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Food Insecurity Is Associated With Inflammation Among Women Living With HIV

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Background. Chronic inflammation is associated with AIDS-defining and non-AIDS–defining conditions. Limited research has considered how food insecurity influences chronic inflammation among people living with human immunodeficiency virus (HIV). We examined whether food insecurity was associated with higher levels of inflammation among women living with HIV (WWH) in the United States.

Methods. We analyzed cross-sectional data collected in 2015 from 421 participants on antiretroviral therapy from the Women's Interagency HIV Study. The exposure was any food insecurity. The outcome was inflammation, measured by proinflammatory cytokine interleukin-6 (IL-6) and tumor necroses factor receptor 1 (TNFR1) levels. We conducted multivariable linear regressions, adjusting for sociodemographic, clinical, and nutritional factors.

Results. Nearly one-third of participants (31%) were food insecure and 79% were virally suppressed (<20 copies/mL). In adjusted analyses, food insecurity was associated with 1.23 times the level of IL-6 (95% confidence interval [CI], 1.06–1.44) and 1.13 times the level of TNFR1 (95% CI, 1.05–1.21). Findings did not differ by HIV control (virally suppressed with CD4 counts ≥500 cells/mm^3 or not) in adjusted stratified analyses.

Conclusion. Food insecurity was associated with elevated inflammation among WWH regardless of HIV control. Findings support the need for programs that address food insecurity among WWH.

Keywords. food insecurity; chronic inflammation; HIV; inflammatory cytokines; women living with HIV.

Chronic inflammation among people living with human immunodeficiency virus (PWH) is associated with AIDS-defining conditions, including opportunistic infections and human immunodeficiency virus (HIV)-related morbidity and mortality, as well as non-AIDS–defining conditions such as cardiovascular disease [1–3]. In PWH, persistent immune activation and inflammation leads to a vicious cycle where proinflammatory cytokines promote further T-cell activation, which, in turn, leads to further HIV replication. Eventually this cycle contributes to increased T-cell apoptosis and the eventual exhaustion of the immune system in PWH [1]. Proinflammatory cytokines interleukin-6 (IL-6) and tumor necrosis factor receptor 1 (TNFR1) have been shown to be intricately linked to HIV infection [1, 4–6]. Both IL-6 and TNFR1 are secreted as a result of immune activation in HIV infection, and PWH have significantly higher plasma levels of these inflammatory markers compared to uninfected individuals, even in the context of viral suppression [1, 4–6]. High levels of IL-6 and TNFR1 have been associated with HIV progression and non-AIDS–defining conditions among PWH [5–7].

Limited research has considered how social and structural factors may influence chronic inflammation among PWH. Food insecurity may be one such factor. Food insecurity refers to having limited or uncertain availability of nutritionally adequate and safe food, or the inability to procure food in socially acceptable ways [8], and is associated with increased morbidity [9, 10] and mortality [11, 12] among PWH. Evidence from the general population in the United States suggests that food insecurity is associated with elevated levels of C-reactive protein [13, 14], an inflammatory marker, which has been linked to chronic

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diseases such as cardiovascular disease [15]. To our knowledge, no study has examined the relationship between food insecurity and inflammation among PWH. This warrants particular attention because PWH are disproportionately affected by food insecurity in the United States; an estimated 50%–60% of PWH are considered food insecure [16, 17]. Women living with HIV (WWH) may be at even greater risk for food insecurity given that nationally, female-headed households experience significantly higher rates of food insecurity than households in general (30% vs 13%) [18]. This study aimed to examine whether food insecurity was associated with higher levels of proinflammatory cytokines IL-6 and TNFR1 among a sample of WWH in the United States.

METHODS

Study Design and Population

The Women's Interagency HIV Study (WIHS) is a large, multisite, prospective cohort study of WWH and demographically similar controls in the United States, established in 1993. Biological, clinical, demographic, and behavioral data are collected semiannually through interviews, physical exams, and laboratory tests. Participants provide signed informed consent at each visit and are compensated for participation in the study.

Beginning in 2013, the Food Insecurity Substudy added measures of food security and dietary intake to WIHS biannual interviews. The present study was a cross-sectional investigation of HIV-positive participants enrolled in the substudy from 10 sites across the United States (Bronx, NY; Brooklyn, NY; Washington, DC; San Francisco, CA; Chicago, IL; Chapel Hill, NC; Atlanta, GA; Miami, FL; Birmingham, AL; and Jackson, MI). Data were collected from April through September 2015. We identified 896 women who were eligible for inclusion in the analysis because they were HIV seropositive, on antiretroviral therapy (ART), had stored peripheral blood mononuclear cell and fasting plasma samples, as well as food security data available at the visit. To improve the homogeneity of the study sample, we excluded 475 women with: (1) confirmed cancer (n = 48); (2) self-reported autoimmune diseases (n = 60); or (3) active or inactive hepatitis B or C (n = 377) to keep the study sample limited to those with HIV monoinfection. The final study sample size was 421. This analytic sample is similar to the rest of the cohort in terms of age, race, and income, but they were less likely to use illicit drugs since the last visit (4% vs 11%) and less likely to be current cigarette smokers (31% vs 44%).

Laboratory Methods

Standard commercially available enzyme-linked immunosorbent assay (ELISA) kits were used to assess inflammation via proinflammatory cytokines IL-6 (R&D Systems, HS600B) and TNFR1 (R&D Systems, DRT100) at Columbia University's Irving Institute for Clinical and Translational Research, Biomarkers Core lab. All samples were stored in a −80°C freezer and assays were performed in triplicate with a positive and negative control on each ELISA plate.

Measures

Outcome variables were 2 measures of immune inflammation: proinflammatory cytokines, IL-6 and TNFR1. These variables were transformed using the natural logarithm to fulfill the assumption of a normal distribution necessary for linear regressions.

The exposure was household food insecurity over the past 6 months, assessed using the validated 18-item United States Department of Agriculture Household Food Security Survey Module (HFSSM) [8]. The HFSSM measures uncertainty about food supplies, insufficient diet quality, and insufficient food quantity. For households with 1 or more children, the raw scores for high, marginal, low, and very low food security are zero, 1–2, 3–7, and 8–18, respectively [8]. For households with no children the raw scores for high, marginal, low, and very low food security are zero, 1–2, 3–5, and 6–10, respectively [8]. A binary variable was created to capture any food insecurity (defined as marginal, low, and very low food security), compared to no food insecurity (ie, high food security). Cronbach alpha for the HFSSM in this sample was 0.91, indicating high internal consistency.

Control variables were selected based on prior research on factors associated with food insecurity and IL-6 and TNFR1. Control variables were age at visit (per 10 years), average annual household income (<$12 000, $12 001–$24 000, or ≥$24 001), race/ethnicity (non-Hispanic white, Hispanic, Black/African American, or other), education (≥ high school education or equivalent, compared to less than high school education), recent illicit substance use, and being a current smoker (vs not). Substance use was defined as self-reported cocaine, crack, heroin, methamphetamine, hallucinogens, club drugs, nonprescribed narcotics, or any other illicit recreational drugs, excluding any form of marijuana, in the last 6 months or since their last visit. Substance use is associated with increased inflammation [19] and food insecurity [20].

HIV-related clinical factors (viral load and CD4 count) and nutritional factors (body mass index [BMI], fat intake, sugar intake, red and processed meat intake, and fruit and vegetable intake) may mediate the relationship between food insecurity and inflammation, or they could confound this relationship. For example, viral load has been associated with both food insecurity [21] and inflammatory markers IL-6 and TNFR1 [5, 22], as has CD4 cell count [5, 11]. Nutritional factors such as BMI, fat, sugar, red and processed meat, and fruit and vegetable intake have also been associated with both food insecurity and clinical markers of inflammation [5, 23–26]. Because neither the HIV-related clinical factors nor the nutritional factors were significantly associated with both the exposure and outcomes in

this sample, they were included in our models as control variables. Viral load was a continuous measure (copies/mL), which was transformed using the logarithm base 10. CD4 count was also a continuous variable (cells/mm^3). BMI was a continuous measure in kg/m^2 (analyzed per every 3 kg/m^2 to facilitate interpretation). An adapted version of the multifactor screener from the 2000 National Health Interview Survey was used to measure intake of fat, sugar, red and processed meats, and fruits and vegetables [27]. All intake measures were continuous (analyzed per every 3 servings a day to facilitate interpretation). Daily servings of fat comprised the number of servings of red meat, processed meat (eg, hot dogs/cold cuts), dessert (eg, donuts/cakes/pastries), butter/margarine/full-fat salad dressing or mayonnaise, whole milk, and French fries. Daily servings of sugar measured sugar intake from 3 sources: sugar-sweetened beverages, desserts, and fruit juices. Daily servings of meat was a combined measure of red meat and processed meats. Daily servings of fruits and vegetables comprised the number of servings of fruit (not including juice) and vegetables or green salad.

Analysis

Summary statistics were analyzed on all study participants for all control variables. Separate bivariate linear regression models assessed the associations of food insecurity and control variables with the 2 outcome variables. Control variables were included in the multivariable model based on a priori knowledge of their potentially confounding relationship with food insecurity and clinical markers of inflammation, and if they were significant at the $P < .1$ level in the bivariate models. To assess multicollinearity, variance inflation factors were assessed for each model using a postestimation command in Stata. For the adjusted model, variance inflation factors were low, ranging from 1.3 to 1.4, indicating little to no multicollinearity. Given that the outcome variables were log transformed, the regression coefficients were exponentiated and interpreted as the relative difference in the outcome compared to the referent group. As an additional sensitivity analysis, we conducted the adjusted analysis stratified by HIV control; HIV control was defined as virally suppressed (<20 copies/mL) and CD4 ≥ 500 cells/mm^3.

This research was conducted as part of the WIHS and was approved by the Institutional Review Boards of all the WIHS study sites. All studies were conducted in accordance with the principles outlined in the Declaration of Helsinki.

RESULTS

Sample Characteristics

The median age of the study sample was 47 years (interquartile range [IQR], 40–52; Table 1). The majority of the study sample (n = 331, 79%) was Black/African American, and 71% (n = 298) had obtained a high school education or higher. Over half (n = 211, 52%) had an average annual household income of $12 000 or less and 31% (n = 129) reported being food insecure. The median BMI of the study sample was 31 (IQR, 27–39), which is in the obese range, and 31% (n = 130) were current smokers. Over three-quarters of the study sample (n = 327, 79%) was virally suppressed, and 70% (n = 292) had a CD4 count of greater than or equal to 500 cells/mm^3. Median IL-6 and TNFR1 values were 1.57 pg/mL (IQR, 1.02–2.64) and 730.99 pg/mL (IQR, 601.99–912.11), respectively.

Association Between Food Insecurity and Inflammation

In the unadjusted analyses (Table 2 and Table 3, column 2), any food insecurity was significantly associated with 1.33 times the level of IL-6 (95% confidence interval [CI], 1.14–1.55) and 1.14 times the level of TNFR1 (95% CI, 1.06–1.23). Among women reporting food insecurity, median IL-6 and TNFR1 values were 1.83 pg/mL (IQR, 1.26–3.05) and 765.52 pg/mL (IQR, 655.84–982.21), respectively. The median values of IL6 and TNFR1 among women who were food secure were 1.39 pg/mL (IQR, 0.93–2.48) and 718.16 pg/mL (IQR, 585.60–879.44), respectively. Women of Black/African American race/ethnicity (compared to non-Hispanic white) had significantly higher levels of IL-6, while women of Black, Hispanic, and other races/ethnicities had lower levels of TNFR1 compared to non-Hispanic whites. Compared to those who earned an average annual household income of $12 000 or less, those who earned between $12 001–$24 000 and $24 001 or more had lower IL-6. Earning an average household income of $24 001 or more was significantly associated with lower levels of TNFR1 (relative difference 0.92, 95% CI, 0.84–0.99). Greater viral load and BMI were also

Table 1. Background Characteristics of Women Living With HIV in the Women's Interagency HIV Study (N = 421)

Characteristic	Value
Any food insecurity, n (%)	129 (31)
Age, y, median (IQR)	47 (40–52)
Race, n (%)	
White	31 (7)
Hispanic	46 (11)
Black/African American	331 (79)
Other	12 (3)
Annual household income, n (%)	
≤$12 000	211 (52)
$12 001–$24 000	97 (24)
≥$24 001	102 (25)
High school education or more, n (%)	298 (71)
Any illicit substance use since last visit, n (%)	18 (4)
Current smoker, n (%)	130 (31)
Virally suppressed, n (%)	327 (79)
CD4 ≥500 cells/mm^3, n (%)	292 (70)
BMI kg/m^2, median (IQR)	31 (27–39)
Intake of high-fat foods, servings, median (IQR)	2 (1–3)
Intake of sugar, servings, median (IQR)	1 (0.4–2)
Intake of red and processed meats, servings, median (IQR)	3 (2–5)
Intake of fruit and vegetables, servings, median (IQR)	1 (0.8–2)

Abbreviation: IQR, interquartile range.

Table 2. Unadjusted and Adjusted Associations Between Food Insecurity and IL-6 Among Women Living with HIV in the Women's Interagency HIV Study (N = 421)

Characteristic	Unadjusted Relative Difference (95% CI)[a]	Adjusted Relative Difference (95% CI)[b]
Any food insecurity	1.33 (1.14–1.55)[e]	1.23 (1.06–1.44)[e]
Age at visit, per 10 years	1.08 (0.99–1.17)	1.15 (1.05–1.25)[d]
High school education or more	1.01 (0.86–1.18)	1.08 (0.92–1.26)
Race/ethnicity		
White	(reference)	(reference)
Hispanic	1.26 (0.89–1.79)	1.26 (0.91–1.75)
Black/African American	1.40 (1.06–1.85)[c]	1.22 (0.94–1.59)
Other	1.10 (0.67–1.83)	1.02 (0.62–1.68)
Annual household income		
≤$12 000	(reference)	(reference)
$12 001–$24 000	0.77 (0.64–0.92)[d]	0.79 (0.67–0.95)[c]
≥$24 001	0.74 (0.62–0.88)[d]	0.83 (0.69–0.99)[c]
Illicit substance use	0.93 (0.65–1.34)	0.99 (0.69–1.42)
Current smoker	1.06 (0.91–1.24)	0.05 (0.89–1.23)
Viral load, $\log_{10}$	1.12 (1.03–1.21)[d]	1.11 (1.02–1.19)[c]
CD4 cells/mm^3	1.00 (1.00–1.00)	…
BMI, per 3 kg/m^2	1.09 (1.07–1.11)[e]	1.09 (1.06–1.11)[e]
Intake of high-fat foods, servings per day, per 3	1.16 (1.03–1.30)[c]	1.14 (0.99–1.32)
Intake of sugar, servings per day, per 3	1.13 (0.98–1.31)	1.08 (0.89–1.29)
Intake of red and processed meat, servings per day, per 3	0.96 (0.89–1.03)	…
Intake of fruits and vegetables, servings per day, per 3	0.96 (0.77–1.17)	…

Abbreviations: BMI, body mass index; CI, confidence interval; HIV, human immunodeficiency virus; IL-6, interleukin-6.

[a]The natural logarithm of IL-6 was used to satisfy the assumption of a normal distribution. The relative differences are natural exponential e of the regression coefficients, and are interpreted as multiplicative factors.

[b]This multivariable linear regression model examined the relationship between food insecurity and IL-6 adjusting for age, education, race/ethnicity, annual household income, illicit substance use, current smoker, viral load, BMI, intake of high fat food, and intake of sugar.

[c]$P < .05$.

[d]$P < .01$.

[e]$P < .001$.

associated with significantly higher IL-6 and TNFR1. Intakes of sugar, red and processed meat, and fruit and vegetables were not significantly associated with either inflammatory marker.

In the multivariable models (Table 2 and Table 3, column 3), food insecurity was significantly associated with 1.23 times the level of IL-6 (95% CI, 1.06–1.44) and 1.13 times the level of TNFR1 (95% CI, 1.05–1.21).

Age was significantly associated with higher levels of IL-6 (relative difference 1.15; 95% CI, 1.05–1.25) and TNFR1 (relative difference 1.05; 95% CI, 1.01–1.09). Women of Hispanic, Black/African-American, and other race/ethnicity all had significantly lower TNFR1 levels compared to non-Hispanic whites. Earning an average annual household income of $12 001–$24 000 and $24 001 or more remained associated with lower IL-6. Being a current smoker was significantly associated with 1.10 times the level of TNFR1 (95% CI, 1.02–1.19). Viral load was significantly associated with higher IL-6 and TNFR1, as was BMI. In the adjusted sensitivity analysis (Table 4), food insecurity remained associated with higher IL-6 and TNFR1, regardless of HIV control, and measures of association did not change substantively.

DISCUSSION

In this diverse sample of WWH on ART in the United States, food insecurity was associated with elevations in inflammatory markers IL-6 and TNFR1. Given that inflammation is associated with a number of negative health outcomes including HIV-related morbidity and mortality [1], our findings bolster the rationale for addressing food insecurity among PWH.

Food insecurity may influence inflammation in PWH through several paths. Food insecurity could lead to increased inflammation through poor HIV viral control. Food insecurity has been identified as a barrier to antiretroviral adherence [28] and ultimately HIV control [10, 21, 29]. PWH who are food insecure may avoid taking ART when they do not have enough food to eat [28]. This is due to fears or actual experiences of acute hunger while on ART without access to a nutritious diet, or increased side effects such as nausea and vomiting from taking ART on an empty stomach [28]. Additionally, in the context of strained resources, the need for food might compete with resources needed to obtain ART [28]. Likely as a consequence of limited ART adherence, food insecurity has been shown to be associated with poor virologic control [21]. Other research

Table 3. Unadjusted and Adjusted Associations Between Food Insecurity and TNFR1 Among Women Living With HIV in the Women's Interagency HIV Study (N = 421)

Characteristic	Unadjusted Relative Difference (95% CI)[a]	Adjusted Relative Difference (95% CI)[b]
Any food insecurity	1.14 (1.06–1.23)[e]	1.13 (1.05–1.21)[d]
Age at visit, per 10 years	1.03 (0.99–1.07)	1.05 (1.01–1.09)[c]
High school education or more	1.04 (0.96–1.12)	1.07 (0.99–1.15)
Race/ethnicity		
White	(reference)	(reference)
Hispanic	0.80 (0.68–0.94)[d]	0.78 (0.67–0.91)[d]
Black/African American	0.85 (0.75–0.98)[c]	0.81 (0.71–0.92)[e]
Other	0.78 (0.61–0.99)[c]	0.76 (0.59–0.97)[c]
Annual household income		
≤$12 000	(reference)	(reference)
$12 001–$24 000	1.05 (0.96–1.14)	1.08 (0.99–1.18)
≥$24 001	0.92 (0.84–0.99)[c]	0.94 (0.87–1.03)
Illicit substance use	1.03 (0.86–1.22)	0.99 (0.83–1.17)
Current smoker	1.07 (0.99–1.16)	1.10(1.02–1.19)[c]
Viral load, $\log_{10}$	1.05 (1.01–1.10)[d]	1.06 (1.02–1.10)[d]
CD4 cells/mm^3	1.00 (1.00–1.00)	…
BMI, per 3 kg/m^2	1.04 (1.03–1.05)[e]	1.04 (1.03–1.05)[e]
Intake of high-fat foods, servings per day, per 3	1.03 (0.97–1.09)	…
Intake of sugar, servings per day, per 3	1.02 (0.09–1.09)	…
Intake of red and processed meat, servings per day, per 3	1.02 (0.98–1.06)	…
Intake of fruits and vegetables, servings per day, per 3	0.98 (0.88–1.08)	…

Abbreviations: BMI, body mass index; CI, confidence interval; HIV, human immunodeficiency virus; TNFR1, tumor necroses factor receptor 1.

[a]The natural logarithm of TNFR1 was used to satisfy the assumption of a normal distribution. The relative differences are natural exponential e of the regression coefficients, and are interpreted as multiplicative factors.

[b]This multivariable linear regression model examined the relationship between food insecurity and TNFR1 adjusting for age, education, race/ethnicity, annual household income, illicit substance use, current smoker viral load, and BMI.

[c]$P < .05$.

[d]$P < .01$.

[e]$P < .001$.

has demonstrated an association between poor HIV control and elevated inflammatory markers among PWH [5].

The association between food insecurity and inflammatory markers persisted even after adjusting for viral suppression. Furthermore, in our adjusted sensitivity analysis, food insecurity remained associated with higher levels of inflammation among both HIV controlled, and noncontrolled participants. This suggests that ART adherence and poor viral suppression did not explain the relationship between food insecurity and inflammation in this sample. Longitudinal research, including longitudinal mediation analyses, is needed to further explore the role HIV control plays in the relationship between food insecurity and inflammation among WWH.

Food insecurity might also lead to increased inflammation through a nutritional path. Food insecurity has been associated with obesity [30] and diabetes [31], likely as a result of the need to consume cheaper, energy-dense foods on a limited budget [32], and binge-eating when food is available [33]. Higher BMI [5] and higher intake of fat, sugar, and red and processed meat are associated with higher levels of inflammation [23–26]. In contrast, consumption of fruits and vegetables is associated with lower inflammation [23–25]. The association between food insecurity persisted after adjusting for nutritional factors. Our measures of nutritional factors were limited by the use of the multifactor screener, which does not capture total energy intake by each food type, and BMI which does not capture micronutrient deficiencies. Further research using comprehensive nutritional assessments and longitudinal data could more fully assess the role that nutritional factors play in the relationship between food insecurity and inflammation.

Food insecurity is a powerful stressor [34, 35], and prior research suggests that stress is associated with elevated levels of inflammation [36]. Thus, it is possible that stress lies on the causal path between food insecurity and inflammation. We did not have available measures of perceived stress or biomarkers related to stress in this sample. Future studies should examine this potential path. Tryptophan catabolism may be another mechanism linking food insecurity to inflammation. The breakdown products of tryptophan, one of the essential amino acids mainly obtained from consumption of protein-rich foods, have been previously noted to contribute to immunosuppression and disease progression in chronic viral infections such as HIV [37]. We were unable to examine this potential path because our data lacked measures of tryptophan catabolism. Finally, evidence suggests that diet can

Table 4. Adjusted Associations Between Food Insecurity and IL-6 and TNFR1, by HIV Control

Characteristic	Relative Difference (95% CI)[a]			
	IL-6		TNFR1	
	HIV controlled[b] (n = 233)	Not HIV controlled (n = 167)	HIV controlled (n = 233)	Not HIV controlled (n = 167)
Any food insecurity	1.27[c] (1.01–1.58)	1.30[c] (1.03–1.63)	1.10 (0.99–1.21)	1.20[d] (1.60–1.37)
Age at visit, per 10 years	1.12 (1.00–1.26)	1.00 (0.87–1.14)	1.03 (0.98–1.08)	1.02 (0.95–1.10)
High school education or more	1.06 (0.84–1.35)	1.13 (0.89–1.43)	1.05 (0.95–1.17)	1.09 (0.96–1.25)
Race/ethnicity				
White	(Ref)	(Ref)	(Ref)	(Ref)
Hispanic	1.11 (0.68–1.81)	1.24 (0.75–2.05)	0.77[c] (0.62–0.95)	0.79 (0.60–1.04)
Black/African American	1.38 (0.94–2.04)	1.26 (0.83–1.91)	0.85 (0.72–1.01)	0.84 (0.66–1.05)
Other	1.78 (0.87–3.63)	0.63 (0.30–1.33)	0.89 (0.66–1.21)	0.67 (0.45–1.01)
Annual household income				
≤ $12 000	(Ref)	(Ref)	(Ref)	(Ref)
$12 001–$24 000	0.86 (0.67–1.09)	0.66[d] (0.49–0.88)	0.98 (0.89–1.09)	1.20[c] (1.02–1.42)
≥$24 001	0.71[d] (0.55–0.92)	0.92 (0.70–1.21)	0.89[c] (0.80–1.00)	0.96 (0.83–1.12)
Illicit substance use	0.92 (0.53–1.61)	0.95 (0.58–1.58)	0.90 (0.71–1.15)	1.07 (0.81–1.41)
Current smoker	0.94 (0.74–1.18)	1.14 (0.89–1.45)	1.05 (0.95–1.16)	1.11 (0.97–1.27)

Both multivariable linear regression models adjusted for all the variables included in the tables

Abbreviations: CI, confidence interval; HIV, human immunodeficiency virus; IL-6, interleukin-6; Ref, reference;TNFR1, tumor necroses factor receptor 1.

[a]The natural logarithm of IL-6 and TNFR1 was used to satisfy the assumption of a normal distribution. The relative differences are natural exponential e of the regression coefficients, and are interpreted as multiplicative factors.

[b]HIV control was defined as undetectable viral load and CD4 ≥ 500 cells/mm^3.

[c]$P < .05$.

[d]$P < .01$.

[e]$P < .001$.

affect the gut microbiota, and the gut microbiome in turn is an important determinant of systemic inflammation [38]. Future studies should assess whether food insecurity and diet intake affect the induction of the tryptophan catabolism pathway and the gut microbiota, and ultimately inflammation.

Findings presented in this study were based on cross-sectional associations, making us unable to specify the temporal relationship between food insecurity and inflammation. There is a need for longitudinal research to assess the relationship between food insecurity and inflammation over time, as well as potential mediators of this relationship. Research is also needed to explore the relationship between food insecurity and inflammation among men living with HIV. The generalizability of these findings are limited to women living with HIV in the United States without hepatitis B or C, cancer, or autoimmune diseases.

CONCLUSION

This study advances our knowledge about the role of food insecurity in inflammation among women living with HIV in the US context. Food insecurity was associated with elevated levels of inflammatory biomarkers IL-6 and TNFR1 in this sample of WWH on ART in the United States. Findings did not differ significantly by level HIV control, suggesting that ART adherence is not driving the relationship between food insecurity and inflammation, and that a biological path may explain our results. These findings reinforce the need for HIV care and treatment services to incorporate programming that addresses food insecurity. Findings from such research could help inform interventions seeking to address chronic inflammation among PWH, and ultimately the negative health outcomes associated with inflammation, including morbidity and mortality.

Notes

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