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Food Insecurity Is Associated with Chronic Disease among Low-Income NHANES Participants[1,2]

Hilary K. Seligman,[3]* Barbara A. Laraia,[4] and Margot B. Kushel[3]

[3]Department of Medicine, San Francisco General Hospital, University of California, San Francisco, CA 94143; and [4]Department of Medicine, Division of Prevention Sciences, University of California San Francisco, San Francisco, CA 94143

Abstract

Food insecurity refers to the inability to afford enough food for an active, healthy life. Numerous studies have shown associations between food insecurity and adverse health outcomes among children. Studies of the health effects of food insecurity among adults are more limited and generally focus on the association between food insecurity and self-reported disease. We therefore examined the association between food insecurity and clinical evidence of diet-sensitive chronic disease, including hypertension, hyperlipidemia, and diabetes. Our population-based sample included 5094 poor adults aged 18–65 y participating in the NHANES (1999–2004 waves). We estimated the association between food insecurity (assessed by the Food Security Survey Module) and self-reported or laboratory/examination evidence of diet-sensitive chronic disease using Poisson regression. We adjusted the models to account for differences in age, gender, race, educational attainment, and income. Food insecurity was associated with self-reported hypertension [adjusted relative risk (ARR) 1.20; 95% CI, 1.04–1.38] and hyperlipidemia (ARR 1.30; 95% CI, 1.09–1.55), but not diabetes (ARR 1.19; 95% CI, 0.89–1.58). Food insecurity was associated with laboratory or examination evidence of hypertension (ARR 1.21; 95% CI, 1.04–1.41) and diabetes (ARR 1.48; 95% CI, 0.94–2.32). The association with laboratory evidence of diabetes did not reach significance in the fully adjusted model unless we used a stricter definition of food insecurity (ARR 2.42; 95% CI, 1.44–4.08). These data show that food insecurity is associated with cardiovascular risk factors. Health policy discussions should focus increased attention on ability to afford high-quality foods for adults with or at risk for chronic disease. J. Nutr. 140: 304–310, 2010.

Introduction

Food insecurity refers to the inability to afford nutritionally adequate and safe foods (1). In 2008, more than 14% of all U.S. households, 49 million people, were food insecure (2). Most adults living in food-insecure households report being unable to afford balanced meals, worrying about the adequacy of their food supply, running out of food, and cutting the size of meals or skipping meals. At the most severe levels of food insecurity, many adults report being hungry because there was not enough money for food and not eating for an entire day (2). Each episode of food insecurity is generally short in duration. However, the dietary changes associated with food insecurity may persist over extended periods, because food-insecure households often experience repeated food budget shortages. On average, households that report being food insecure at some time during the year are food insecure for 7 mo during the year (2).

Common household responses to inadequate food supplies include food budget adjustments, reduced food intake, and alterations in types of food served (3–6). Dietary variety decreases and consumption of energy-dense foods increases. These energy-dense foods, including refined grains, added sugars, and added saturated/trans fats, tend to be of poor nutritional quality and less expensive calorie-for-calorie than alternatives (7,8). U.S. adults living in food-insecure households consume fewer weekly servings of fruits, vegetables, and dairy and lower levels of micronutrients, including the B complex vitamins, magnesium, iron, zinc, and calcium (5,9,10). These dietary patterns are linked to the development of chronic disease, including hypertension, hyperlipidemia, and diabetes (11,12).

There have been many studies suggesting that food insecurity among children has adverse health effects, including increased rates of iron-deficiency anemia, acute infection, chronic illness, and developmental and mental health problems (13–19). A

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* To whom correspondence should be addressed. E-mail: hseligman@medsfgh.ucsf.edu.

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number of studies have demonstrated associations between food insecurity and overweight/obesity among children and adult women using both self-reported and objective measures of BMI (20), although results among children have been inconsistent (21–23).

However, there have been few studies evaluating the association between food insecurity and chronic disease among adults. What studies have been done generally rely on self-reported measures without confirmation of the association using objective measures of chronic disease. Using clinical evidence of disease is important in low-income populations, because delays in medical care may preferentially reduce disease diagnosis among the most financially vulnerable, biasing results toward an underestimate of the association between food insecurity and chronic disease (24).

In this study, we used a population-based sample to evaluate associations between food insecurity and objectively measured cardiovascular risk factors, including hypertension, hyperlipidemia, and diabetes. Because we used examination and laboratory data to assess the presence of chronic disease, we were able to quantify both the prevalence and severity of these conditions among adults living in food-insecure households.

Methods

Sample. The NHANES is a cross-sectional, nationally representative survey of the noninstitutionalized U.S. civilian population (25). Details of recruitment have been published previously (26). We combined data from the 1999–2000, 2001–2002, and 2003–2004 waves of NHANES. We restricted our analysis to adults aged 18–65 y reporting household incomes ≤200% of the federal poverty level (FPL).[4] We excluded children because of the low prevalence of the chronic diseases in which we were interested; the elderly because of the high prevalence of the chronic diseases in which we were interested (suggesting alternative disease pathways) and because of differences in access to health care between poor elderly and poor nonelderly adults; pregnant women because pregnancy affects the clinical variables in which we were interested; and individuals living in households reporting incomes >200% of the FPL because the prevalence of food insecurity in these households is <4% (27). The FPL is a number updated annually by the Census Bureau to reflect the household income at which a family is considered to be in poverty. The FPL varies according to household size and age composition; e.g. the FPL for a family of 2 adults and 2 children in 2008 was $21,834.

Data collection. All NHANES participants completed an English or Spanish version of an interviewer-administered questionnaire in their own home. Participants then attended a specially equipped mobile examination center where they underwent a standardized physical (including height, weight, and blood pressure measurements) and laboratory examination (28). A random subset of participants underwent a fasting laboratory examination.

Food security. More than 99% of the eligible sample participated in the Food Security Survey Module, which is a well-validated questionnaire developed by the USDA to measure household food security over the prior 12 mo (6,29). Because we were interested in adult health outcomes, we used responses to only the 10 household and adult items in the 18-item scale. (The remaining 8 items refer to household children.) Using validated cutpoints, we considered an adult to be food secure if ≤2 items in the scale were answered affirmatively (often referred to as high and marginal food security) and food insecure if ≥3 items were answered affirmatively (often referred to as low and very low food security) (6). In a sensitivity analysis, we also dichotomized the scale as ≤5 affirmative responses (high, marginal, and low food security) and ≥6 affirmative responses (very low food security). The Chronbach's α for the scale ranges from 0.74 to 0.93 (30).

Clinical variables. We evaluated as dependent variables both a self-reported and a clinical diagnosis of hypertension, hyperlipidemia, and diabetes. Self-reported disease was identified by affirmative answers to the question "Has a doctor or other health professional ever told you (that you had hypertension, also called high blood pressure) (that your blood cholesterol level was high) (that you have diabetes or sugar diabetes)?" Either of 2 criteria was used to define participants with clinical disease: out-of-range laboratory/examination measures or medication use. Using this method, we included as having clinical disease those who had undiagnosed disease (out-of-range laboratory/examination measures) as well as those with diagnosed disease who were being successfully treated such that their laboratory/examination measures were now in the normal range (medication use). That is, we considered clinical evidence of hypertension to be either a measured systolic blood pressure (SBP) >140 mm Hg or diastolic blood pressure (DBP) >90 mm Hg, consistent with current Joint National Committee guidelines (31), or self-reported use of antihypertensive medication. We considered clinical evidence of hyperlipidemia to be either a total cholesterol concentration ≥240 mg/dL (6.22 mmol/L) or a fasting LDL cholesterol concentration ≥160 mg/dL (4.14 mmol/L), consistent with current Adult Treatment Panel-III guidelines (32), or self-reported use of lipid-lowering therapy. We considered clinical evidence of diabetes to be either a fasting plasma glucose ≥126 mg/dL (6.99 mmol/L), consistent with American Diabetes Association and WHO guidelines (33,34), or self-reported use of insulin or oral hypoglycemic medication.

Our covariates included sociodemographic factors known to be associated with chronic disease and/or food insecurity, including age, gender, race/ethnicity (self-reported non-Hispanic White, non-Hispanic Black, Hispanic, or other/multiple), education (less than high school, high school degree, or more than high school degree), and household income. To fully adjust for income, we included income as both continuous and categorical (0–50, 50.1–100, 100.1–150, or 150.1–200% of the FPL) variables.

Statistical analysis. Results were weighted to represent the noninstitutionalized U.S. population aged 18–65 y. We used the complex survey commands in Stata 9.2 (2007) to obtain weighted estimates, SE, and *P*-values that correctly reflected the stratification and clustering of observations induced by NHANES' design. We used 6-y interview weights for self-reported variables and mobile examination center weights for laboratory and examination data. We considered all *P*-values < 0.05 as significant.

We compared differences in baseline characteristics using the design-based F statistic. We estimated the association between food insecurity and disease prevalence using Poisson regression with robust SE and estimated probabilities directly from the regression models. We report an unadjusted prevalence, a crude relative risk (CRR; adjusting for age, gender, and race/ethnicity), and an adjusted relative risk (ARR; adjusting for age, gender, race/ethnicity, education, and income). For participants with more than 1 diagnosis (hypertension, hyperlipidemia, or diabetes), we evaluated each condition independently.

To determine whether self-management capacity among those with chronic disease was limited by food insecurity, we evaluated the subset of adults who self-reported a disease diagnosis for inadequate control. We defined control as adherence to current treatment goals, because we were interested in whether food-insecure adults were at increased risk of long-term complications related to chronic disease. For hypertension, we defined inadequate control as blood pressure >140/90 mm Hg, for hyperlipidemia a total cholesterol concentration ≥200 mg/dL (5.18 mmol/L) or LDL cholesterol ≥140 mg/dL (3.63 mmol/L) (35), and for diabetes a glycated hemoglobin (HbA1c) >7% (36). Where significant associations between food insecurity and inadequate disease control were found, we used linear regression to estimate the association with continuous outcomes.

[4] Abbreviations used: ARR, adjusted relative risk; CRR, crude relative risk; DBP, diastolic blood pressure; FPL, federal poverty level; HbA1c, glycated hemoglobin; RR, relative risk; SBP, systolic blood pressure.

We performed 3 sensitivity analyses. In the first, we defined food insecurity using all 18 items of the Food Security Survey Module, including the 8 items referring to the food security status of children in the household that were not included in the main analysis. Second, we used a more severe definition of food insecurity (≥6 affirmative responses rather than ≥3 affirmative responses). Finally, we included BMI (calculated in kg/m^2 from measured height and weight) in the adjusted models to determine the extent to which food insecurity might independently contribute to the development of cardiovascular risk factors.

The available sample size varies for the dependent variables because of skip patterns and the way in which NHANES participants are chosen to undergo the fasting laboratory evaluation. For example, all adults had total cholesterol level data as part of the laboratory evaluation (n = 4559), but only adults reporting they had had their cholesterol checked in the past were asked if they had been diagnosed with hyperlipidemia (n = 1930). Because weighting accounts for differential sampling probabilities, we used the entire available sample for each analysis rather than restricting the analysis to the subset of adults for whom we had access to complete data. The sample sizes available for each analysis are indicated in the tables. This secondary data analysis was approved by the Committee of Human Research at the University of California San Francisco.

Results

NHANES 1999–2004 included 31,126 individuals. We restricted from the analysis pregnant women (n = 679), youth (<18 y of age) (n = 14,047), and the elderly (>65 y of age) (n = 4099). We excluded another 7162 participants because of a household income >200% of the FPL and 45 participants because they did not respond to the Food Security Survey Module. These exclusions resulted in 5094 participants. Of these participants, 95.6% of those from food-secure households and 98.5% of those from food-insecure households also participated in the mobile examination/laboratory evaluation (n = 4908).

Latino households were more likely to be food insecure than White households ($P < 0.001$). Household food insecurity was also associated with low educational attainment, low household income, lack of health insurance, and tobacco use (**Table 1**). Consistent with previous studies, we observed an association between food insecurity and a higher BMI among women only.

We combined results for men and women because stratification by gender yielded similar risk estimates for men and women. Among adults from food-secure households, 18.6% had clinical evidence of hypertension compared with 22.4% of adults from food-insecure households (**Table 2**). In adjusted analyses, adults from food-insecure households had a 21% (95% CI, 4–41%) higher risk of clinical hypertension than adults from food-secure households (P = 0.02). Mean SBP and DBP did not differ by food security status (data not shown). The association between household food insecurity and clinical evidence of hyperlipidemia was not significant (P = 0.4).

There was clinical evidence of diabetes among 7.4% of adults living in food-secure households and 10.2% of adults living in food-insecure households. The risk of clinical diabetes was ~50% higher among adults living in food-insecure households compared with adults living in food-secure households (crude P = 0.03; adjusted P = 0.09).

Among adults already diagnosed with disease, food insecurity was associated with inadequate control of diabetes (ARR 1.35; 95% CI, 1.05–1.74) but not hypertension or hyperlipidemia (**Table 3**). Mean HbA1c among adults with a self-reported diagnosis of diabetes (n = 428) was 7.4% among adults living in food-secure households and 8.1% among adults living in food-insecure households (crude P = 0.09, adjusted P = 0.1).

When we dichotomized food security at ≥6 affirmative responses (instead of ≥3 affirmative responses), indicating more severe food insecurity, the association between food insecurity and hypertension was no longer significant, although the prevalence was similar, suggesting an insufficient sample size.

TABLE 1 Characteristics of eligible, low-income NHANES participants[1]

	Secure, n = 3714	Insecure, n = 1380	P-value
Weighted %	75.5	24.5	
Female, *%*	53.9	52.3	0.3
Age, *mean y*	36.1	37.2	0.06
Self-reported race, *%*			0.001
White	53.4	47.3	
African American	17.1	17.7	
Latino	22.3	31.3	
Other/multiple	7.2	3.7	
Education, *%*			<0.001
<High school	32.1	45.4	
High schooldegree/GED[2]	30.3	27.5	
>High school degree	37.5	27.1	
Income, *%*			<0.001
0–50% of the FPL	15.3	19.3	
>50–100%	25.2	35.9	
>100–130%	30.4	31.1	
>130–200%	29.1	13.8	
Health insurance, *%*			<0.001
None	38.1	46.4	
Private	18.5	25.2	
Public (Medicaid/Medicare)	43.3	28.3	
Time during last 12 mo without health insurance, *%*	13.6	17.9	0.09
Parity (women only), *mean births*	3.8	3.9	0.5
BMI (women only), *mean kg/m^2*	28.7	29.7	0.02
BMI (men only), *mean kg/m^2*	27.6	27.0	0.1
Current daily smoker, *%*	27.2	35.0	<0.001
Occupational physical activity, *%*			0.05
Sits during day, doesnt walk much	22.1	23.2	
Stands or walks, but doesnt carry or lift heavy things often	50.3	47.7	
Lifts light loads, or climbs hills or stairs often	17.8	15.5	
Does heavy work or carries heavy loads	9.8	13.4	
Leisurely physical activity, *%*			
Vigorous physical activity (≥10 min in last 30 d)	32.1	27.4	0.06
Moderate physical activity (≥10 min in last 30 d)	41.3	40.4	0.7
Health status			<0.001
Excellent	19.4	8.2	
Very good	25.9	15.9	
Good	31.7	39.8	
Fair/Poor	23.1	36.2	

[1] The sample includes adults aged 18–65 y with incomes < 200% of the FPL participating in NHANES, 1999–2004. The weighted % uses the analytic weights to generate population-based estimates of prevalence for the included sample. Numbers may not sum to 100% due to rounding.

[2] GED, General Educational Diploma, certifies educational attainment equivalent to high school-level academic skills.

TABLE 2 Prevalence and crude and adjusted odds ratios for the association between food security and chronic disease among low-income NHANES participants

	Hypertension		Hyperlipidemia		Diabetes	
Assessment of diagnosis	Self-report, *n* = 4957	Clinical,[1] *n* = 4627	Self-report, *n* = 1930	Clinical,[2] *n* = 4559	Self-report, *n* = 5089	Clinical,[3] *n* = 2239
Food secure						
Unadjusted prevalence, %	20.2	18.6	33.3	19.8	6.8	7.4
Food insecure						
Unadjusted prevalence, %	24.6	22.4	43.3	21.7	8.3	10.2
CRR (95% CI)[4]	1.20 (1.05–1.38)	1.21 (1.03–1.42)	1.31 (1.10–1.56)	1.09 (0.90–1.33)	1.21 (0.92–1.59)	1.51 (1.04–2.19)
ARR (95% CI)[4]	1.20 (1.04–1.38)	1.21 (1.04–1.41)	1.30 (1.09–1.55)	1.09 (0.90–1.33)	1.19 (0.89–1.58)	1.48 (0.94–2.32)

[1] Clinical hypertension is defined as SBP >140 mm Hg, DBP >90 mm Hg, or taking antihypertensive medication.
[2] Clinical hyperlipidemia is defined as a total cholesterol ≥240 mg/dL (6.22 mmol/L), LDL cholesterol ≥160 mg/dL (4.14 mmol/L), or taking cholesterol-lowering medication.
[3] Clinical diabetes is defined as a fasting plasma glucose ≥126 mg/dL (6.99 mmol/L) or taking insulin and/or a hypoglycemic medication.
[4] Relative risk is for food-insecure adults compared with food-secure adults. CRR is adjusted for age, gender, and race/ethnicity. ARR is adjusted for age, gender, race/ethnicity, educational attainment, and income as both a continuous and an ordinal variable.

The association between food insecurity and both self-reported and clinical diabetes was highly significant. The unadjusted prevalence of self-reported diabetes was 6.7% among adults in food-secure households compared with 10.6% among those in food-insecure households (CRR 1.54; 95% CI, 1.07–2.24; ARR 1.52; 95% CI, 1.04–2.25). Unadjusted prevalence of clinical diabetes was 7.0% among adults in food-secure households compared with 15.9% among those in food-insecure households (CRR 2.52; 95% CI, 1.61–3.94; ARR 2.42; 95% CI, 1.44–4.08). The risk of clinical diabetes among those who did not self-report a diagnosis of diabetes (suggesting undiagnosed disease) appeared higher among adults living in food-insecure households than in food-secure households; however, this difference remained significant in the crude analysis only (CRR 2.73; 95% CI, 1.34–5.58; ARR 1.22; 95% CI, 0.55–2.72).

Our results were unchanged if we used the full 18-item scale to assess food insecurity rather than only the 10 items that reference adults in the household. Adding BMI as a covariate to the adjusted models did not change the association between food insecurity and hypertension or hyperlipidemia but made the association between food insecurity and clinical diabetes significant (CRR 1.63; 95% CI, 1.08–2.45; ARR 1.64; 95% CI, 1.02–2.65).

Discussion

Among nonelderly adults with household incomes <200% of the FPL, food insecurity is associated with clinical evidence of hypertension and diabetes. Evidence for an association between food insecurity and hyperlipidemia is weak and not significant. We previously reported an association between food insecurity and a self-reported diagnosis of diabetes (37). This study confirms these findings with clinical evidence of disease and suggests that adults living in food-insecure households may be more likely to underreport a diagnosis of diabetes.

A number of studies have reported cross-sectional associations between food insecurity and self-reported chronic disease, including heart disease, diabetes, hypertension, and general health status (5,11,12,38). In a sample of >2500 adults recruited from community sites in rural Ohio, Holben et al. (39) found no relationship between food insecurity and clinical indicators of disease, including hypertension, hyperlipidemia, and hyperglycemia, except for higher mean HbA1c levels among women. Our work in a population-based sample suggests that food insecurity may in fact be a risk factor for hypertension and diabetes among nonelderly adults.

Controlling for BMI did not attenuate the association between food insecurity and chronic disease, suggesting that these associations cannot be entirely attributed to an increased prevalence of obesity among food-insecure women. However, hypotheses to explain the association between food insecurity and obesity among women might also explain associations between food insecurity and the diet-sensitive chronic diseases we evaluated in this study (11,12,39). These hypotheses include food substitutions that emphasize relatively inexpensive but

TABLE 3 Association of food security status and inadequate disease control among low-income NHANES participants with a chronic disease diagnosis

	Definition of inadequate disease control		
	Hypertension	Hyperlipidemia	Diabetes
	SBP >140 mm Hg or DBP >90 mm Hg	Total cholesterol ≥5.18 mmol/L or LDL cholesterol ≥ 3.63 mmol/L	HbA1c >7%
Food secure			
Unadjusted prevalence, %	34.8	73.8	48.7
Food insecure			
Unadjusted prevalence, %	34.4	75.6	69.5
CRR (95% CI)[1]	1.00 (0.78–1.28)	1.03 (0.91–1.16)	1.39 (1.10–1.74)
ARR (95% CI)[1]	1.02 (0.79–1.32)	1.04 (0.92–1.17)	1.35 (1.05–1.74)

[1] Relative risk is for food-insecure adults compared with food-secure adults. CRR is adjusted for age, gender, and race/ethnicity. ARR is adjusted for age, gender, race/ethnicity, educational attainment, and income as both a continuous and an ordinal variable.

energy-dense foods (3,9,40), overconsumption during food plenty in expectation of future food shortages (41,42), and the "thrifty gene hypothesis," which suggests that it is adaptive to more efficiently accumulate fat when food is unpredictable (43). For example, the high sodium and low potassium content of highly processed foods, common in the diets of food-insecure adults, may increase risk of developing hypertension (44,45). We are aware of no studies demonstrating a pattern of increased sodium and decreased potassium intake associated with food insecurity (9,10), although qualitative work has suggested that food-insecure adults have difficulty affording low-sodium food alternatives (46).

Food insecurity also appears to be more strongly associated with diabetes than with hypertension, particularly at the most severe levels of food insecurity. There are a number of reasons why this stronger association may exist. First, diabetes may be more highly sensitive to diet, whereas hypertension and hyperlipidemia may be more highly sensitive to medication adherence. Second, an extension of the thrifty gene hypothesis suggests that peripheral insulin resistance, a precursor to diabetes, may be adaptive in association with food insecurity insofar as it allows for the preservation of muscle tissue during food restriction (47). Third, food insecurity is a highly stressful state, both emotionally and physiologically (48). The elevated cortisol associated with such stress is frequently linked to adiposity, particularly the visceral adiposity that is a strong risk factor for diabetes. Finally, replacement of dietary fruits and vegetables with relatively inexpensive carbohydrates, such as refined starches, increases dietary glycemic load and may increase the risk of developing diabetes in predisposed individuals (49–51).

We also showed an association between food insecurity and inadequate glycemic control among adults diagnosed with diabetes. This result might also be explained by the highly diet-sensitive nature of diabetes. In addition, self-management capacity may be limited by difficulty affording prescription medications in the face of extreme financial hardship. The depression, fatigue, and poor self-efficacy that often accompany food insecurity may exert additional effects (52). These self-management barriers may establish a cycle in which higher health care expenditures reduce food budgets; in turn, reduced food budgets decrease self-management capacity and poor self-management further increases medication needs, complication rates, and health care expenditures (53). This cycle may be exacerbated by the relative dearth of full-service grocery stores in many low-income, and particularly African-American, neighborhoods across the US (54,55). While healthy adults may be able to access transportation outside of the neighborhood, those with chronic disease may rely more heavily on corner stores and convenience stores for food purchases. Food available from corner and convenience stores tends to be of lower nutritional quality and more expensive, further limiting healthy food purchases (56).

There are 2 mechanisms whereby diabetes may increase the risk of food insecurity. First, the out-of-pocket health care expenditures associated with chronic disease may force adults into food insecurity (57). Second, health education provided to patients with chronic disease may increase awareness of dietary intake and ability to afford healthy foods, causing a perception of food insecurity that may not have been otherwise noted. These potential explanations for an effect-cause relationship deserve further study.

Our study was limited by small sample sizes for some variables, in particular for inadequate disease control among adults with self-reported disease. The Food Security Survey Module assesses food security at the household level, which may misclassify some individuals as food insecure if other household members are food insecure. However, most adults in a household are food insecure when the household is food insecure (6) and any misclassification biases our results toward the null hypothesis. A single blood pressure or blood sugar measurement is generally not adequate for a diagnosis of hypertension or diabetes. This misclassification is also likely to bias our results toward the null hypothesis. Differential rates of nonparticipation may have biased study results. We limited our sample to households with incomes <200% of the FPL. This sample restriction limits the generalizability of our findings to the U.S. population as a whole but provides important new information about the population generally at risk for food insecurity and increases the homogeneity of our sample with respect to unmeasured potential confounders (such as neighborhood environment). Finally, we used a self-reported measure of food insecurity, because no objective measures of food insecurity (as it is currently conceptualized) exist. However, use of a self-reported measure is appropriate because many of the effects of food insecurity may be mediated by an individual perception of the degree to which food budgets are inadequate. Despite these limitations, this study adds important population-based data to our understanding of the association between food insecurity and chronic disease.

A number of interventions designed to reduce the financial burden associated with healthy dietary intake have been successful at improving dietary intake. In 1 study, breast-feeding mothers receiving Special Supplemental Nutrition Program for Women, Infants, and Children benefits, a public health program providing food subsidies to pregnant and postpartum women and their children, were offered 10-dollar vouchers for fresh fruits and vegetables. Voucher redemption rates were ~90% and fruit/vegetable consumption increased (58,59). In another study, delivery of fresh fruits and vegetables to low-income, homebound elders, some of whom reported an inability to afford fruits and vegetables, effectively increased fruit and vegetable consumption (60,61). The long-term health implications of these interventions are unclear.

The Supplemental Nutrition Assistance Program (formerly the Food Stamp Program) has a well-established and highly successful infrastructure to reduce rates of food insecurity across the US. To the extent to which SNAP can encourage shifts in dietary intake toward healthy food alternatives, it might also have the potential to prevent the development of diet-sensitive chronic disease and improve health outcomes. Ongoing and planned modifications to the Supplemental Nutrition Assistance Program attempt to encourage these dietary shifts. Using physiologic measures to rigorously evaluate the health implications of these modifications will help determine the extent to which these programs may improve the health of the population by reducing the prevalence of hypertension and diabetes.

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All authors designed the research and wrote the paper. H.K.S. conducted the research and analyzed the data. H.K.S. had primary responsibility for final content. All authors read and approved the final manuscript.

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