AAs.

Furthermore, when contrasting the Southern diet with the prudent diet, which is characterized by whole-wheat grains, fish, fruits, and vegetables, our results show a positive dietary influence on cognitive performance in CCs that was not as pronounced in AAs. While this discrepancy may be partially attributed to the small sample of AAs, a significant relationship was detected with 100% grape juice and cognitive performance in AAs. Within the cognitive domains that we found performance differences in CCs vs. AAs, results show a positive relationship between dark breads, whole wheat, rye, and pumpernickel bread, baked fish, 100% grape and 100% orange juice.

It is interesting to note the significant influence of beverages on cognitive function. It is possible that individuals may be more consistent with their beverage choices, i.e. coffee or orange juice each morning, versus alternating morning protein from food, and thus it is possible that beverages show a greater impact on cognitive function than food choices that tend to exhibit a greater variance. This being said, we were still able to see a significant relationship between both foods of the Southern diet contributing to poorer cognitive performance and foods of the prudent diet benefiting cognitive performance. Of note, because of the small number of participants in this pilot study, particularly AAs, it is possible that our results would be magnified in a larger sample size.

When analyzing the influence of dietary patterns on cognitive performance, it is difficult to assess whether the Southern diet or prudent diet plays a larger role in contributing to cognitive performance. It is likely that the differences observed in the MOCA, Buschke delay, MINT, and Trails B between racial groups could be accounted for by both a Southern diet negatively influencing cognitive performance and a prudent diet positively influencing cognitive performance.

The racial discrepancy in AD incidence and prevalence may be the result of a combination of biological, psychological, and socioeconomic influences, however these factors may not be as easily modifiable as diet modification (Chin et al., 2011; Steenland et al., 2015). AD is most likely a multifactorial disorder influenced by interactions between numerous genes that are not yet fully understood (Chin et al., 2011); this being said, current research has suggested that factors other than genetics account for a large percentage of the increased risk observed in AAs (Chin et al., 2011; Norton et al., 2014; Tang et al., 1998). While researchers have accounted for some of the increased risk in AAs by vascular disease prevalence (Chin et al., 2011), our results did not reveal significant differences in any vascular risk factor by race, and thus vascular factors do not explain our present disparities in cognitive task performance.

Researchers have stressed the need for advancements in preventative and treatment strategies specific to race in an attempt to combat the higher incidence of AD in AAs (Froehlich et al., 2001). Since our healthy middle-aged AAs and CCs report different dietary patterns that were related to cognitive performance, it is possible that dietary patterns may be contributing to early cognitive decline in our AA subjects, or preservation of cognitive functioning in our CCs subjects. This finding is important, as the current literature suggests while even though late-life positive dietary patterns may result in notable health related improvements (Bardach et al., 2016; Shakersain et al., 2016), mid life is the optimal time to incorporate these changes, before the irreversible AD cascade begins(Barage and Sonawane, 2015). According to Laitinen et al., who followed individuals for an average of 21 years and compared the seven ideal health behaviors defined by the American Heart Association, including diet, to cardiovascular health metrics, individuals who improved their ideal health behaviors performed comparably on cardiovascular health metrics relative to those that had persistently positive health behaviors (Laitinen et al.,

2015). Thus, it is possible that modifying one's dietary pattern favorably later in life will likely still have beneficial results on both cardiovascular and cognitive health. While the potential monetary and cultural ramifications of changes in overall dietary patterns are unknown, we postulate that diet holds promise as an AD related modifiable risk factor (Baumgart et al., 2015). Furthermore, our results may have underestimated the impact of diet, as the sample size in this pilot project is small and the cohort is overall very healthy. It is possible that dietary patterns may have an even more pronounced impact in individuals with preexisting health related complications.

Our data are of particular importance in light of current racial U.S. population projections. By 2060, not only will the population aged 65 and older more than double, but the racial demographics are estimated to shift (Steenland et al., 2015). The AA population is expected to increase from 14% to 18% and the Hispanic population is expected to increase from 18% to 29%(Steenland et al., 2015). Both of these populations have a higher prevalence for AD than CCs (Steenland et al., 2015), and thus it is imperative to target modifiable risk factors unique to these groups that may be contributing to their increased risk. If, in the absence of a disease-modifying treatment, our population projections proceed as predicted, it is of utmost importance for researchers to advance knowledge on mechanisms by which developing dementia can be reduced (Baumgart et al., 2015).

Notable strengths of this study include a comprehensive battery of neuropsychology testing, complete peripheral vascular measures, and a racially diverse cohort. Furthermore, our study sample includes individuals who have a parent with AD, either autopsy-confirmed or probable AD as defined by NINDS-ADRDA criteria. AD diagnosis was verified using parental medical records and the Dementia Questionnaire (Kawas et al., 1994). Participants had at least one

biological parent diagnosed with late onset AD with no known autosomal mutations. Furthermore, independent of race, researchers have linked depression to increased AD risk (Diniz et al., 2013). Thus obtaining data on depression and comparing prevalence between racial groups, eliminated another variable that may have been contributing to the racial differences on cognitive performance. The peripheral vascular measures were selected based on prior evidence showing that these tests were capable of detecting preclinical alterations in peripheral function (Wharton et al., 2014a).

One limitation of the study is that we were not able to assess the duration of participants' dietary patterns. As discussed, we selected this FFQ because it was both racially and geographically sensitive (Carithers et al., 2009) to our cohort. However, this survey did not ask the participant to report how long they had practiced the reported diet. Future FFQs should include dietary pattern duration because individuals may experience dietary pattern changes across their lifespan (Montero et al., 2000). For example, Montero et al. reported that 48.8% of women in a socio-economically disadvantaged class in Spain had changed their dietary patterns due to migration or marriage (Montero et al., 2000). Chapman and Ogden suggest other reasons for dietary pattern change include accumulation of evidence against, or in support of specific foods as a result of a health concern(Chapman and Ogden, 2009). Future research should investigate current trends, incidence, and prevalence surrounding changes in dietary patterns.

In summary, our results stress the need for further research investigating the influence of racial differences in dietary patterns on cognition in individuals at risk for AD. Because AAs have an increased incidence and prevalence of AD (Dilworth-Anderson et al., 2008; Steenland et al., 2015), extensive investigation of modifiable risk factors that may target high-risk groups is essential. Our results show that AAs who consume foods characteristic of the Southern diet

performed worse on cognitive assessments than CCs, and thus altering dietary patterns characteristic of the Southern diet may provide one intervention by which our healthy middle-aged participants could combat their increased risk for AD.

Tables and Figures

Table 1: Participant Demographics

	AA (N=21)	CC (N=45)
Age	57.76 ±7.69	58.98 ± 5.78
Gender (% Female)	85.7% *	57.8% *
College graduate or higher level of education	85.7%	84.5%
Income**		
Income $39,000 or less	38.1%	11.1%
Income $40,000-$79,000	38.1%	24.4%
Income $80,000 or more	23.8%	64.4%
Mean days/week performing cardiovascular exercise	1.5	1.71
Report having smoked	19%	26.7%
Reported smoking in last month	4.8%	8.9%
Reported feeling down, depressed, or hopeless several days of the week or more	28.6%	17.7%

*$P < 0.05$
** $P < 0.001$

Table 2: Cardiovascular Data for All Participants

	AA	CC
Systolic (mmHg)	129.79 ± 12.33	125.52 ±12.75
Diastolic (mmHg)	77 ± 5.39	77.55 ± 9.73
% Nocturnal Dipping	6.48 ± 6.85	7.29 ± 6.31
FMD 60 s.	6.11 ± 6.06	5.90 ± 4.19
FMD 90 s.	4.27 ± 6.91	4.47 ± 3.83
EndoPat RHI	2.40 ± 0.80	2.26 ± 0.75
EndoPat AI	28.81 ± 19.22	22.09 ± 17.43

RHI=reactive hyperemia index

AI= augmentation index

Table 3: Cognitive Data for All Participants

	AA	CC
MOCA	25.39 ± 2.40 *	27.0 ± 2.33*
Benson Delay	11.89 ± 3.35	10.78 ± 2.78
Buschke Delay	5.37 ± 3.33 †	6.88 ± 2.80 †
MINT	29.53 ± 2.09 *	30.7 ± 1.98*
MRT	17.37 ± 3.29	17.8 ± 5.50
Trails B.	120.32 ± 102.45*	81.9 ± 42.56*
Backwards Digit Span	4.11 ± 1.20	4.78 ± 1.75
Backwards Digit Span Accuracy	5.47 ± 1.87	6.63 ± 2.94

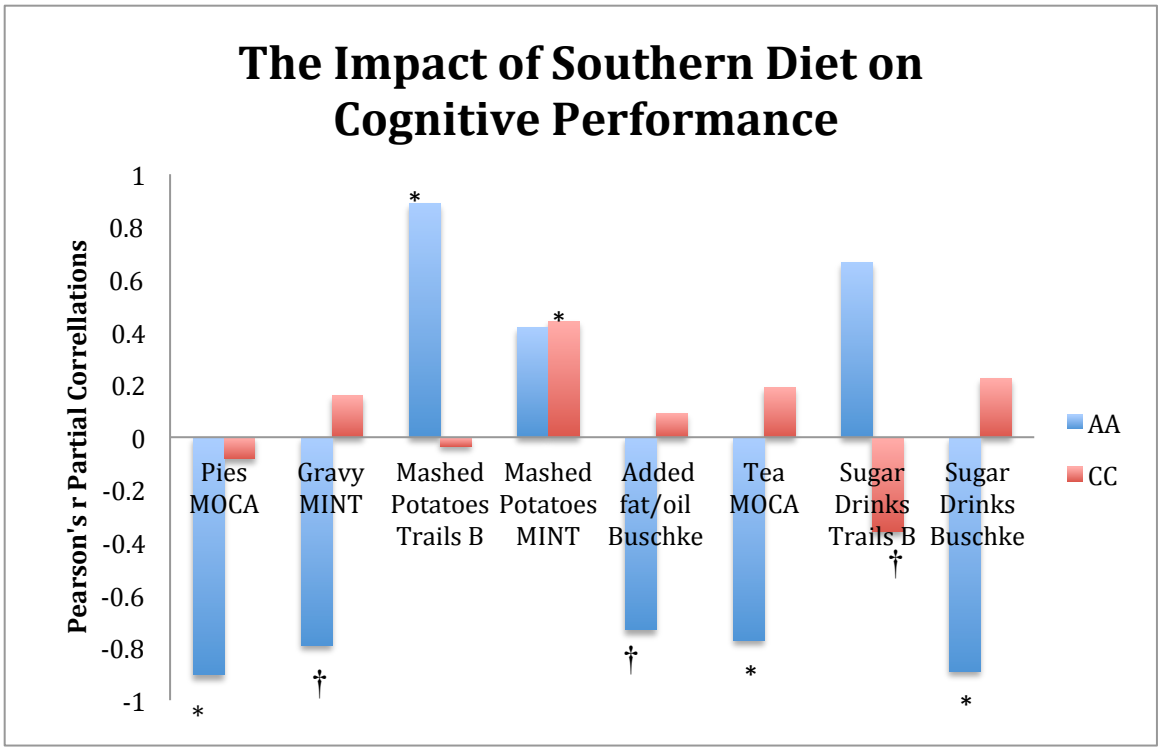
†$P<0.1$
*$P < 0.05$

Table 4: Pearson's r Correlations between Cognition and Southern Diet by Ethnicity for individuals who completed FFQ. All correlations controlled for age and education.

	Pies	Gravy	Mash potatoes	Added fat/oil while cooking	Tea	Sugar Drinks
MOCA						
AA	-.904 *	-.056	.693	.096	-.772*	.529
CC	-.082	.085	.1	.089	.189	.024
Buschke delay						
AA	.534	-.483	-.662	-.733†	-.045	-.890 *
CC	-.144	-.298	-.128	.092	.129	.224
MINT						
AA	-.295	-.791†	.417	.112	-.686	.419
CC	.224	.157	.441 *	.062	.128	.059
Trails B						
AA	-.431	.047	.889 *	.551	.179	.664
CC	.192	-.111	-.034	.057	-.160	-.359†

†$P<0.1$
*$P < 0.05$

Figure 1: Pearson's r Correlations between Cognition and Southern Diet by Ethnicity for individuals who completed FFQ. All correlations controlled for age and education.



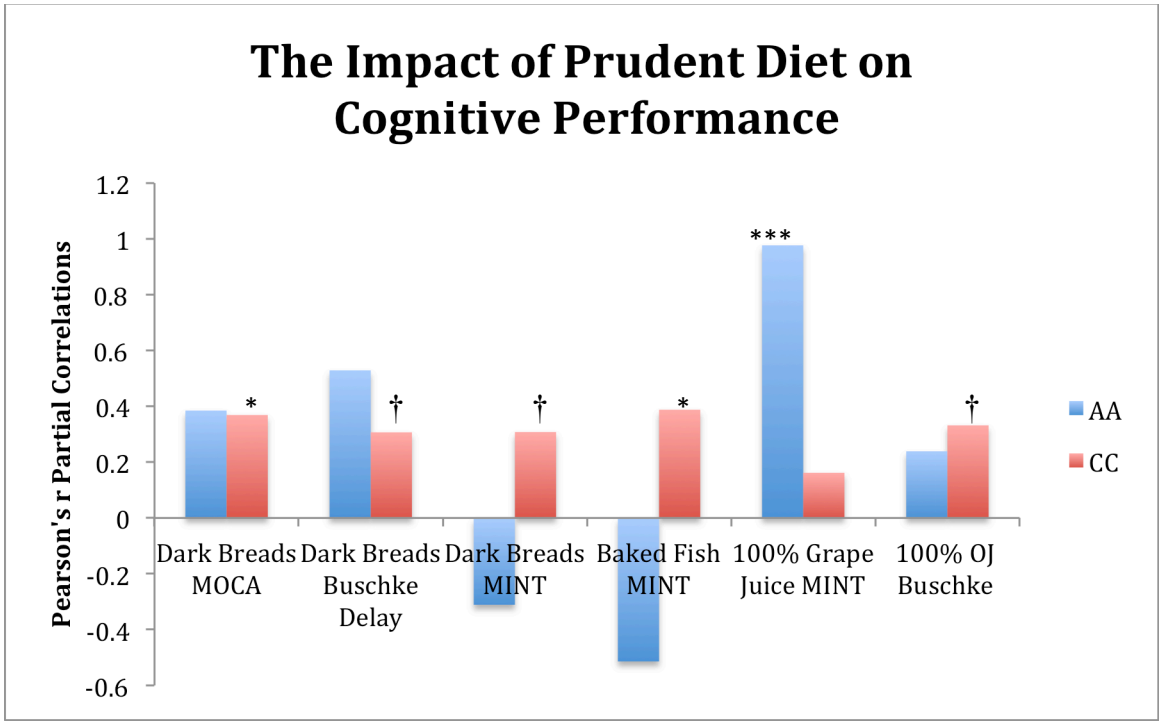
Graph 1. This graph represents foods characteristic of the Southern diet and their relationship to cognitive performance in both AAs and CCs. The foods and cognitive tests in which a relationship was present are listed on the x-axis. A negative Pearson's R partial correlation indicates a negative relationship with the cognitive test. However, for Trails B an increasing time is indicative of poorer performance on the cognitive test. Thus, a positive Pearson's r partial correlation indicates that the person is taking a longer time to complete the task and thus has poorer cognitive function.

Table 5: Pearson's r Correlations between Cognition and Prudent Diet by Ethnicity for individuals who completed FFQ. All correlations controlled for age and education.

	Dark Breads	Baked Fish	100% Grape juice	100% Orange Juice
MOCA				
AA	.385	-.520	.098	.264
CC	.369 *	-.155	-.057	.023
Buschke Delay				
AA	.529	.442	.201	.239
CC	.307 †	-.075	-.198	.332 †
MINT				
AA	-.311	-.514	.977 ***	.054
CC	.308 †	.388 *	.162	.032
Trails B				
AA	-.497	-.023	.145	.192
CC	-.299	.071	-.264	-.211

†$P<0.1$
*$P < 0.05$

Figure 2: Pearson's r Correlations between Cognition and Prudent Diet by Ethnicity for individuals who completed FFQ. All correlations controlled for age and education.



Graph 2: This graph represents foods characteristic of the prudent diet and their relationship to cognitive performance in both AAs and CCs. The foods and cognitive tests in which a relationship was present are listed on the x-axis.

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