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Food insecurity, poor diet, and metabolic measures: The roles of stress and cortisol

Dorothy T. Chiu[a], **Jordan E. Parker**[b], **Cameron R. Wiley**[c], **Elissa S. Epel**[d], **Barbara A. Laraia**[e], **Cindy W. Leung**[f], **A. Janet Tomiyama**[b,*]

[a]Osher Center for Integrative Health, University of California, San Francisco, 1545 Divisadero St 4th Floor, San Francisco, CA, 94115, USA

[b]Department of Psychology, University of California, Los Angeles, 1285 Psychology Building Box 951563, Los Angeles, CA, 90095, USA

[c]Department of Psychological Science, University of California, Irvine, 4201 Social & Behavioral Sciences Gateway, Irvine, CA, 92697, USA

[d]Weill Institute for Neurosciences, University of California, San Francisco, 675 18th Street, #5104, San Francisco, CA, 94107, USA

[e]School of Public Health, University of California, Berkeley, 2121 Berkeley Way, Room 5302, Berkeley, CA, 94720, USA

[f]Department of Nutrition, Harvard T.H. Chan School of Public Health, 665 Huntington Ave, Building 2, 3rd Floor, Boston, MA, 02115, USA

Abstract

Food insecurity is highly prevalent and linked to poorer diet and worse metabolic outcomes. Food insecurity can be stressful, and could elicit chronic psychological and physiological stress. In this study, we tested whether stress could be used to identify those at highest risk for worse diet and metabolic measures from food insecurity. Specifically, we hypothesized that cortisol (a physiological marker of stress) and perceived psychological stress would amplify the link between food insecurity and hyperpalatable food intake as well as metabolic measures. In a sample of 624 Black and White women aged 36–43 who participated in the NHLBI Growth and Health

*Corresponding author. tomiyama@psych.ucla.edu (A.J. Tomiyama).

Ethical statement

All procedures were approved by the Institutional Review Board of the University of California, Berkeley.

CRediT authorship contribution statement

Dorothy T. Chiu: Writing – original draft, Methodology, Formal analysis, Conceptualization. **Jordan E. Parker:** Writing – original draft, Formal analysis, Data curation. **Cameron R. Wiley:** Writing – original draft. **Elissa S. Epel:** Writing – review & editing, Supervision, Methodology, Funding acquisition, Conceptualization. **Barbara A. Laraia:** Writing – review & editing, Supervision, Methodology, Funding acquisition, Conceptualization. **Cindy W. Leung:** Writing – review & editing. **A. Janet Tomiyama:** Writing – original draft, Supervision, Project administration, Methodology, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.appet.2024.107294.

Study's midlife assessment, we assessed associations between food insecurity with hyperpalatable food intake (high fat + high sodium foods; high fat + high sugar foods; and high carbohydrate + high sodium foods), and metabolic measures (fasting glucose, insulin resistance, and waist circumference). We found that food insecurity was associated with higher levels of perceived stress (R^2 = 0.09), and greater intake of high fat + high sugar (hyperpalatable) foods (R^2 = 0.03). In those with higher cumulative cortisol (as indexed by hair cortisol), food insecurity was associated with higher levels of fasting glucose. Neither cortisol nor perceived stress moderated any other relationships, and neither variable functioned as a mediator in sensitivity analyses. Given these largely null findings, further research is needed to understand the role stress plays in the chronic health burdens of food insecurity.

Keywords

Food insecurity; Stress; Cortisol; Diet; Waist circumference; Insulin resistance; Glucose

1. Introduction

Over 36 million individuals in the United States experience food insecurity, defined by the USDA as situations in which the "availability of nutritionally adequate and safe foods or the ability to acquire acceptable foods in socially acceptable ways is limited or uncertain" (p.1560 (Andersen, 1990)). Food insecurity is associated with greater chronic disease risk. For example, nationally representative data indicate that food insecure individuals have over twice the prevalence of diabetes compared to those without food insecurity, even after controlling for socioeconomic status (Lehrer et al., 2016). However, it is unclear which individuals within the food insecure population are most susceptible to risk factors (e.g., high blood glucose, insulin resistance, waist circumference) that contribute to the development of metabolic disorders like diabetes.

Stress is a biopsychosocial factor that could help identify those at higher risk of the metabolic consequences of food insecurity. Seligman and Schillinger's model of food insecurity includes stress as factor that exacerbates the effects of food insecurity on obesity, hypertension, and diabetes (Seligman & Schillinger, 2010). "Stress" is a multidimensional construct, with both psychological and physiological conceptualizations. Here, we first focus on physiological stress markers (Wosu et al., 2015) because we theorize that they could be key to pinpointing those at greater risk for poorer metabolic outcomes of food insecurity. Physiological responses to stress can be captured in part by the endocrine hormone cortisol, which is the end product of the hypothalamic-pituitary-adrenal (HPA) axis (Dallman et al., 2000; Kyrou & Tsigos, 2009; Tsigos & Chrousos, 1994). When the hypothalamus detects stressful events, corticotropin-releasing hormone sends a signal to the pituitary, which then sends a signal via adrenocorticotropic hormone to the adrenal glands, ultimately causing the release and production of cortisol.

Cortisol is a hormone relevant to metabolic risk for several reasons. Since cortisol stimulates gluconeogenesis (i.e., the creation of glucose from non-glucose stores in the body), it is theorized to contribute directly to negative metabolic states like the metabolic syndrome

(Khani & Tayek, 2001). Related to this, cortisol has been associated with HbA1c (an index of average blood sugar levels) across three months, (Lehrer et al., 2016) and a study of 1258 participants found that higher concentrations of cortisol in hair was related to greater odds of having metabolic syndrome and higher HbA1c (Stalder et al., 2013). Moreover, results from the Multi-Ethnic Study of Atherosclerosis showed that women who had higher salivary cortisol levels across three days were more likely to have diabetes (Champaneri et al., 2012). Cortisol also stimulates deposition of fat in the abdominal region, (Bjorntorp, 2001; Bjorntorp and Rosmond, 2000a, 2000b) as seen prominently in the case of Cushing's Syndrome.

Cortisol can also drive mechanisms of metabolic risk, such as increased food consumption, and particularly the consumption of hyperpalatable foods (i.e., foods high in fats, sugar, sodium, and carbohydrates) (Fazzino et al., 2019; Yau & Potenza, 2013). For example, a robust non-human animal literature has demonstrated the causal effects of corticosterone (i.e., the rodent analog of cortisol) on eating of high-sugar, high-calorie food (Dallman et al., 2003). Research in humans has also indicated that cortisol can stimulate consumption of greater foods and greater hyperpalatable foods specifically. For example, a placebo-controlled administration study of exogenous cortisol in the form of dexamethasone showed that cortisol stimulated greater caloric intake (Tataranni et al., 1996). Additionally, endogenous levels of cortisol reactivity to a laboratory stress paradigm also predicted greater intake of sweet, high-fat food (Epel et al., 2001).

As these studies show, stress can also be categorized according to its timescale. Chronic stressors are those that are ongoing and continually disrupt daily life, (Crosswell & Lockwood, 2020) the effects of which can be captured via aggregated cortisol levels from hair. In contrast, acute stressors are events that are typically short-term and often event-based, the effects of which can be captured via stress reactivity measures like acute salivary cortisol responses. Because food insecurity is often chronic, (Insolera, 2023) here we focus on hair cortisol as our operationalization of chronic stress.

Therefore, the first objective of this study was to test whether chronic stress levels (indexed in hair) could be used to identify those at highest risk for unfavorable health correlates of food insecurity. We hypothesized that cortisol would amplify relationships between food insecurity and negative health correlates (see Fig. 1). Despite the importance of being able to identify—and thus target—those at greatest risk of the negative outcomes of experiencing food insecurity, very few investigations have tested cortisol as a modifier in this way. In fact, we could identify only one study, conducted in 52 Mexican American children, that found that higher hair cortisol levels modified the association between food insecurity and body mass index (BMI), such that only those children with high cortisol levels displayed a relationship between food insecurity and BMI (Distel et al., 2019). As BMI is a highly imperfect (Tomiyama et al., 2016; American Medical Assocation) and even problematic (Strings, 2019) indicator of metabolic health, here we focus on more direct and clinically relevant markers of metabolic health, in a study of adult non-pregnant women, where lean body and fat mass are less likely to be changing during periods of growth and development.

Given cortisol's dual role in promoting poor metabolic health directly and affecting pathways of metabolic risk via eating behavior, we examined two sets of measures. The first set captured dietary intake and comprised the three operationalizations of hyperpalatable food intake as identified by Fazzino and colleagues (Fazzino et al., 2019). The second set comprised blood glucose level, insulin resistance, and waist circumference.

Notably, "stress" can be conceptualized as a psychological phenomenon in addition to a physiological one. Even within psychological definitions of stress, there are different operationalizations such as perceptions of stress levels, experiencing stressful events, or appraisals of those stressful events (Crosswell & Lockwood, 2020). Here, we focus on perceived stress given the existing literature linking perceived stress levels to eating behavior (Hill et al., 2022; Kaiser et al., 2022; Tomiyama, 2019).

Research indicates that the experience of food insecurity can be psychologically stressful, as demonstrated in a meta-analysis that found food insecurity was significantly associated with psychological stress (OR = 1.34, 95% CI = 1.24, 1.44; note that the studies included in this meta-analysis measured perceived stress, psychological distress, anxiety, and other symptoms) (Pourmotabbed et al., 2020). This meta-analysis also found that the relationship between food insecurity and psychological stress was consistently observed across adult women and men, and across North America and Asia. Similarly, a Gallup Poll analysis of 149 countries similarly found a dose-response relationship between food insecurity and an index of negative mental well-being that included "worry" and "stress." (Jones, 2017).

Perceived stress is also linked to greater overall food intake and specifically the intake of unhealthy foods, as identified in a recent systematic review and meta-analysis (Hill et al., 2022). Individuals who identify as stress-overeaters have been found to gravitate toward "comfort foods" such as sweets, fast foods, and salty snacks, (Kaiser et al., 2022) which fit within the definition of hyperpalatable foods (Fazzino et al., 2019).

Because of these literatures linking perceived stress with both food insecurity and eating behavior, in secondary analyses we additionally tested the hypothesis that perceived stress would amplify relationships between food insecurity and the two sets of measures. Given that self-reported questionnaires are more feasible for wide-scale deployment than biomarker testing, we contend that examining perceived stress could yield valuable additional information.

Finally, in sensitivity analyses we (a) tested whether cortisol and perceived stress mediated (rather than moderated) relationships between food insecurity and the measures; and (b) tested an additional eating behavior variable—added sugars—that captured sugar from sugar-sweetened beverages in addition to hyperpalatable foods.

2. Methods

2.1. Sample and participants

Women (N = 624) aged 36–43 participated in the NHLBI Growth and Health Study's (NGHS) mid-life assessment. NGHS was designed to understand cardiometabolic risk

factors, and recruited participants from three sites (Cincinnati, OH; Richmond, CA; and Washington, D.C.) that were chosen to minimize socioeconomic differences between Black and White participants. Two sets of eligibility criteria were used: one assessed at baseline, when the participants were 9/10 years old, and one at the mid-life assessment point. The baseline criteria were: age within 2 weeks of 9 or 10 years at first clinic visit, self-reported Black or White race, living with racially concordant parent/guardian, and having a parent/guardian consenting to provide information on household. The follow-up criteria were: being part of the Richmond, CA cohort of the original study, not pregnant, not incarcerated, and given birth/miscarried within three months. Overall, the sample comprised healthy women and rates of disease diagnoses were low: 6.83% with pre-diabetes, 5.69% with diabetes, 2.56% with any cancer, and 18.11% with hypertension.

2.2. Procedures

All procedures were approved by the Institutional Review Board of the University of California (UC), Berkeley. Participants completed a follow-up questionnaire, then underwent either an in-person home or clinic visit, or a distance protocol (for those living over 65 miles from UC Berkeley) within six months to complete additional questionnaires, anthropometric measurements, and/or provide biological samples including hair. Distant participants were mailed a study package including anthropometric measurement tools, a 3-day food record, applicable surveys and a Visa gift card. During a phone call, study staff reviewed the package contents, verbally assisted with anthropometric measurements, and also provided training on how to complete the food recall. For anthropometric measures, trained staff followed NHANES protocols (Centers for Disease Control and Prevention, 2007). Distant participants utilized the NHANES-aligned tools and printed instructions they were sent to conduct self-measurements while staff provided real-time verbal instructions over the phone. All measures were taken three times. Participants provided fasting blood samples at their local LabCorp facility. For the food recall, staff helped participants select three non-consecutive days (two weekdays and one weekend day) to record and training covered topics such as what type of information and level of detail to report. Participants were instructed to mail the records back to the study center after completion. When received, trained study staff reviewed the records with participants over the phone and then entered diet data in the 2018 University of Minnesota Nutrition Coordinating Center's Nutrition Data System for Research (NDSR) software (University of Minnesota Nutrition Coordinating Center, 2018). Foods not found in the NDSR database were substituted for in consultation with registered dietitians. More information on study protocols can be found elsewhere (Laraia et al., 2023).

2.3. Measures

2.3.1. Food insecurity—The USDA Household Food Security Survey Module (United States Department of Agriculture, 2012) operationalizes the aforementioned food security definition by Andersen (Andersen, 1990) and was used to assess food security status over the past year. The Module comprises 18 items (sample items: *"The food that (I/we) bought just didn't last, and (I/we) didn't have money to get more"; "(I/we) couldn't afford to eat balanced meals", "(I/we) relied on only a few kinds of low-cost food to feed (my/our) (child/the children) because (I was/we were) running out of money to buy food."*). Questions referencing the whole household as well as adults vs. children separately were included

and Module reliability (α′s ranging from 0.86 to 0.93) and validity were evaluated and deemed acceptable during its development (United States Department of Agriculture, 1997). Affirmative response options (e.g., "yes," "often," "sometimes," "almost every month," and "some months but not every month") were scored with one point each and summed. Women were then classified into having "food security" (corresponding to households with high food security [scores of 0] and marginal food security [scores of 1 or 2])) or "food insecurity" (corresponding to households with low and very low food security [scores 3 and above]) per USDA classification specifications (United States Department of Agriculture, 2012). Raw score sums were calculated from women that provided codable responses to all 18 items.

2.3.2. Cortisol—Cortisol was indexed in hair, which provides an aggregated estimate of cortisol levels for one month per centimeter of hair. Hair samples required trained study staff, and thus they were only possible to obtain from local participants (n = 340). The protocol instructed collection of about 3 cm of hair (about three months' worth of hair growth) cut from the back of the head (posterior vertex) as close to the scalp as possible. Adhering to the recommendations of Wosu et al., (Wosu et al., 2015) this corresponded to the 2.4 cm closest to the scalp for Black participants and 3 cm for White participants, so as to account for differential hair growth rates. Staff also considered guidance provided by Wright et al., (Wright et al., 2018) for hair sample collection from Black women that was tailored to length of hair: short hair (1–3 cm) or long (longer than 6 cm) and advised coordination with participants if they had hairstyles that might prevent sampling of natural hair. After collection, samples were stored and mailed in aluminum foil to the Behavioral Immunology and Endocrinology lab of Dr. Mark Laudenslager at the University of Colorado, Anschutz Medical Campus for assaying (Wosu et al., 2015). The Laudenslager lab is a Salimetrics Center of Excellence and participates in a quality control program run by the Society of Hair Testing. They have maintained inter- and intra-assay coefficients of variation under 11% and 4%, respectively. For processing, protocols mirrored prior work by this laboratory described in-depth elsewhere (Hagan et al., 2021; Hoffman et al., 2017; Russell et al., 2015). Briefly, samples were weighed, washed in isopropanol, reweighed, frozen, and ground. Powdered hair was extracted with methanol, pelleted, and assayed based on the hair weight and extraction volume. Cortisol levels were determined by following manufacturer's protocol for Salivary Cortisol Enzyme Immunoassay (EIA), a commercial high sensitivity EIA kit (Salimetrics LLC; State College, PA, USA). For additional quality assurance, women were asked questions about their hair care including hair washing frequency, use of chemical hair straighteners, perms, or steroid use in the past 3 months, and general use of products like conditioner, bleach, gel, or medications for scalp conditions per the literature (Wright et al., 2018). These factors are known to bias cortisol measurement and four participants were excluded based on these data. Cortisol was reported in pg/mg to correct for the weight of hair relative to assay extraction volume and values were natural-log transformed prior to analysis to correct for non-normality.

2.3.3. Perceived stress—Perceived psychological stress was measured using the 10-item Perceived Stress Scale (Cohen et al., 1983) and asked about appraisals of stress over the past month. A sample item is, *"In the last month, how often have you felt nervous and*

'stressed'?" answered on a scale of 0 (Never) to 4 (Very Often). A sum score was calculated, and Cronbach's alpha for this measure was 0.88 in this sample.

2.3.4. Hyperpalatable food intake—Hyperpalatable food intake was calculated from dietary intakes assessed from three-day (non-consecutive) food records women self-completed after training by project staff. We used the operationalization of hyperpalatable foods generated by Fazzino and colleagues, (Fazzino et al., 2019) who identified three subtypes in addition to overall intake: (1) high fat + high sodium foods (meats and meal-based foods like hot dogs, bacon, and pizza), (2) high fat + high sugar foods (dessert-type foods like cake, ice cream, and brownies), and (3) high carbohydrate + high sodium foods (bread and snack-type foods like crackers, pretzels, biscuits, and popcorn). After trained study staff entered food records into the University of Minnesota Nutrition Coordinating Center's Nutrition Data System for Research (NDSR) system, mean daily intakes of corresponding food items to the hyperpalatable food categories were calculated and then summed among women with at least one day's food record to generate a mean "daily" intake of servings of hyperpalatable foods overall and by subtype. Supplemental Table 1 displays the specific NDSR foods summed to create the subtypes.

2.3.5. Metabolic measures—Blood draws were conducted for the measurement of fasting glucose and insulin concentrations. Insulin resistance was determined by the homeostatic model assessment formula for insulin resistance (HOMA-IR): glucose (mg/dL) x insulin (uU/mL)/405. Waist circumference was measured using the protocol from the National Health and Nutrition Examination Survey, (Centers for Disease Control and Prevention, 2007) which measures the circumference at the iliac crest.

2.3.6. Demographic measures/covariates—Black or White race (dummy coded) was self-reported by the participants' caregivers at the baseline assessment. At the mid-life follow-up, participants reported annual income by selecting from one of fifteen specified income ranges binned into $10,000 increments (e.g., $20,000–20,999, $30,000 - $30,999, etc.) ranging from less than $20,000 to $150,000 or more. Participants also reported household size, which we used to generate household size-adjusted annual income, defined as the total household income divided by the square root of the number of people living in the house. Finally, participants also reported their highest educational attainment which was dummy coded into three nominal categories: high school or less, some college, and college or advanced degrees.

2.3.7. Added sugars (sensitivity analyses)—Mean daily added sugar intake was calculated across valid food records as provided in NDSR output. Added sugars in NDSR are defined as total sugars added to foods (e.g., as syrups and sugars) during food preparation and commercial food processing and does not include mono- and disaccharides naturally occurring in foods (University of Minnesota Nutrition Coordinating Center, 2018).

2.4. Analytic plan

All hypotheses were formulated after data collection but prior to data analysis. The following data analytic plan was also pre-specified prior to data analysis. However, the analytic plan was not pre-registered.

2.4.1. Data handling procedures—To address missing data, we leveraged a Bayesian model-based imputation and inferential approach using the Blimp 3 application. (Enders and Keller) This model-based approach fills in (imputes) missing data that is conditional on the observed data, logically compatible with the target inference, and is particularly well-suited for models with interactive (moderation models) and non-linear effects (mediation models), (Enders et al., 2020) based on a conditionally missing at random assumption. Literature demonstrates that this assumption is associated with substantial tolerance for missingness, with studies suggesting that at least 50% (Dong & Peng, 2013; Enders, 2010) and even up to 90% missingness (Madley-Dowd et al., 2019) still results in unbiased estimates. Here, missingness ranged from 0% to 53.69%.

Each model of interest received a specific analysis-specific approach to estimate parameters of interest and fill in missing values, where necessary. For each model, we also leveraged an inclusive analysis strategy which includes additional auxiliary variables that are correlated with the analysis variables or their missingness (Collins et al., 2001). Specifically, for each analysis variable, we identified whether additional variables contained in the dataset (1) predicted an analysis variable, or (2) were correlated with the residuals of the model of interest fit on complete data. Using the Collins typology, (Collins et al., 2001) we identified relevant auxiliary variables for each analysis variable which were then entered into the imputation as additional model wherein they were predicted by all focal analysis variables.

Model convergence was verified using the potential scale reduction factor diagnostics, (Gelman & Rubin, 1992) based on standard recommendations in the missing data literature. For each imputation, we began with a default of 20,000 burn-in cycles (the trial period during which the imputation reaches a stable point) followed by 10,000 iterations to then estimate each parameter across two Markov Chain Monte Carlo chains. This algorithm iteratively estimates the model parameters conditioned on the complete data, and then uses said parameter estimates to fill-in missing values. Across 10,000 iterations, these steps produce a distribution of estimates for each parameter, the center and spread of which can be interpreted as point estimates (medians) and measures of uncertainty (standard errors). Significance is determined by the absence of a null value of zero from the 95% credible interval. Of note, the use of a Bayesian inferential framework does not require adjustment for multiple comparisons as is practice in frequentist inference (Dienes, 2011). Our general imputation approach using Bayesian missing data handling aligns with and is further detailed in Parker et al. (Parker et al., 2022)

2.4.2. Moderation analyses—To test the hypothesis that hair cortisol (and perceived stress, in secondary analyses) moderates the association between food insecurity and each of the hyperpalatable food intake measures (total and by subcategory) as well as food security status and each of the metabolic measures (fasting blood glucose, insulin resistance, and

waist circumference), we fit seven multiple linear regression models. Each of the seven measures were predicted by food insecurity, natural log transformed cortisol (grand mean centered/average perceived stress), and their interaction, controlling for all covariates listed above. The key test of the hypothesis was the significance of the interaction term between food insecurity and hair cortisol/perceived stress. Since food insecurity was dummy-coded, the interaction term indicates whether the association between hair cortisol/perceived stress and each respective measure is significantly different across participants who had vs. did not have food insecurity. The hypothesis was supported if the 95% credible interval for the interaction term did not include zero. Moreover, where interaction terms were significant, we additionally report the conditional effect of hair cortisol/perceived stress on respective measures for each food insecurity group, separately.

2.4.3. Sensitivity analysis – mediation—In sensitivity analyses, we examined whether hair cortisol might mediate, rather than moderate, the association between food insecurity and hyperpalatable food intake and metabolic measures. Accordingly, models were specified such that hair cortisol was predicted by food insecurity status and all covariates (model for *M),* and each measure was predicted by food insecurity, hair cortisol, and all covariates (model for *Y*). The indirect effect was defined as the product of the effect of food insecurity on hair cortisol controlling for covariates in the model for M (a path) and the effect of hair cortisol on each measure in the model for Y (b path), controlling food insecurity and other covariates. The key test of the hypothesis was the significance of the indirect effect as well as its component paths. The hypothesis was supported if the 95% credible interval for the indirect effect or its component paths did not include a null value of zero. Bayesian estimation and inference surrounding the indirect effect was performed in accordance with prior studies (Parker et al., 2022, 2024) based on the guidelines from Yuan & MacKinnon (Yuan & MacKinnon, 2009). In parallel with the moderation analyses, we additionally estimated each mediation model with perceived stress in place of hair cortisol.

2.4.4. Outlier detection—Outlier detection was performed on a model-specific basis, using filled in datasets from each model-based imputation. We specifically estimated each model with and without the inclusion of cases with standardized residuals ≥ |3| and examined whether inference was robust to said process. Evidence for impact of outliers on inferential conclusion was evident for four models for which subset results are presented below and respective sample sizes are noted.

3. Results

Table 1 displays demographic and descriptive information of the sample obtained from pooled, imputed datasets. Missingness for all relevant study variables is also reported, calculated from our original dataset prior to imputation. In addition, 8.49% of the sample had missing food security status data. Zero-order correlation analysis revealed that cortisol levels and perceived stress levels were not significantly correlated ($r = -0.08$, 95% $CI = -0.19, 0.04$).

We first examined the relationship between food insecurity and (1) the hypothesized moderators (cortisol, perceived stress) as well as (2) the measures of diet and metabolic

health. Table 2 displays the results of these adjusted multivariable linear models. Greater food insecurity was significantly related to greater perceived stress but not cortisol. Greater food insecurity was also related to higher intake of high-fat + high-sugar food intake.

We next tested our key hypothesis that cortisol would amplify relationships between food insecurity and diet and metabolic measures using adjusted multivariable linear models. These estimates are displayed in Table 3. Cortisol emerged as a modifier of the relationship between food insecurity and blood glucose. Follow-up conditional analysis revealed that the unstandardized coefficient for glucose was only significant for those with food insecurity ($b_{food\ insecure}$ = 12.43) compared to those without food insecurity ($b_{food\ secure}$ = 0.51). Perceived stress did not modify any relationships.

An initial set of sensitivity analyses tested whether cortisol or perceived stress might mediate the relationship between food insecurity and the diet and metabolic measures. Mediation analyses for both cortisol and perceived stress showed no significant indirect effects (see Table 4). However, individual paths emerged as significant. Higher cortisol levels were related to higher glucose levels, and food insecurity was related to greater perceived stress.

A second set of sensitivity analyses tested added sugars in the place of hyperpalatable food. These analyses mirrored the primary analyses; there was no main effect of food insecurity on added sugars, and no indication of moderation by either cortisol or perceived stress. The sensitivity analyses testing mediation showed a similar pattern, whereby for perceived stress, the *a* path was significant (unstandardized estimate 2.703, 95% CI = 1.180, 4.199).

4. Discussion

Due to the increased prevalence of biobehavioral risk factors and poor metabolic health outcomes among food insecure populations, it is important to understand the roles of physiological and psychological stress in this context to better understand which individuals are at the highest risk of disease. The current study sought to probe this area by examining if hair cortisol and self-reported perceived stress modified the association of food insecurity with negative dietary (i.e., hyperpalatable food intake) and metabolic (i.e., blood glucose, insulin resistance, waist circumference) measures in a sample of Black and White women. Chiefly, cortisol and perceived stress were not associated with one another, as seen in prior studies (Crosswell & Lockwood, 2020). Consequently, those with food insecurity had higher perceived stress levels but not higher cortisol levels.

Contrary to our primary hypotheses, cortisol did not amplify the effects of food insecurity on any measures with the exception of glucose levels. In the case of glucose, we found that cortisol amplified the effects of food insecurity on blood glucose levels. The conditional effects analyses revealed that the unstandardized estimate for the association between cortisol and glucose was over 24 times higher in those with food insecurity vs. those without. It did not appear that cortisol and perceived stress were functioning as mediators (rather than modifiers) of the relationship between food insecurity and the measures. However, mediation analyses showed that higher cortisol was associated with higher blood

glucose, and additionally confirmed earlier analyses linking food insecurity to higher perceived stress.

Prior work has indicated that food insecurity is associated with poorer diet (Jia et al., 2021; Larson & Story, 2011; Leung et al., 2014). However, other work does not find such an association (Gamba et al., 2016). The current study may offer a potential explanation: that food insecurity is exclusively linked with sugary, high-fat foods and not other subtypes of diet. Prior evidence underscores the particularly salient role of sweetness in eating behaviors and outcomes. For example, cross-species research by Berridge (Berridge, 2009) identified hedonic behavioral (e.g., lip-licking) and neurological activation responses unique to sweet-tasting stimuli. In humans, prior evidence has suggested that stress-related eating behaviors may be especially geared towards food choices high in fat and sugar (O'Connor et al., 2008; Rutters et al., 2008). Given that there are environmental stressors (e.g., low income, reduced food availability) that both characterize food insecurity and promote unhealthier food choices, (Gordon-Larsen, 2014; Leung et al., 2014) it is possible that higher food insecurity may encourage an increased consumption of specific types of foods, such as those high in sugar and fat.

Our finding that cortisol amplified the effects of food insecurity on glucose levels aligns with previous literature linking cortisol with metabolic outcomes and could provide context to the physiological health of food insecure individuals. Higher cortisol being linked with higher glucose is supported by mechanisms such as gluconeogenesis, where increased cortisol levels stimulate the production of glucose from precursors other than glycogen (Melkonian et al., 2023). Continued excess production of glucose from this process can lead to an increased risk of poorer metabolic outcomes (Khani & Tayek, 2001). These physiological connections may have particular relevance for food insecure populations.

Mediation models did not indicate that the perceived stress associated with food insecurity necessarily related to worse eating or metabolic measures. However, the stress of food insecurity could beget other negative outcomes not examined here. For example, stress is a known contributor to poor mental health, and begets increased risk of depression (Hammen, 2005) and anxiety (Daviu et al., 2019). Stress also increases risk for other physical health indicators, such as accelerated aging (Polsky et al., 2022).

We note the following limitations. We examined our research questions using a cross-sectional design, and thus causality cannot be inferred. For example, it is possible that reverse causation could be underlying our findings, as our previous work has found that unhealthy eating behaviors related to food insecurity (e.g., restricted caloric intake) can lead to higher perceived stress and cortisol output (Tomiyama et al., 2010). Furthermore, while we assessed chronic levels of stress via validated measures and indexed the past 3 months of cortisol via hair, using a longitudinal design would allow us to better capture the downstream effects of food insecurity on behavioral and biological mechanisms that drive metabolic health. Additionally, examining the role of physiological stress *reactivity* (rather than cumulative cortisol production) in the association of food insecurity with dietary and metabolic outcomes would likely provide important additional context to our research aims. For example, previous evidence suggests that increased hyperpalatable food intake in

women is associated with higher cortisol responses following an acute stressor, (Epel et al., 2001; Newman et al., 2007) while other research has found that higher cortisol reactivity is linked with higher food intake in individuals with (vs. without) obesity (Herhaus et al., 2020). Finally, cortisol production is multiply determined, with factors like smoking, alcohol use, sleep, exercise, and others playing a role (Fukuda & Morimoto, 2001). Stress, therefore, is not the only contributor to the cortisol levels observed here.

In terms of sample composition, we exclusively used women to address our research questions, as prior research has shown that women especially vulnerable to the negative dietary and metabolic outcomes associated with stress and food insecurity (Broussard, 2019; Groesz et al., 2012). However, extending our line of inquiry to men would allow for a better contextualization of our findings as well as the examination of group differences in the consequences of food insecurity between men and women. Despite these limitations, the current study addresses a critical gap in the literature by examining the moderating role of multiple indices of stress on the association of food insecurity with several key dietary and metabolic measures, all in a sample of Black and White women.

4.1. Conclusion

Our study is among the first to determine the role of both psychological and physiological stress in food insecure individuals. Although we found that food insecurity was related to greater perceived stress, overall, we found limited evidence of psychological and physiological stress amplifying the negative effects of food insecurity on dietary and metabolic measures in the current sample. However, cortisol appears to be especially relevant for glucose levels among individuals with food insecurity compared to those without food insecurity. Our inquiry has hopefully laid the groundwork for future research to utilize interdisciplinary methods to better understand the role of stress in the persistent health burdens of this underserved population.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Data availability

Data will be made available on request.

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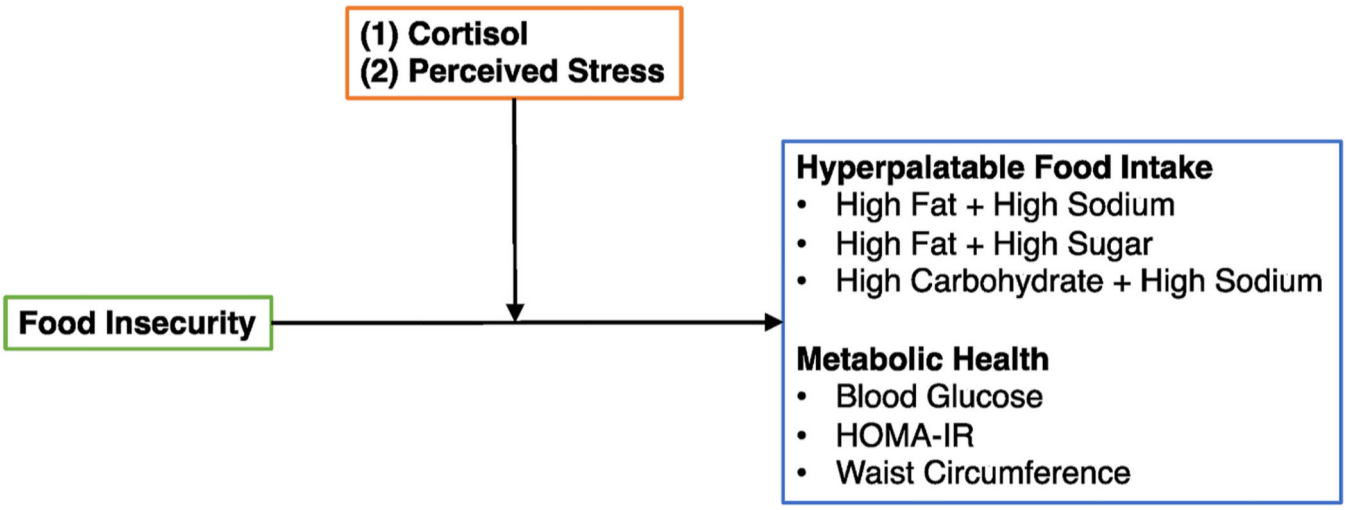

Fig. 1.
Hypothesized moderation model.

Table 1

Participant characteristics – mean (sd), proportions [n (%)] and missingness (%) (n = 624).

Characteristics	Food Secure (n = 501)	Food Insecure (n = 123)	*p*-value of Difference	Missing (%)
Race – Black, *n* (%)	228 (45.5%)	79 (64.2%)	**0.001**	0
Annual Household Income – adj. For household size, *mean* (SD)	$41,592.92 ($23,914.61)	$21,761.24 ($15,377.58)	<**0.0001**	7.05
Highest Educational Attainment, *n* (%)	93 (18.6%)	43 (35.0%)	<**0.0001**	0.16
HS or less Some college	187 (37.4%)	66 (53.7%)		
College graduate or more	222 (44.3%)	14 (11.4%)		
Stress Related Measure[e]				
Cortisol (ln), *mean (SD)*	2.71 (2.14)	2.94 (2.16)	0.377	53.69
Perceived Stress, *mean* (*SD*)	16.89 (6.71)	20.29 (6.67)	0.913	0.64
Dietary Intake, # of Servings (mean[sd])				
Total Hyperpalatable Foods	5.33 (3.70)	6.04 (3.55)	0.161	38.62
High fat + High sodium foods (e.g., meats/meal-based foods)	2.23 (1.82)	2.66 (1.90)	0.080	38.62
High fat + High sugar foods (e.g., desserts)	0.83 (1.31)	1.11 (1.48)	0.092	38.62
High carbohydrate + High sodium foods (e.g., bread, snacks)	2.26 (2.18)	2.38 (1.87)	0.689	38.62
Diabetes Measures (mean[sd])				
Blood Glucose (mg/ dL)	91.9 (18.8)	110.0 (49.6)	<**0.0001**	37.5
HOMA-IR	2.66 (3.61)	4.63 (5.21)	**0.0005**	37.66
Waist Circumference (inches)	39.2 (7.29)	42.1 (7.67)	**0.001**	18.27

Note. Summary values are calculated from imputed datasets. As such, some fractional values may be rounded. *p* values are from *t*-tests, except for race and educational attainment, where *p* values are from χ^2 tests using the D2 procedure (Heymans & Eekhout, 2019).

Table 2

Coefficient estimates of the associations between food security status and stress, dietary, and metabolic measures estimated from mulitivariable adjusted[a] linear models in a Black-White cohort of U.S. women at midlife (n = 624, unless otherwise indicated)[b].

Predictor: Food Insecurity	Unstandardized Coefficient (95% CI)[a]	Standardized Coefficient (95% CI)	R (Seligman & Schillinger, 2010)
Stress Measures			
Hair Cortisol, ln	0.01 (−0.51, 0.55)	0.00 (−0.11, 0.12)	0.13
Perceived Stress	**2.69 (1.20, 4.15)**	**0.16 (0.07, 0.24)**	**0.09**
Dietary Intake (# servings)			
Total Hyperpalatable Food Intake	0.74 (−0.28, 1.75)	0.08 (−0.03, 0.18)	0.03
High fat + High sodium foods (e.g., meats/ meal-based foods)	0.33 (−0.18, 0.84)	0.07 (−0.04, 0.18)	0.05
High fat + High sugar foods (e.g., desserts)	**0.37 (0.00, 0.73)**	**0.11 (0.00, 0.21)**	**0.03**
High carbohydrate + High sodium foods (e.g., bread, snacks)	0.03 (−0.55, 0.63)	0.01 (−0.10, 0.12)	0.02
Metabolic Measures			
Blood Glucose[c] (mg/dL)	1.23 (−2.03, 4.64)	0.04 (−0.07, 0.15)	0.06
HOMA-IR	0.90 (−0.30, 2.05)	0.09 (−0.03, 0.20)	0.07
Waist Circumference (inches)	1.56 (−0.15, 3.31)	0.08 (−0.01, 0.17)	0.09

[a] Adjusted for household size-adjusted annual income, race, and highest educational attainment.

[b] Bold reflects statistical significance at $p < .05$.

[c] Inference affected by outliers. Nine cases removed (n = 615).

Table 3

Coefficients of interaction terms between food insecurity status and a) cortisol and b) perceived stress in adjusted[a] multivariable linear models for hyperpalatable food intake and metabolic measures in a Black-White cohort of U.S. women at midlife (n = 624, unless otherwise indicated).[b] Conditional effects are displayed where interaction terms were significant.

	Unstandardized Coefficient (95% CI)	Standardized Coefficient (95% CI)	R^2
Food Insecurity x Hair Cortisol – ln, centered			
Dietary Intake (# servings)			
Total HPF Intake	0.29 (−0.34, 0.90)	0.06 (−0.08, 0.20)	0.05
High fat + High sodium foods (e.g., meats/meal-based foods)	0.01 (−0.25, 0.29)	0.01 (−0.11, 0.12)	0.06
High fat + High sugar foods (e.g., desserts)	0.18 (−0.07, 0.42)	0.11 (−0.04, 0.25)	0.05
High carbohydrate + High sodium foods (e.g., bread, snacks)	0.15 (−0.21, 0.50)	0.06 (−0.08, 0.19)	0.03
Metabolic Measures			
Blood Glucose	**11.91 (8.42, 15.38)**	**0.36 (0.25, 0.46)**	0.23
Conditional Effects:			
with Food Security	*0.50 (−1.19, 2.20)*	*0.03 (−0.08, 0.15)*	–
with Food Insecurity	***12.43 (9.36, 15.46)***	***0.86 (0.63, 1.02)***	–
HOMA-IR	0.18 (−0.40, 0.77)	0.04 (−0.08, 0.15)	0.08
Waist Circumference (inches)	−0.46 (−1.55, 0.68)	−0.05 (−0.17, 0.07)	0.10
Food Insecurity x Perceived Stress – centered			
Dietary Intake (# servings)			
Total HPF Intake	0.05 (−0.09, 0.19)	0.04 (−0.08, 0.17)	0.04
High fat + High sodium foods (e.g., meats/meal-based foods)	0.04 (−0.03, 0.11)	0.07 (−0.06, 0.19)	0.06
High fat + High sugar foods (e.g., desserts)	0.00 (−0.05, 0.05)	0.00 (−0.12, 0.13)	0.04
High carbohydrate + High sodium foods (e.g., bread, snacks)	0.00 (−0.09, 0.08)	0.00 (−0.13, 0.13)	0.02
Metabolic Measures			
Blood Glucose (mg/dL)	0.91 (−0.15, 1.97)	0.10 (−0.02, 0.22)	0.09
HOMA-IR[c]	0.02 (−0.07, 0.10)	0.03 (−0.09, 0.15)	0.09
Waist Circumference (inches)	0.10 (−0.14, 0.34)	0.04 (−0.06, 0.14)	0.10

[a] Adjusted for household size-adjusted annual income, race, and highest educational attainment.

[b] Bold reflects statistical significance at $p < .05$.

[c] Inference affected by outliers. Seven cases removed (n = 617).

Table 4

Adjusted[a] path estimates and indirect effects of food security status on hyperpalatable food intake and metabolic measures through cortisol and perceived stress, respectively, among Black and White women at midlife (n = 624)[b].

	Unstandardized Coefficient (95% CI)/Standardized Coefficient (95% CI)		Unstandardized Effect (95% CI)/ Standardized Effect (95% CI)
	a	**b**	**ab**
Cortisol, ln			
Dietary Intake (# servings)			
Total Hyperpalatable Foods[c]	−0.02 (−0.55, 0.50)/*−0.02 (−0.36, 0.33)*	0.07 (−0.18, 0.31)/*0.04 (−0.10, 0.17)*	−0.00 (−0.09, 0.08)/*−0.00 (−0.03, 0.03)*
High fat + High sodium foods	0.01 (−0.51, 0.53)/*0.00 (−0.34, 0.35)*	0.02 (−0.10, 0.15)/*0.02 (−0.10, 0.14)*	−0.00 (−0.04, 0.04)/*−0.00 (−0.03, 0.02)*
High fat + High sugar foods	0.01 (−0.53, 0.53)/*0.00 (−0.35, 0.35)*	0.05 (−0.07, 0.17)/*0.07 (−0.09, 0.24)*	−0.00 (−0.05, 0.05)/*−0.00 (−0.04, 0.04)*
High carbohydrate + High sodium foods	0.01 (−0.51, 0.54)/*0.01 (−0.34, 0.35)*	−0.08 (−0.26, 0.11)/*−0.07 (−0.22, 0.09)*	−0.00 (−0.07, 0.07)/*−0.00 (−0.04, 0.04)*
Metabolic Measures			
Blood Glucose[d] (mg/dL)	−0.07 (−0.60, 0.46)/*−0.05 (−0.40, 0.30)*	**0.98 (0.07, 1.86)/*0.15 (0.01, 0.28)***	−0.05 (−0.68, 0.54)/*−0.01 (−0.07, 0.05)*
HOMA-IR	0.01 (−0.53, 0.53)/*0.01 (−0.35, 0.35)*	0.01 (−0.24, 0.26)/*0.01 (−0.11, 0.12)*	0.00 (−0.07, 0.07)/*0.00 (−0.02, 0.02)*
Waist Circumference (inches)	−0.01 (−0.53, 0.52)/*−0.01 (−0.35, 0.34)*	0.20 (*−0.27, 0.67*)/*0.05 (−0.07, 0.16)*	−0.00 (−0.19, 0.18)/*−0.00 (−0.03, 0.03)*
Perceived Stress			
Dietary Intake (# servings)			
Total Hyperpalatable Foods	**2.71 (1.21, 4.19)/*0.49 (0.22, 0.75)***	−0.01 (−0.07, 0.05)/*−0.02 (−0.12, 0.09)*	−0.03 (−0.20, 0.13)/*−0.01 (−0.07, 0.04)*
High fat + High sodium foods	**2.70 (1.23, 4.17)/*0.48 (0.22, 0.75)***	−0.01 (−0.03, 0.02)/*−0.02 (−0.12, 0.08)*	−0.01 (−0.10, 0.06)/*−0.01 (−0.07, 0.04)*
High fat + High sugar foods	**2.81 (1.34, 4.32)/*0.51 (0.24, 0.78)***	−0.01 (−0.03, 0.01)/*−0.06 (−0.16, 0.05)*	−0.03 (−0.10, 0.03)/*−0.03 (−0.09, 0.02)*
High carbohydrate + High sodium foods	**2.71 (1.23, 4.17)/*0.48 (0.22, 0.75)***	0.08 (−0.03, 0.04)/*0.02 (−0.09, 0.13)*	0.02 (−0.08, 0.12)/*0.01 (−0.04, 0.07)*
Metabolic Measures			
Blood Glucose (mg/dL)	**2.69 (1.18, 4.16)/*0.48 (0.21, 0.74)***	0.09 (−0.34, 0.50)/*0.02 (−0.08, 0.12)*	0.21 (−0.96, 1.48)/*0.01 (−0.04, 0.06)*
HOMA-IR	**2.70 (1.20, 4.21)/*0.48 (0.21, 0.75)***	−0.02 (−0.08, 0.04)/*−0.03 (−0.13, 0.07)*	−0.04 (−0.23, 0.12)/*−0.01 (−0.07, 0.04)*
Waist Circumference (inches)	**2.68 (1.20, 4.17)/*0.48 (0.21, 0.74)***	0.01 (−0.09, 0.10)/*0.01 (−0.08, 0.09)*	0.01 (−0.25, 0.29)/*0.00 (−0.04, 0.05)*

Note: ***a*** path represents food insecurity→mediator (either cortisol or perceived stress); ***b*** path represents mediator→measure; ***ab*** path represents the indirect effect of food insecurity on measures through the mediator.

[a] Adjusted for household size-adjusted annual income, race, and highest educational attainment.

[b] Bold reflects statistical significance.

[c] Inference affected by outliers. Four cases removed (n = 620).

[d] Inference affected by outliers. Nine cases removed (n = 615).