

**Structural Analysis of the Relationship of Food Insufficiency to Disease Risk and
Outcomes Among Adults from NHANES III**

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Background

Food security is defined as “access by all people at all times to enough food for an active, healthy life.” Food insecurity exists whenever the availability of nutritionally adequate and safe foods is limited or there is uncertainty as to the ability to acquire acceptable foods in socially acceptable ways (1). Food insufficiency is a related term seen frequently in the literature. This term refers to an inadequate amount of food intake due to a lack of resources (2) as opposed to the broader concept of food insecurity, which includes both psychological and physiological dimensions.

Research throughout the latter part of the 1980’s clarified the understanding of food insecurity and hunger in industrialized nations as managed processes whereby adults first begin to worry that food supplies will run out before there are resources to obtain more. Adjustments in the quality of the food supply may accompany this early stage of food insecurity in an effort to extend the food supply without having to reduce the quantity of food eaten. Next, adults may begin to reduce the quantity of their own food intake in addition to the quality in order to spare food supplies for the children in the family. Finally, food supplies may be limited to the point that children in the household have to reduce the amount of food consumed and adults may be going a whole day without eating (3-5).

Understanding the processes by which households attempt to maintain food security led Campbell (6) to propose a conceptual framework for risk factors and consequences of food insecurity. Her paper raised the question of whether food insecurity is the nutritional outcome of economic risk factors or a predictor variable for more definitive/measurable outcomes such as poor health. The conclusion of the paper, which was reflected in Campbell’s conceptual framework, was that food insecurity could be both an outcome of risk factors as well as a predictor of other more definitive outcomes. Her conceptual model (Appendix, Figure 1) indicates that risk factors for poor diet and secondary malnutrition lead to effects on diet as well as nutritional status. These outcomes can be measured by things such as food intake patterns, food insecurity and biochemical markers. In addition, these outcomes in turn become predictors of other outcomes such as physical health and quality of life. This model indicates that food insecurity can affect health outcomes either directly or indirectly through nutritional status. Since her conceptual model was developed, research has provided evidence for the relationship between food insecurity and each construct proposed by Campbell, but has not attempted to demonstrate inter-relationships among all model components.

The first part of Campbell’s theoretical model consisted of factors that may influence diet and food insecurity. She hypothesized that risk factors for food insecurity included anything that limited household resources, or the proportion of those resources available for food. In addition, limited employment opportunities, low wage and benefit scales, limited social assistance benefits, high housing and utility costs, health care costs, high taxes and childcare, and unexpected expenses (i.e. emergencies) would be considered risk factors for food insecurity. Indeed, studies have shown that factors related to socio-economic status are associated with being food insecure/insufficient. Factors found to have a positive relationship to food insecurity/insufficiency are single parenthood, lack of

savings, large household size, unexpected expenses, inadequate food stamp allotment (7-10), as well as low educational attainment (9;11;12).

Nutrient and food intake are measurable components of the second construct in Campbell's model called "diet". Studies have attempted to elucidate the effects of food insecurity/insufficiency on diet. In general, studies using both primary and secondary data have shown nutrient intake differences among adults from households reporting either food insecurity or food insufficiency when compared to their food sufficient counterparts. Nutrient intakes among food insecure/insufficient adults have been reported to be lower than food sufficient adults for vitamins A, C, B₆, E, and folate and for minerals such as calcium, potassium, magnesium, iron, and zinc (9;13-16), as well as dietary components such as fiber or fruits and vegetables (9;15;17).

It would follow that if there is a relationship between food insecurity/insufficiency and nutritional imbalances, there may be relationships between food insufficiency and nutritionally related disease risk factors that mediate poor health outcomes, a construct called "nutritional status" in Campbell's conceptual model. This has been the subject of two recent studies. Dixon, Winkleby, and Radimer (16) reported that younger adults (aged 20-59) from food insufficient households had lower concentrations of serum total cholesterol, vitamin A, alpha-carotene, beta-cryptoxanthin, and lutein/zeaxanthin than the younger food sufficient adults who participated in the Third National Health and Nutrition Examination Survey (NHANES III). Likewise, older adults (60 years and older) from food insufficient households had lower concentrations of serum high-density lipoprotein cholesterol, albumin, vitamin A, beta-cryptoxanthin, and vitamin E compared to their counterparts in food sufficient households. Townsend, Peerson, Love, Achterberg, and Murphy (18) reported that food insecurity was related to overweight status among women, but not men participating in the 1994-96 CSFII. In addition, 41% of the women who were classified as mildly food insecure based on answers to the household food insufficiency question and 52% of the women classified as moderately food insecure were overweight compared to 34% of the food secure women. Logistic regression showed that food insecurity was a significant predictor of obesity among the women even after adjusting for potential confounding demographic and lifestyle factors such as education level and television viewing. These studies provide evidence that food insecurity/insufficiency could be a predictor variable for poor health outcomes through its effect on nutrition-related disease risk factors.

The final construct of Campbell's model consists of health outcomes (consequences of a poor nutritional state). This construct could be composed of various "factors" such as physical, social or mental health, and quality of life. Campbell proposed that food insecurity might influence this construct indirectly through its effects on diet as discussed above, or it may have a direct influence on health outcomes. Recently studies have begun to investigate more direct relationships of food insecurity to health outcomes. Nelson, Brown, and Lurie (19) investigated the relationship of diabetic care and episodes of hypoglycemia to food security. Thirty-one percent of the 103 patients reporting episodes of hypoglycemia attributed it to the inability to afford food. Eight percent of the 170 diabetic patients interviewed reported that they decreased or stopped taking their

insulin because of food insecurity. Likewise, Nelson, Cunningham, Andersen, Harrison, and Gelberg (20) reported that adult diabetics from NHANES III who were food insufficient were more likely to report fair or poor health status, and reported more physician contacts than those who were food sufficient. Lee and Frongillo (21) reported that elderly participating in NHANES III and the Nutrition Survey of the Elderly in New York State were 2.33 times more likely to report fair/poor health status and had higher nutritional risk compared to their food sufficient counterparts. Financial difficulty acquiring food was reported to be associated with higher levels of depression, poorer quality of life and physical performance among white women, more medical conditions among black women, and anemia in disabled women participating in the Women's Health and Aging Study (22). When taken together, these studies suggest a relationship between food insufficiency and health outcomes both indirectly through nutrition status and directly as noted above.

Campbell's model also included a construct referred to as risk factors for secondary malnutrition. This construct could be composed of factors such as disease states or behaviors that alter nutrient needs, intakes, or metabolism. These factors could affect health outcomes indirectly through their effect on diet quality or indirectly through nutritional status. For example higher levels of physical activity have been associated with better diet quality (23) as well as higher levels of serum high-density lipoprotein (HDL) cholesterol (24;25). These factors must also be considered when assessing health outcomes associated with diet or food security.

Research to date has provided evidence for the relationship between food insecurity and each single construct proposed by Campbell, but has not investigated inter-relationships among all model components simultaneously. By using a comprehensive analytical method such as structural equation modeling, researchers can evaluate theoretical models, such as the one proposed by Campbell that include constructs (known as latent constructs) that are measured by multiple variables or measurement instruments (known as indicator or observed variables). National datasets such as NHANES III that included data on food insufficiency, dietary intake measurements, biochemical, clinical, and anthropometric measurements, socio-economic and demographic indicators and lifestyle behaviors as well as measures of various disease states can provide data to explore the direct and indirect relationships among food security, diet, nutritional status, disease risks, and health outcomes.

Heart disease was the leading cause of death in the U.S. in 1998 (26). Death rates for heart disease were higher than the national average for 11 out of 13 southern states between 1996-1998 (27). This combined with the fact that rates for food insecurity in eight of these 13 southern states was also higher than the national average (28) indicates the need to investigate the inter-relationships between food insecurity/insufficiency, disease risk, and health outcomes. Again, NHANES III contains information on many of the factors known to impact cardiovascular (CVD) disease as well as food insufficiency information. Since many of those reporting food insufficiency in NHANES III were of lower income levels that qualify for federal assistance programs (11), using this data set to study these relationships may help policy makers set priorities for food and nutrition

assistance programs as well as health care initiatives. Therefore the objective of this research study was to develop and test a model for the relationships among food insufficiency, diet quality, CVD risks, and CVD outcomes among adults residing in the southern region and participating in NHANES III. The model to be developed and tested would be derived from Campbell's conceptual model of the risk factors for and consequences of food insecurity (Figure 2, Appendix). In addition, the model developed and tested for the southern region would also be applied to the remaining regions in NHANES III to determine if there were regional differences in the relationships among the model constructs.

Methods

Sample

The sample for this study was drawn from the NHANES III dataset. Adults who completed both the family interview component and the medical examination component of NHANES III were chosen for this study. Pregnant and lactating women, cases with "lost" 24-hour recalls, cases determined to be unreliable by the interviewer, and extreme energy intake outliers (for example, reported calorie intakes less than 400 or greater than 5500) were removed from the data set leaving an initial sample of 15,770 cases. Once all variables were identified and/or re-coded in the form appropriate for the data analyses, one adult from each household was selected for inclusion in the final sample. The criterion used to determine which adult to select from a household with more than one adult interviewed was that the sample adult should have no missing data. If all the adults in the household had complete data, the first one in the household was chosen as a sample person. This resulted in a sample of 10,772. Then all adults identified as residing in the southern region of the U.S. were separated from the dataset to create a "southern" dataset. The sample size of this dataset was 4,675. The final step in sample selection was to randomly split the southern dataset into two datasets. One dataset was used to test the hypothesized measurement and structural model (model-fitting sample). Sample size for the model-fitting (MF) sample was 2,328. The other dataset was used for cross-validation or replication purposes (model-testing sample). Sample size for the model-testing (MT) sample was 2,347.

Variables

Three primary sets of latent constructs were identified for this study. Table 1 (Appendix) shows the study variables used to construct the hypothesized model (Figure 2, Appendix). The first latent construct was health behaviors and was indicated by the variables of smoking status (yes =1, no=0) computed from three questions related to the number of cigarettes, cigars, or pipes smoked over the previous five days, alcohol intake frequency (number days over previous year), alcohol intake quantity (usual number of drinks on a drinking day), physical activity frequency (average number of occasions of 1-13 different types of physical activity), average physical activity intensity rating (measured in metabolic equivalent tasks – METs), and change in diet due to CVD health reasons (yes=1, no=0). This latent construct corresponds to the construct of "risk factors for secondary malnutrition" in Campbell's model. The second latent construct was CVD risks which was indicated by the variables of serum cholesterol (serchol), serum HDL cholesterol (hdl), serum triglycerides (trigly), systolic blood pressure (systolic), diastolic

blood pressure (diastol), body mass index (bmi), waist-to-hip ratio (waisthip), cardiovascular medications (meds), serum vitamin E (svite), and serum folate (sfolate). These variables were taken directly from the NHANES III dataset. This latent construct would most closely correspond to the construct of nutritional status in Campbell's model, but from a disease risk perspective. The third latent variable was CVD outcome that was indicated by the dichotomous variables hypertension (htnx2, yes=1, no=0), heart attack (mi, yes=1, no=0), congestive heart failure (chf, yes=1, no=0) and stroke (yes=1, no=0). These four variables were self-reported by the respondent. This latent construct most closely corresponds to Campbell's construct of "consequences of a poor nutritional state."

A dichotomous variable for food insufficiency status was computed from the NHANES III exam questions related to the number of days the respondent did not eat or skipped meals due to resource constraints. Respondents were classified as food insufficient (coded 1) if they had gone without food or had gone without food due to reasons other than no money. This variable was used as a predictor variable of diet quality and CVD outcomes to be consistent with Campbell's model. Diet quality was indicated by computing a modified diet quality index based on the methods described by Patterson, Haines, and Popkin (29). This index is a score derived from percent calories from total fat, percent calories from saturated fat, dietary cholesterol, dietary sodium, dietary calcium, and dietary protein. Table 2 (Appendix) shows the procedure used to compute the index scores for each component of the index. The index was computed by assigning a score to each variable in the index then summing the scores to yield the modified diet quality index. This index could range from zero to twelve with lower scores indicating a more healthful diet. Therefore, this variable was actually a measure of an "unhealthy" diet. It was used as a predictor for CVD risk corresponding to Campbell's model relating the construct of "diet" to "nutritional status." Likewise, the latent construct health behaviors was hypothesized to have a relationship with diet quality to correspond with Campbell's construct of "risk factors for secondary malnutrition" influencing the construct of "diet."

In addition, variables noted to be associated with food insufficiency and diet quality in the literature were included in the model as predictors of food insufficiency status and an unhealthy diet (as measured by the modified diet quality index). These variables were poverty income ratio, education level of the respondent, gender of the respondent, and race/ethnicity of the respondent (white, coded 1 and non-white, coded 0). This component of the model would most closely correspond to Campbell's component of "risk factors for malnutrition."

Data Analysis

Data was extracted from NHANES III using SAS 6.0 (30). Data recoding was done using SPSS 10.0 (31). Analysis of Moment Structures (AMOS) 4.0 (32) software was used to conduct structural equation modeling (SEM).

Data analysis proceeded in two phases. The first phase was testing of the measurement model. This phase involved (1) determining relationships between the indicator variables and the latent constructs, (2) determining correlations between pairs of indicator

variables, and (3) determining correlations among the errors associated with the indicator variables. The second phase was testing the structural model using structural equation modeling. Evaluation of the measurement and structural models involved the use of “fit indices.” The theoretical model implies a specific covariance matrix. Applying the structural model to the actual data results in an “observed” covariance matrix. A model is said to “fit” the observed data to the extent that the covariance matrix it implies is equal to the observed covariance matrix that is produced from the data. If the elements of the residual matrix are equal to zero, then the model is said to “fit” the data perfectly. Three goodness of fit indices were used to evaluate the overall fit of the measurement and structural models in this study. The first was the Chi-square statistic. This statistic is a measure of whether the residual covariances obtained by comparing the observed and predicted values differ from 0. If the Chi-square is small with regards to the degrees of freedom, the statistic is not significant and the model is considered to be a good fit. However, the Chi-square statistic is sensitive to sample size, which was large in this study. Therefore, two additional goodness of fit criteria were used. These were the goodness of fit index (GFI) and the adjusted goodness of fit index (AGFI). The goodness of fit index is analogous to squared multiple correlation. It indicates the proportion of the observed covariances explained by the model-implied covariances. Values in this index theoretically range from 0 (poor fit) to 1 (perfect fit). The AGFI adjusts the goodness of fit index for model complexity. The rationale behind this index is that more complex models (those with more parameters) tend to fit the data better than simpler models. The AGFI takes this into consideration and adjusts the value of the GFI downward as the number of parameters increases. This index also assumes a value between 0 (poor fit) and 1 (perfect fit) (33). Both these indices are more independent of sample size than the chi-square statistic and relatively robust against departures from normality. Using these three criteria, the model was said to fit the data if the Chi-square statistic was not significant ($p > 0.05$), or the GFI was high (≥ 0.90) and the AGFI was high (preferably ≥ 0.90). We also took into account the population error of approximation (expressed as root mean square error of approximation, RMSEA), a measure of the degree to which the model holds in larger samples. Values up to 0.05 indicated a close fit in larger populations and values up to 0.08 represent reasonable errors of approximation in the population (34;35).

Preliminary Results

Description of Model-Fitting and Model-Testing Samples

Independent T-tests were used to determine any differences between continuous variables of the MF and the MT samples. The results are presented in Table 3 as means and standard deviations (SD). There were no significant differences between the two samples on any of these variables. The mean age of the MF sample was 48.5 years while the mean age of the MT sample was 47.5 years. The education level of the MF sample was 10.5 years while the education level of the MT sample was 10.6 years. The standard deviations of the serum triglycerides, alcohol frequency, and alcohol quantity variables were quite high. The data were checked for coding errors and found to have none. Triglycerides ranged from 23 mg/dl to 1495 mg/dl. Number of days drank alcohol

ranged from 0 to 365 while the number of drinks on a drinking day ranged from 0 to 90. Therefore the standard deviations of these variables were most likely due to true outliers in the NHANES III dataset. Quintana and Maxwell (35) note that outliers in the data may distort relationships between variables, however they advise against removing outliers unless they are known to be coding errors or situations in which the score is known to be inaccurate. Therefore the researchers chose to leave these outliers in the dataset.

Table 3 Comparisons of Continuous Indicator Variables in Measurement and Model-Fitting Samples

Variable	<u>Model-Fitting Sample</u> n=2,283		<u>Model-Testing Sample</u> n=2,347	
	Mean	SD	Mean	SD
Age (years)	48.48	20.06	47.48	19.56
Poverty Income Ratio	2.25	1.59	2.27	1.58
Education Level (years)	10.48	4.05	10.61	4.02
Systolic Blood Pressure (mmHg)	127.90	20.14	127.34	19.75
Diastolic Blood Pressure (mmHg)	75.71	11.23	75.48	10.48
Waist-to-Hip Ratio	0.93	0.09	0.93	0.09
Serum Folate (ng/ml)	5.98	4.69	5.96	5.40
Serum Vitamin E (µg/dl)	1087.80	429.79	1094.34	445.39
Serum Cholesterol (mg/dl)	204.79	43.47	205.11	43.31
Serum Triglycerides (mg/dl)	141.34	105.27	141.42	101.98
Alcohol Frequency (no. drinks/yr)	45.82	87.53	44.29	84.38
Alcohol Quantity (no. drinks/drinking day)	1.71	2.96	1.65	3.39
Physical Activity Frequency (no. days past month)	1.48	1.95	1.57	2.16
Physical Activity Intensity Rating (METs)	0.69	0.70	0.69	0.70
Modified Diet Quality Index	5.87	2.58	5.97	2.56

Chi-square was used to determine if there were any differences between the dichotomous variables of the two samples. Description of the dichotomous variables and the results of the chi-square tests are presented in Table 4. There were no statistically significant differences in the demographic characteristics of the MF and MT samples. However, the proportion of adults who reported that they had had a heart attack was statistically significantly higher in the MF sample compared to the MT sample. The difference between the two groups, however was small with 6.1% of the MF sample reporting a history of heart attack compared to 4.0% of the MT sample.

Table 4 Characteristics of Dichotomous Indicator Variables in Measurement Model and Model-Fitting Samples

Variable (abbreviation)	Model-Fitting Sample n=2,328		Model-Testing Sample n=2,347	
	n	%	n	%
<u>Gender (gender)</u>				
Male	1293	55.5	1299	55.3
Female	1035	44.5	1048	44.7
<u>Race-Ethnicity (race2)</u>				
White	919	39.5	869	37.0
Non-white	1409	60.5	1478	63.0
<u>Food Sufficient (FI)</u>				
Yes	2172	93.3	2203	93.9
No	156	6.7	144	6.1
<u>Smoke (smoking)</u>				
Yes	679	29.2	741	31.6
No	1649	70.8	1606	68.4
<u>Changed Diet for CVD (chgdiet2)</u>				
Yes	1270	54.6	1269	54.1
No	1058	45.4	1078	45.9
<u>Self-reported CHF (chf)</u>				
Yes	105	4.5	103	4.4
No	2223	95.5	2244	95.6
<u>Self-reported Stroke (stroke)</u>				
Yes	60	2.6	64	2.7
No	2268	97.4	2283	97.3
<u>Self-reported Hypertension (htnx2)</u>				
Yes	524	22.5	515	21.9
No	1804	77.5	1832	78.1
<u>*Self-reported Heart Attack (mi)</u>				
Yes	141	6.1	93	4.0
No	2187	93.9	2254	96.0

*p<0.05 by Chi-square

Measurement Model

The MF sample from the southern region of NHANES III was used to test the measurement model for each latent construct in the hypothesized model in order to construct the structural model. Fit of each measurement model was indicated by a GFI \geq 0.90, an AGFI \geq 0.90, and a RMSEA $<$ 0.07.

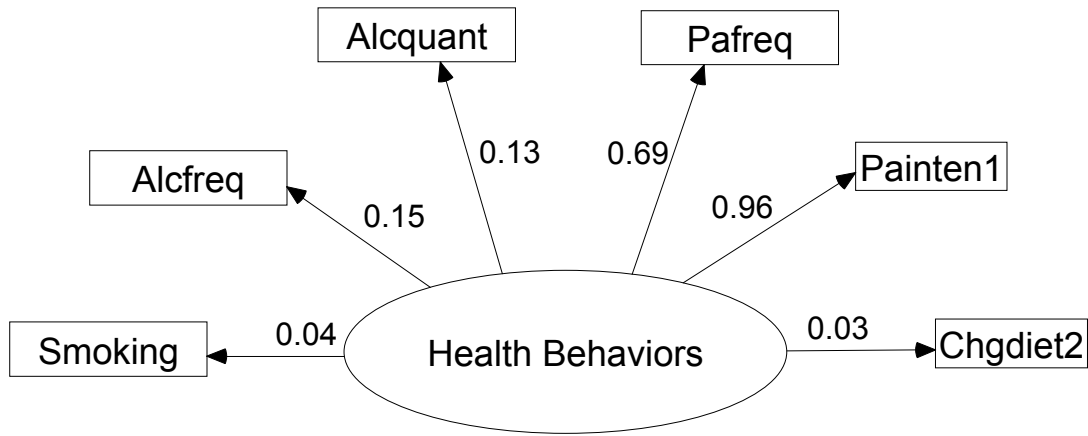
There was a significant relationship between the latent construct health behaviors and each of the indicator variables except “change diet” and “smoking.” Table 5 shows standardized regression weights for the indicator variables in the measurement model. The Chi-square was quite large ($\chi^2=622.657$, $df=2$, $p<0.001$). However, the overall fit statistics indicated a fair fit with a GFI of 0.909, and an AGFI of 0.789. The RMSEA of

0.171 indicated greater than reasonable errors of approximation in the population. The model for health behaviors is shown in Figure 3.

Table 5 Standardized Regression Weights for the Health Behaviors Latent Construct

Indicator Variable	Standardized Regression Weight
Smoking	0.042
*Alcohol Frequency	0.148
*Alcohol Quantity	0.131
*Physical Activity Frequency	0.691
*Physical Activity Intensity	0.960
Change in Diet	0.025

* $p \leq 0.05$



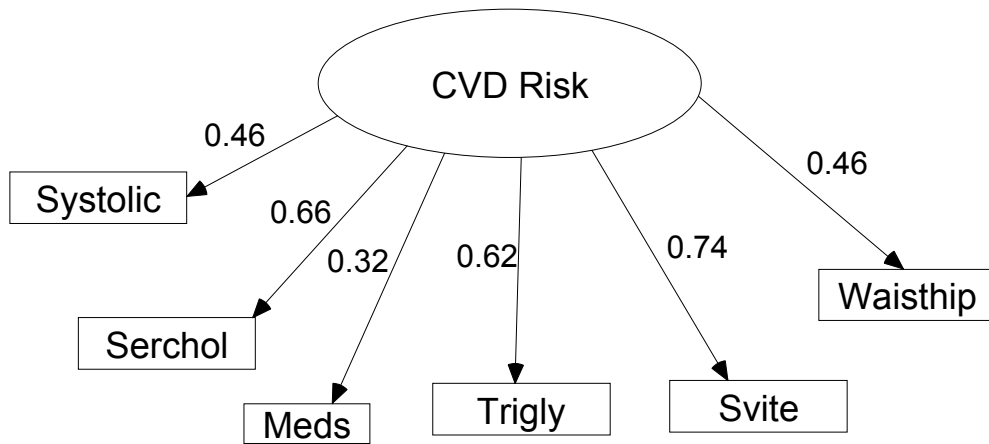
Alfreq=Number of Days Drank Alcohol Last Year, Alcquant=Number of Drinks on a Drinking Day,
Pafreq=Average Number of Occasions of 1-13 Physical Activities,
Chgdiet2=Change diet for CVD

There was a significant relationship between the latent construct cardiovascular disease risks and each of the indicator variables in the model. Table 6 shows the standardized regression weights for these indicator variables. However, the measurement model for the latent variable cardiovascular disease risks was not confirmed (GFI = 0.796, AGFI = 0.679, RMSEA = 0.184). The indicator variables that explained 30 percent or less of the variance were removed from the measurement model. When these indicator variables were removed from the model, the paths remained significant for the other indicator variables and the overall model fit improved (χ^2 decreased from 2796.624 to 617.794, df decreased from 35 to 9, $p=0.000$, χ^2/df decreased from 79.904 to 69.644, GFI increased from 0.796 to 0.916, AGFI increased from 0.679 to 0.803, and RMSEA decreased from 0.184 to 0.170). The modified model for cardiovascular disease risks is shown in Figure 4.

Table 6 Standardized Regression Weights for the Cardiovascular Disease Risks
Latent Construct

Indicator Variable	Standardized Regression Weight
*Medications	0.344
*Systolic Blood Pressure	0.463
*Diastolic Blood Pressure	0.290
*Serum Cholesterol	0.664
*Serum High Density Lipoprotein	-0.165
*Serum Triglycerides	0.617
*Serum Vitamin E	0.739
*Serum Folate	0.306
*Body Mass Index	0.217
*Waist-to-Hip Ratio	0.461

* $p \leq 0.05$



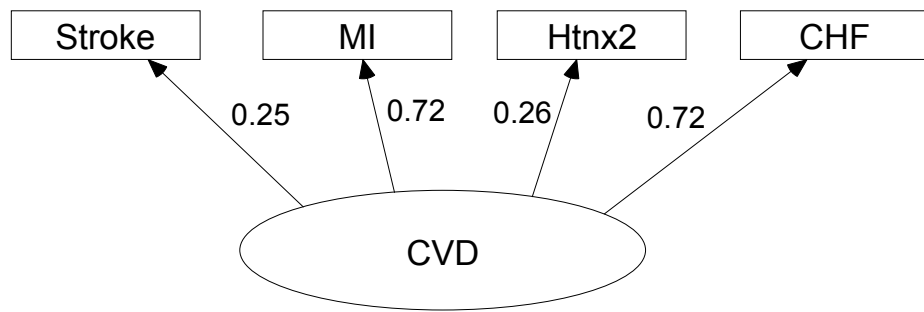
Systolic=Mean Systolic Blood Pressure, Serchol=Serum Cholesterol, Meds=CVD Medications,
Trigly=Serum Triglycerides, Svite=Serum Vitamin E, Waisthip=Waist-to-hip Ratio

There was a significant relationship between the latent construct cardiovascular disease outcomes and each of the indicator variables in the model ($\chi^2 = 23.784$, $df = 2$, $p = 0.000$, $\chi^2/df = 11.892$). Table 7 shows standardized regression weights for each of the indicator variables. The overall fit statistics confirmed the measurement model with a GFI = 0.995, an AGFI = 0.975 and a RMSEA of 0.068. The model for cardiovascular disease outcomes is shown in Figure 5.

Table 7 Standardized Regression Weights for Cardiovascular Disease Outcomes
Latent Construct

Indicator Variable	Standardized Regression Weight
*Stroke	0.245
*Hypertension (htnx2)	0.258
*Heart Attack (mi)	0.715
*Congestive Heart Failure (chf)	0.720

* $p \leq 0.05$



Htnx2=Hypertension, MI=Heart Attack, CHF=Congestive Heart Failure

Structural Model

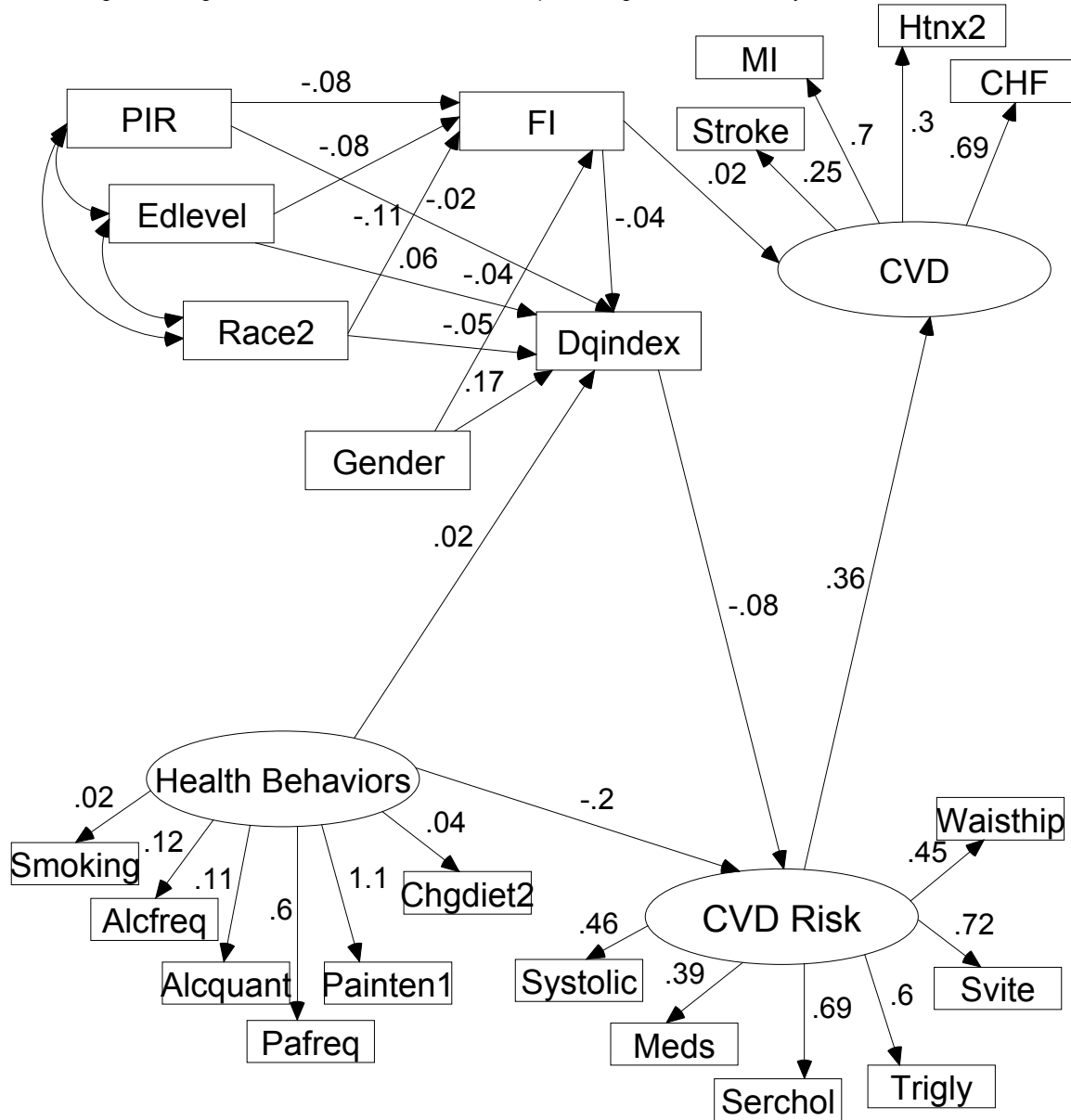
Next, structural equation modeling was used to test the full hypothesized model. Structural equation modeling did not confirm the theoretical model as it was hypothesized ($\chi^2 = 5565.865$, $df=198$, $p<0.001$, $GFI=0.808$, $AGFI=0.755$, $RMSEA=0.108$). Standardized regression weights for the hypothesized model are shown in Table 8. The hypothesized structural model is shown in Figure 6.

Table 8 Standardized Regression Weights for Structural Model of Relationships Among Food Insecurity, Health Behaviors, Cardiovascular Risks and Cardiovascular Disease

Path	Standardized Regression Weight
*PIR → Food insufficiency	-0.076
*Edlevel → Food insufficiency	-0.077
*Race → Food insufficiency	-0.113
*Gender → Food insufficiency	-0.040
*Gender → DQ Index	0.169
*Race → DQ Index	-0.052
*Edlevel → DQ Index	0.062
PIR → DQ Index	-0.019
Health Behaviors → DQ Index	0.019
*Food insufficiency → DQ Index	-0.044
*Health Behaviors → CVD Risk	-0.203
*DQ Index → CVD Risk	-0.080
Food insufficiency → CV Disease	0.020
*CV Disease → CVD Risk	0.356
Health Behaviors → Smoking	0.019
*Health Behaviors → Alcohol Frequency	0.116
*Health Behaviors → Alcohol Quantity	0.106
*Health Behaviors → Phys Activity Intensity	1.100
Health Behaviors → Change Diet	0.036
*Health Behaviors → Phys Activity Freq	0.604
*CV Disease → CHF	0.691
*CV Disease → HTN	0.302
*CV Disease → MI	0.724
*CV Disease → Stroke	0.25
*CVD Risk → Systolic	0.455
*CVD Risk → Meds	0.385
*CVD Risk → Serum Cholesterol	0.687
*CVD Risk → Triglycerides	0.599
*CVD Risk → Serum Vitamin E	0.719
*CVD Risk → Waist-Hip Ratio	0.445

* $p \leq 0.05$

Figure 6 Original Structural Model of Relationships Among Food Insecurity, CVD risk and CVD



PIR=poverity income ratio, Edlevel=education level of respondent, Race2=race/ethnicity, FI=food insufficiency status, Dqindex=diet quality index, MI=heart attack, Htnx2=hypertension, CHF=congestive heart failure, Alcfreq=alcohol frequency, Alcquant=alcohol quantity, pafreq=physical activity frequency, painten1=physical activity intensity, Chgdiet2=CVD-related change in diet, Systolic=systolic blood pressure, Meds=CVD medications, Serchol=serum cholesterol, Trigly=serum triglycerides, Svite=serum vitamin E, waisthip=waist-to-hip ratio

The hypothesized model was re-evaluated according to Campbell's proposed model of risk factors for and consequences of food insufficiency and non-significant paths and/or indicator variables were removed to create an alternate model.

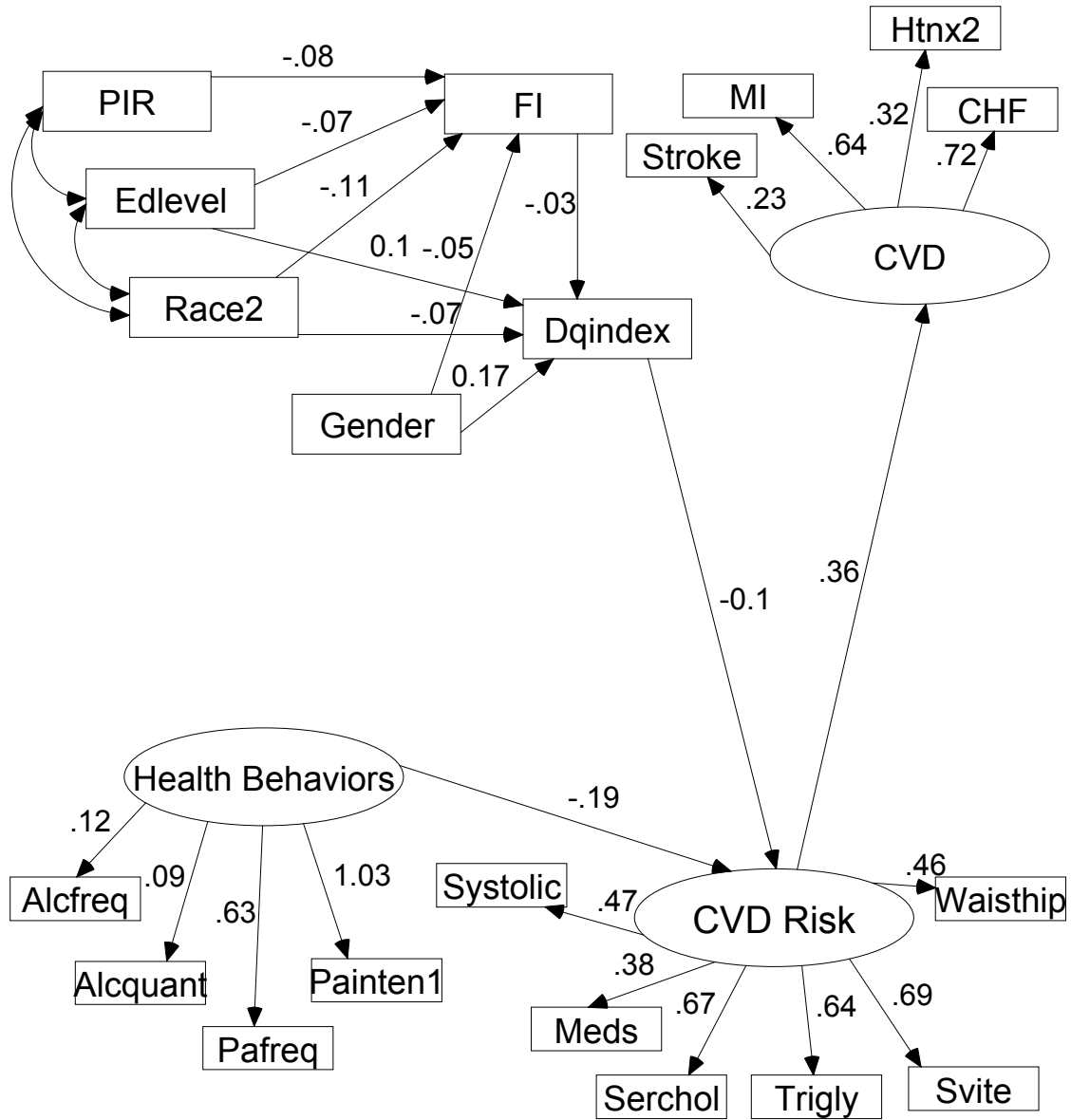
The alternate structural model was tested against the MT sample as a means of cross-validation. This procedure is recommended whenever model-generation techniques (post-hoc modifications) are used to modify a model that does not meet fit criteria (36). Structural equation modeling failed to confirm the alternate model ($\chi^2=4551.247$ df=162 GFI=0.837, AGFI=0.789, RMSEA=0.107). Table 9 shows the standardized regression weights for the modified structural model. Figure 7 shows the alternate structural model that was tested using structural equation modeling Due to failure to confirm the alternate structural model, we were unable to apply the model to the data from the remaining regions in the NHANES III dataset.

Table 9 Standardized Regression Weights for Modified Structural Model of Relationships Among Food Insufficiency, Health Behaviors, Cardiovascular Risks and Cardiovascular Disease

Path	Standardized Regression Weight
*PIR → Food insufficiency	-0.084
*Edlevel→ Food insufficiency	-0.066
*Race→ Food insufficiency	-0.111
*Gender→ Food insufficiency	-0.046
*Gender→ DQ Index	0.165
*Race→ DQ Index	-0.068
*Edlevel→ DQ Index	0.098
*Food insufficiency→ DQ Index	-0.026
*Health Behaviors→ CVD Risk	-0.185
*DQ Index→ CVD Risk	-0.096
*CV Disease→ CVD Risk	0.353
*Health Behaviors→ Alcohol Frequency	0.120
*Health Behaviors→ Alcohol Quantity	0.087
*Health Behaviors→ Phys Activity Intensity	1.033
*Health Behaviors→ Phys Activity Freq	0.626
*CV Disease→ CHF	0.722
*CV Disease→ HTN	0.324
*CV Disease→ MI	0.642
*CV Disease→ Stroke	0.227
*CVD Risk→ Systolic	0.465
*CVD Risk→ Meds	0.379
*CVD Risk→ Serum Cholesterol	0.669
*CVD Risk→ Triglycerides	0.644
*CVD Risk→ Serum Vitamin E	0.691
*CVD Risk→ Waist-Hip Ratio	0.459

*p<0.05

Figure 7 Modified Structural Model of Relationships Among Food Insecurity, CVD Risk, and CVD



PIR=Poverty Income Ratio, Edlevel=Education Level of Respondent, Race2=Race/Ethnicity,

Hypertension, CHF=Congestive Heart Failure, Alcfreq=Alcohol Frequency, Alquant=Alcohol Quantity, Pafreq=Physical Activity Frequency, Painten1=Physical Activity Intensity, Meds=

Vitamin E, Waisthip=Waist-to-Hip Ratio

Discussion

The present study used structural equation modeling to determine relationships among food insufficiency, diet quality, CVD risks and CVD outcomes based on the conceptual framework of risk factors for and consequences of food insecurity developed by Campbell (6). The hypothesized model was a mixed model containing measured variables predicting other measured variables as well as latent constructs being predicted by measured variables and in turn predicting other latent constructs. Structural equation modeling allowed for testing this fairly complex theoretical model by evaluation of how well the model reproduced the observed pattern of empirical relationships in the “southern sample” of the NHANES III dataset with the final goal being to compare the model from the “southern sample” to the same model applied to the remaining regions of NHANES III.

Failure of the model to fit the observed data resulted in the researcher rejecting the hypothesized model. Post-hoc modification was conducted to develop an alternate structural model. However, the alternate model also failed to fit the observed data in the MT sample. Mathematically, the variables chosen did not account for the researcher’s hypothesized association between food insufficiency and CHD outcomes. A number of factors may have contributed to this. First, NHANES III data are intended to be nationally representative. Therefore our future analysis will include the entire NHANES III dataset. This will still allow us to evaluate whether geographic difference is an effect modifier for the relationship between CVD and food insufficiency and to explore the etiologic factors for the differences, if any. Second, the health behaviors construct may not have been adequately identified. This may have contributed to the misfit observed in the structural model. In addition, the associations between the diet quality variable, the health behaviors construct, the CVD risk construct and several other indicator variables were not in the expected directions. Further analysis is needed to clarify the cause of these findings.

In spite of the mathematical problems with this empirical testing, Campbell’s model is still sound and appropriate. While overall model fit was the primary focus of this study, there are many confounding factors both known and unknown for CVD. In this study, the chi-square result was a very small p-value, but the GFI, AGFI, and RMSEA were considered to indicate reasonably good model fit in the measurement models but less than good fit in the structural model. It is possible that one of the indicators in the model is incorrect/inappropriate and this is causing the misfit in the structural model. By taking a closer look at relationships between indicator variables and latent constructs, relationships between latent constructs, and relationships between specific predictor variables we conceive of several design, analytical, and methodological considerations that we will address in future research.

Latent Constructs

The latent construct of health behaviors could be contributing to the misfit of the structural model. The indicators chosen to represent the latent construct of health behaviors were based on the literature suggesting an association between health behaviors and CVD risks/outcomes. In the final modified model, smoking and change in diet were dropped due to non-significant regression coefficients. The finding of a non-significant relationship between smoking and health behavior was rather surprising based on evidence from the report to the Surgeon General on smoking and CVD (37). The smoking variable was coded as a dichotomous variable from three questions related to the number of cigarettes, cigars or pipes reportedly smoked in the last five days by the respondent. In future analysis, we will use the more detailed questions on smoking from the entire NHANES III dataset to develop a better estimate for the daily amount of cigarettes, cigars, and pipes.

We were also surprised that the physical activity frequency and intensity variables loaded onto the same construct as the smoking and alcohol variables with the signs of the coefficients of these variables being in the same direction (positive). Correlations among these variables may be contributing to the misfit observed in this study. It is possible that the underlying construct is not “health behaviors,” but some other undetermined construct that includes other personal, behavioral, or lifestyle factors. For example, we did not assess the effects of poverty income ratio or education level on health behaviors. These variables probably do have some effect on health behaviors. We plan further analysis of this latent construct to include assessment of indirect, direct and total effects of the indicators to the outcome variables in the model.

The latent construct CVD risk was also difficult to assess. In the measurement model, all indicator variables had a significant association with the latent construct. However the measurement model did not meet fit criteria. The researcher removed variables with regression weights of less than 31%, which improved the fit to a marginally acceptable level. Perhaps there were inter-relationships between the indicator variables that had an impact on the outcome of the measurement model. For example the researcher expected serum vitamin E (svite) to have a protective effect against CVD, therefore a low svite would increase CVD risks (a negative association). However, an opposite association was detected since the serum vitamin E variable had a positive regression weight. High levels of serum triglycerides are associated with increased risk for CVD (38). Triglycerides are transported predominantly by very low-density lipoproteins (VLDL), vitamin E is distributed between the various lipoprotein fractions where it helps to protect against oxidation of the lipoprotein (39). If the increased level of triglycerides were to be accompanied by increased levels of all lipoprotein fractions an accompanying increase in serum vitamin E might be expected. This question was not a focus of the present research, however, this might explain why serum vitamin E had a positive association with the latent construct and resulted in a measurement model that met fit criteria.

Likewise, serum folate (sfolate) was chosen as an indirect indicator of serum homocysteine due to the lack of data on serum homocysteine in the NHANES III dataset available to the researchers. High levels of serum homocysteine have been correlated

with increased CHD (40). High levels of serum folate are associated with lower levels of serum homocysteine (40;41). Therefore, serum folate should have been a negative weight associated with CVD risks, yet this variable had a positive regression weight in the measurement model. This is counter to a great deal of research associated with the metabolism of folate and homocysteine. Further research is necessary to clarify why the regression weight of this variable was positive.

Another possibility is that the indicator variables chosen measure more than one latent construct. Future research should attempt to determine if there are two distinct latent constructs measured by these variables. Perhaps one construct is best indicated by factors having a positive association with CVD (high risks for CVD) and one is best indicated by factors that have a negative association with CVD (low risks for CVD). There may also be other indicator variables (such as having diabetes or a family history of CVD) that would strengthen the measurement model and be better indicators than some of the variables chosen.

The measurement model for the latent construct CVD was quite strong indicating the variables chosen to represent this construct were appropriate. In future studies of the relationship between food insufficiency, CVD risks and CVD outcomes, this latent construct could be maintained.

Relationships between Latent Constructs

Structural equation modeling identified a negative relationship between the latent constructs health behaviors and CVD risk. This would be interpreted as an increase in these behaviors lowers CVD risk. This is the opposite of what was expected for the variables smoking, alcohol frequency, and alcohol quantity. As noted earlier, this may be the result of correlations among the indicators in the health behaviors construct, not specifying all the indicators associated with that construct, or an underlying construct that is not health behaviors. Further consideration of this finding is noted in the following paragraphs.

The SD of the alcohol frequency and quantity variables (alcfreq and alcquant) was quite large due to outliers in the data that also may have affected the direction of the association. The literature suggests that excessive alcohol consumption should be positively associated with at least one type of CVD (hypertension)(42). However, that same report noted that moderate alcohol consumption (2 drinks/day or less) might reduce the risk of CHD through its influence on other factors associated with CHD. The mean frequency of alcohol consumption for both MF and the MT samples was approximately 45 days, and mean number of drinks on a drinking day was approximately 1.68. Seventy-six percent of the respondents in the samples reported two drinks or less on a drinking day. Therefore there may have been biological benefits associated with the moderate consumption of alcohol that influenced the direction of the association between these variables. Future analysis will include combining alcohol intake frequency and quantity to calculate a single continuous variable for the average amount of alcohol consumed per person to determine if this effect remains.

Physical activity should have a negative relationship to CVD risks, as was the result of the hypothesized model. However, the finding that physical activity frequency and intensity variables (pafreq and painten1) “loaded” on the same latent construct as smoking, alcfreq and alcquant is puzzling as was discussed above.

The Centers for Disease Control/American College of Sports Medicine (CDC/ACSM) recommendations (43) for physical activity levels that promote CVD health encourage moderate to vigorous physical activity most if not every day of the week. These same guidelines define moderate physical activity as those activities with an intensity of effort rating of 3.0-6.0 METs while vigorous physical activities have a MET rating of greater than 6.0. In the combined MF and MT samples the mean activity frequency (pafreq) was 1.53 occasions of physical activity with individual means ranging from 0-18 occasions of physical activity over the previous month. As a result of our recoding, of the pafreq variable, a pafreq of 1.00 would translate into 13 occasions of physical activity over the previous month, which met recommended minimum frequency of three days per week. However, the pafreq questions were not measured in days/month, but in occasions/month. Fifty-six percent of the combined MF and MT samples did not meet the CDC/ACSM recommendations for minimum physical activity frequency. A similar profile was observed for the physical intensity rating (painten1) variable. The mean painten1 was 0.69 METs with a mean range of 0-4.85 METs. Ninety-eight percent of the MF and MT samples had mean painten1 ratings of less than 3 METs. This implies a negative impact of physical inactivity, combined with the low proportion of smokers and the high proportion of moderate drinkers in the sample attenuated the relationship of health behaviors forcing the indicator variables to “load” onto one latent construct resulting in a cumulative negative relationship between health behaviors and CVD risk. As noted earlier, we plan further investigation of the correlation among the physical activity variables, the alcohol and smoking variables and the CVD risk construct to try to clarify the finding of a negative relationship between health behaviors and CVD risk.

The original structural model hypothesized a relationship between the latent variable health behaviors and the quality of the diet (dqindex). This was based on studies of the dietary intakes of persons practicing particular health behaviors compared to those not practicing the behaviors (44)-(23;45;46). However, there was not a significant relationship between health behaviors and dqindex in this researcher’s model.

The dqindex variable included intakes of cholesterol, sodium, saturated fat, total fat, calcium, and protein due to their purported relationship with chronic diseases such as CHD (29). These nutrients are also hypothesized to be associated with other indicator variables in the latent construct of health behaviors. Beaton, Milner, Corey, et al. (47) noted that intra-individual variation in dietary intakes will bias estimates of correlation coefficients from single 24-hour dietary recalls. It is likely that the limitations associated with a single 24-hour dietary recall introduced either error or bias in the dqindex, resulting in the non-significant association between it and the health behaviors latent construct. Another possibility is that the problems noted with the health behaviors construct are influencing its association with the dqindex. Re-evaluation of both the health behaviors construct and the dqindex construct are necessary to clarify this finding.

Specific Predictor Variables

There was a significant negative relationship between poverty income ratio (pir), education level (edlevel), race (white=1, non-white=0), gender (male=1, female=0), and food insufficiency status (FI). In the present study, FI was coded 1 for those reporting one or more days without food or skipped meals due to resource constraints. Therefore a negative relationship between the predictor variables (pir, edlevel, race, and gender) and food insufficiency would be expected based on the literature. In the present study, those with low pir, those with low edlevel, non-white and female would be more likely to report being food insufficient than those of opposite demographic and economic characteristics. Findings are in agreement with those reported in the literature on risk factors related to food insecurity/insufficiency. Therefore future studies assessing relationships between food security/sufficiency, disease risks, and disease outcomes should continue to include these relationships in theoretical models.

When FI was used as a predictor of diet quality (dqindex), the relationship was found to be negative in the present structural model. With food insufficiency being coded 1 and higher scores for the dqindex indicating a poorer quality diet, this means that the food insufficient had a better quality diet. While the relationship was significant in both the original and the alternate structural models, the magnitude of the effect was low with both models having a regression weight of -0.04 . Cristofar and Basiotis (9) reported significantly lower intakes of protein, total fat, and saturated fat among women reporting food insufficiency than those not reporting food insufficiency in data from the 1985-1986 low income samples of the CSFII. If this held true for the NHANES III data and depending on the magnitude of the differences between the food sufficient and food insufficient, a lower dqindex score for food insufficient adults might be possible since lower intakes of these particular nutrients result in a lower dqindex score. However, Dixon, Winkleby, and Radimer (16) reported only lower intakes of calcium among young adults from food insufficient households and lower intakes of energy, vitamin B6, magnesium, iron and zinc among older adults from food insufficient households compared to their food sufficient counterparts from NHANES III. Mean intake of these nutrients was less than 50% of the RDA for the food insufficient groups. Since calcium was the only nutrient mentioned above that is also a component of the dqindex, this suggests that the dqindex would not be influenced by food insufficiency status in the NHANES III sample.

Relationships between the predictor variables pir, edlevel, race, and gender and the dependent variable dqindex were also assessed in the structural model. There was a significant relationship between edlevel, race, gender, and dqindex, but not between pir and dqindex. In general, the literature would suggest that income is related to the quality of the diet at least with respect to intake of specific nutrients (48-52). It is possible that the dqindex used in this study did not capture the specific nutrient intakes that did not meet dietary recommendations in the southern sample of the NHANES III dataset. As noted previously, Dixon et al. (16) recently found lower intakes of calcium, energy, vitamin B6, magnesium, iron and zinc among a sample of adults from food insufficient households in NHANES III compared to the food sufficient adults. The dqindex used in

this study was computed from intakes of total and saturated fat, cholesterol, sodium, protein, and calcium because of their relationship to CHD. This may explain the non-significant relationship between *pir* and *dqindex*, but further investigation is needed.

While the other predictor variables (gender, *edlevel*, race) had significant relationships with *dqindex*, the direction of the association was somewhat contrary to that expected based on the literature. In the present study, *edlevel* had a positive association with *dqindex* indicating that those with higher education levels had higher *dqindex* scores (poor diet quality). Higher education level has been reported to be associated with better nutrient intakes as related to specific health recommendations (51;53;54) as well as with Healthy Eating Index scores indicative of a better quality diet (55). It is unclear whether the findings in the present study are due to the limitations noted earlier with the computation of the *dqindex* or if perhaps the *edlevel* of the sub-sample from NHANES III in this study was associated with better jobs, thereby better income, allowing for excessive intakes of the nutrients comprising the *dqindex* that are related to CVD risk.

The gender relationship to diet quality demonstrated being male resulted in higher *dqindex* scores (or poorer quality diet) than females. This finding is in general agreement with the findings of Bowman et al. (55) that females generally had HEI scores indicating higher quality diet than males. The race relationship to *dqindex* demonstrated non-whites in this sample had higher *dqindex* scores than whites. Bowman et al (55) and Variyam, Blaylock, Smallwood and Basiotis (56) reported HEI scores indicating poor diet quality among African Americans compared to other race groups, but no difference between HEI scores of Hispanics and non-Hispanics. In the current study, blacks were grouped with Mexican-Americans and other race/ethnicity groups in order to create a dichotomous variable for race. The proportion of blacks in the MF and MT samples was 56.3%, and Mexican-American and others was 43.6%. The proportion of blacks in the sample should not unduly influence the direction of the association, even if this group's diet quality was indeed poorer than the other groups (and it was not determined that this was the case). Therefore, it is unclear whether the association of non-white race with a high *dqindex* score is a result of truly poor quality diets in this group, or some other reason associated with the computation of the *dqindex* from a single 24-hour recall, use of the southern sub-sample from NHANES III, or some other undetermined reason.

Finally, there was a negative relationship between the *dqindex* and the latent construct CVD risk and the magnitude of the regression coefficient was very small (-0.08). This means that a better diet was associated with increased CVD risk. This finding directly conflicts with the literature. The Surgeon General's Report on Nutrition and Health (57) concluded a positive association between CHD and the dietary components of total fat, saturated fat, and cholesterol through their impact on CHD risk factors such as serum cholesterol. In addition, this report noted that high sodium intakes and low calcium intakes might be positive risk factors for HTN. These nutrients (with the exception of protein) were used to compute the *dqindex* in the present study. Therefore a high *dqindex* denoting a poor quality diet based on these nutrients should have a positive association with CVD risk. Again, this study's finding might be an artifact introduced by

intra-individual variation in dietary intakes associated with a single 24-hour dietary recall, which could bias the regression coefficients for diet quality, or due to one of the other possibilities previously discussed.

Future research will need to investigate whether the problems encountered with the diet quality index are associated with using a single 24-hour dietary recall to compute the index, using the southern sub-sample from NHANES III, correlations among indicators that influence diet quality or some other factor. As noted earlier, it would be extremely difficult to overcome the limitations associated with using a dqindex computed from a single 24-hour dietary recall if it were determined that this was the cause of the present study findings. At the same time, it is important to remember that the impact of dietary quality on CVD risk (or any nutrition related disease) is cumulative and occurs over time. If the diet quality index used in the present study is determined, through further investigation, to be an inappropriate measure to use in SEM, future studies will need to develop an alternate measure of diet quality in order to assess the indirect effects of food insufficiency on CVD risk.

Recommendations for Future Research

The problems encountered in the present study serve to inform our future research efforts on the study of relationships among food insufficiency, disease risks, and disease outcomes. First, research is needed to determine why the indicator variables used in the health behavior latent construct loaded together in the manner previously discussed. Re-creating the variables used to indicate this latent construct so that the smoking variable as well as the alcohol variable is better defined and investigating correlations among the variables may help to clarify this finding. In addition further investigation of this may help to identify inappropriate indicators as well as indicators that were not identified previously.

Research is also needed to determine whether CVD risk could be separated into high risk and low risk latent constructs. It is unclear whether this would be beneficial due to the unusual finding of a positive association between serum vitamin E levels and CVD risks as noted previously and the finding that serum folate levels were not a significant predictor of CVD risk. Perhaps this latent construct would be better defined if only those factors noted as high risk factors in the literature were used to define this construct (serum cholesterol, blood pressure, serum triglycerides, waist-to-hip ratio). Furthermore, there may be other factors that should be included in the latent construct (such as diabetes, family history).

Our findings suggest that more research is needed to select and compute a measure of diet quality using the NHANES III data. The diet quality index (29) that the researcher chose and computed from a single 24-hour recall did not result in the expected relationships with other variables and constructs in the model. However, the reason for this cannot be clarified without further investigation. It is important to have an appropriate measure of diet quality because without this, researchers who wish to use NHANES III data to assess relationships among food insufficiency, disease risks, and disease outcomes may assess only the direct relationship of food insufficiency to disease

risk or disease outcomes. Omission of the dietary variable results in the loss of the ability to assess the indirect affects of food insufficiency with these constructs through its relationship to diet quality. It is important to clarify the nutritional influence of food insecurity on disease risk and outcomes so that policy initiatives that address nutrition and health can appropriately target vulnerable populations. Therefore, should further investigation reveal that the diet quality index chosen for this study is not appropriate, some alternative measure of diet quality will need to be developed from NHANES III data for this purpose.

Campbell's model included direct and indirect relationships between food security/sufficiency, nutrition status and health outcomes. In the present study, food insufficiency did not have a direct relationship to CVD. Although, structural equation modeling failed to confirm the model, a direct relationship could exist but was not detected with the data used in this study.

Finally, we suggest that further research assess the direct effect of the indicators on the end outcome of CVD. One of the strengths of SEM is to assess direct effects by summing the direct and indirect effects and determining the total effect using the regression coefficients of the indicators. While the structural model was not confirmed in this study, there appear to be some interesting direct and indirect effects among indicators in the model that were not addressed at this time. Assessment of these effects may suggest areas of future investigation in cardiovascular disease prevention and/or management.

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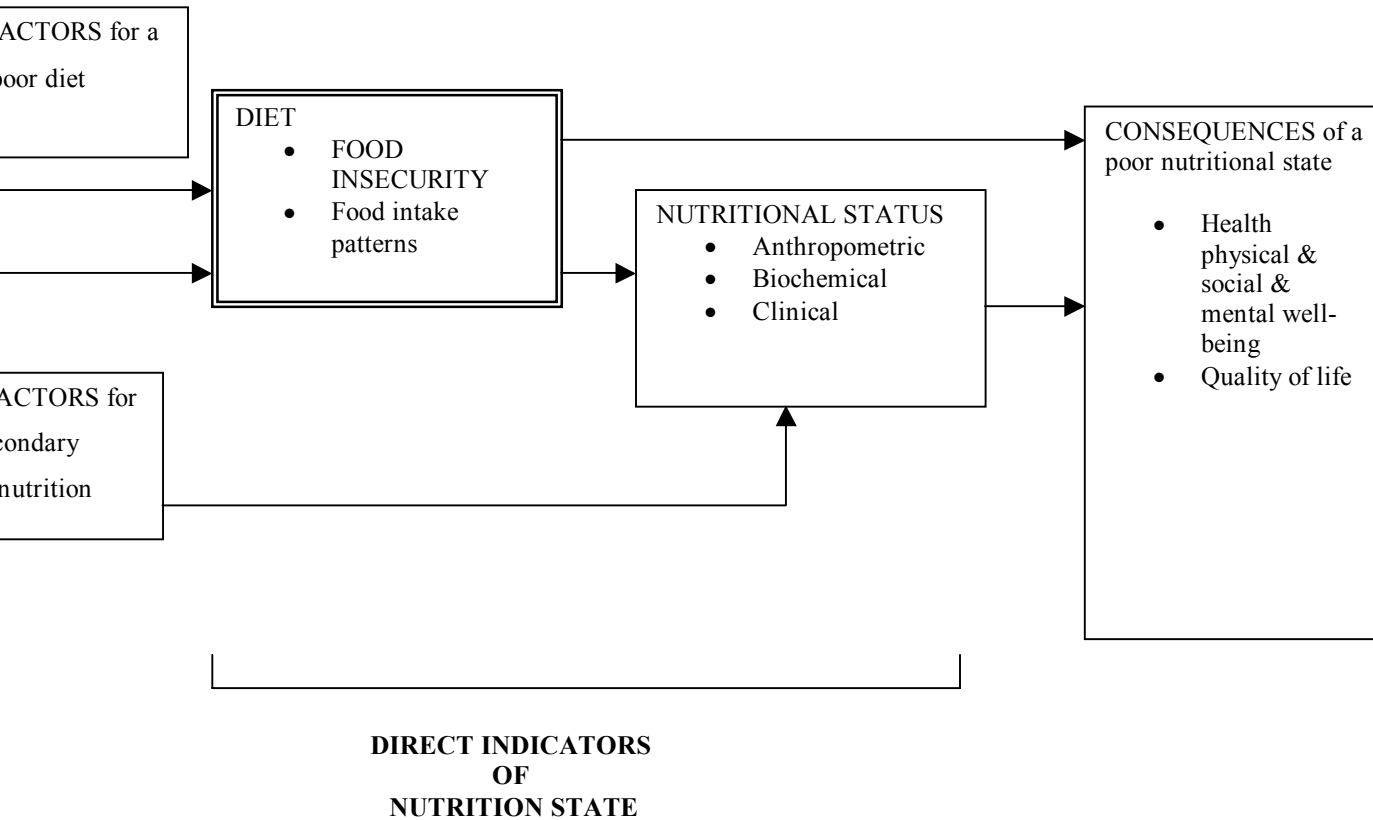
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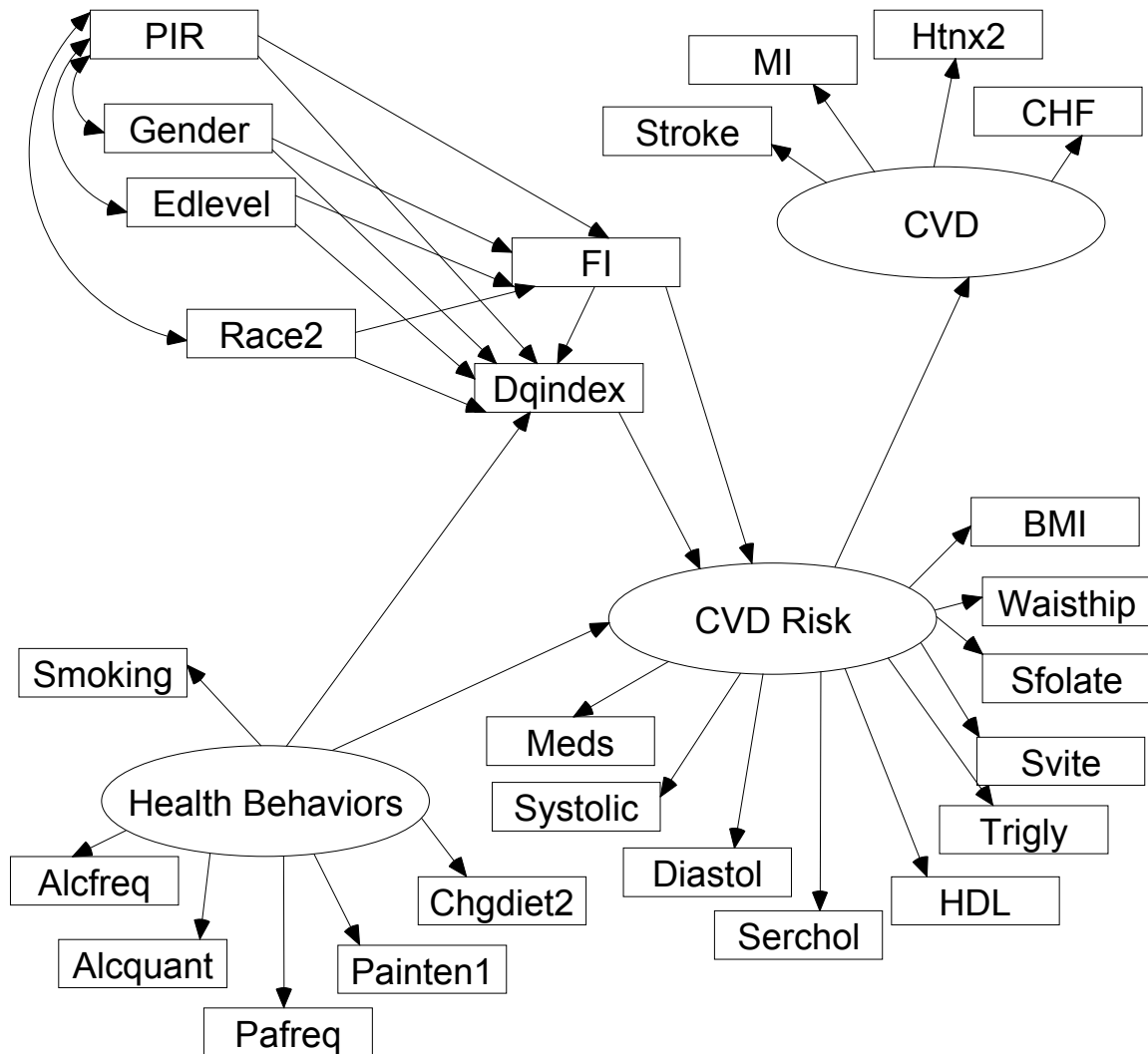
Appendix

Figure 1 Campbell's Conceptual Framework of Risk Factors for and Consequences of Food Insecurity



al Status of Nutrition Related Indicators
bell (1991)

CVD risks, and CVD Prior to Structural Equation Modeling



PIR=poverity index ratio, Edlevel=education level of respondent, Race2=race/ethnicity, MI=heart attack, Htnx2=hypertension, CHF=congestive heart failure, CVD=cardiovascular disease, FI=food insufficiency, Dqindex=modified diet quality index, Alcfreq=alcohol frequency, Alcquant=alcohol quantity, Pafreq=physical activity frequency, Painten1=physical activity intensity, Chgdiet2=change diet for CVD, Meds=CVD medication, Systolic=systolic blood pressure, Diastol=diastolic blood pressure, Serchol=serum cholesterol, HDL=high density lipoprotein, Trigly=serum triglycerides, Svite=serum vitamin E, Sfolate=serum folate, Waisthip=waist-to-hip ratio, BMI=body mass index, CVD risk=cardiovascular disease risk

Table 1 Variables Used in Hypothesized Structural Model

Construct/Variable	Abbreviation	Units	Variable Type	Coded
HEALTH BEHAVIOR				
No. Days Drank Alcohol	Alcfreq	Days	Continuous	N/A
No. Drinks on Drinking Day	Alcquant	Drinks	Continuous	N/A
Mean Physical Activity Freq. (computed from 13 questions related to various types of exercise)	Pafreq	Mean no. occasions/mo.	Continuous	N/A
Mean Physical Activity Intensity (computed from 13 variables related to the intensity of reported types of exercise)	Painten1	Average METs	Continuous	N/A
Smoking Status (computed from 3 questions on number of cigarettes, cigars, or pipes smoked past three days)	Smoking	N/A	Dichotomous (Yes/No)	1/0
Change Diet (computed from 8 questions related to changes in diet due to CVD)	Chgdiet2	N/A	Dichotomous (Yes/No)	1/0
CARDIOVASCULAR RISK				
Mean Systolic Blood Pressure	CVDrisk			
Mean Diastolic Blood Pressure	Systolic	mmHg	Continuous	N/A
Body Mass Index	Diastol	mmHg	Continuous	N/A
Waist-to-Hip Ratio	BMI	Ratio	Continuous	N/A
Serum Folate	Waisthip	Ratio	Continuous	N/A
Serum Vitamin E	Sfolate	ng/ml	Continuous	N/A
Serum Cholesterol	Svite	µg/dl	Continuous	N/A
Serum Triglycerides	Serchol	mg/dl	Continuous	N/A
Serum HDL Cholesterol	Trigly	mg/dl	Continuous	N/A
Medication for CVD (computed from 48 variables related to classification of drugs reported by the respondent)	HDL	mg/dl	Continuous	N/A
	Meds	N/A	Dichotomous (Yes/No)	1/0
CARDIOVASCULAR DISEASE				
Self-reported Congestive Heart Failure	CVD			
Self-reported Stroke	CHF	N/A	Dichotomous (Yes/No)	1/0
Self-reported Hypertension	Stroke	N/A	Dichotomous (Yes/No)	1/0
Self-reported Heart Attack	Htnx2	N/A	Dichotomous (Yes/No)	1/0
	MI	N/A	Dichotomous (Yes/No)	1/0
OTHER VARIABLES				
Gender	Gender	N/A	Dichotomous (Male/Female)	1/0
Race/Ethnicity (computed from 4 categories of race/ethnicity reported by the respondent)	Race2	N/A	Dichotomous (white/non-white)	1/0
Food Insufficiency (computed from responses to 2 questions on no. of days w/o food or skipped)	FI	N/A	Dichotomous (Yes/No)	1/0
Poverty Income Ratio	PIR	Ratio	Continuous	N/A
Education Level of Respondent	Edlevel	Years completed	Continuous	N/A
Modified Diet Quality Index	Dqindex	N/A (Score)	Continuous (0-12)	N/A

Table 2 Modified Diet Quality Index‡

Recommendation*	Score†	Intake
Reduce total fat intake to 30% or less of total energy	0	≤ 30%
	1	>30-40%
	2	>40%
Reduce saturated fatty acid intake to less than 10% of energy	0	<10%
	1	10-13%
	2	>13%
Reduce cholesterol intake to less than 300 mg daily	0	<300 mg
	1	300-400 mg
	2	>400 mg
Maintain protein intake at moderate levels (levels lower than twice the RDA)	0	< 100% RDA
	1	100-150% RDA
	2	>150% RDA
Limit total daily intake of sodium to 2400 mg or less	0	≤ 2400 mg
	1	> 2400-3400 mg sodium
	2	>3400 mg sodium
Maintain adequate calcium intake (approximately the RDA)	0	≥100% RDA
	1	67-99% RDA
	2	<67% RDA

‡ Modified from Patterson, Haines and Popkin (1994).

* RDA denotes Recommended Daily Allowance.

† Scores for the six recommendations are totaled to generate a Modified Diet Quality Index score for a respondent (range 0 to 12).