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Acculturative stress and inflammation among Chinese immigrant women

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Abstract

Objective—Among Chinese immigrant populations, increasing duration of US residence is associated with elevated risk for various chronic diseases. Although lifestyle changes following migration have been extensively studied in immigrant populations, less understood is the psychosocial impact of acculturative stress upon biological markers of health. Thus, the purpose of the present study is to examine associations between acculturative stress and inflammatory markers in a Chinese immigrant population.

Methods—Study participants (n=407 foreign-born Chinese American women) completed questionnaires assessing levels of stress, including acculturative stress and positive and negative life events in the past year. Participant height and weight were measured using standard protocols and blood samples were drawn for assessment of circulating serum levels of C-reactive protein (CRP) and soluble tumor necrosis factor receptor 2 (sTNFR2).

Results—Higher levels of acculturative stress were significantly associated with higher levels of CRP (B=0.07, 95% CI=0.01-0.13, p=0.031) and sTNFR2 (B=0.02, 95% CI=0.004-0.03, p=0.012), adjusting for age and body mass index (BMI). The latter association was no longer statistically significant when overall acculturation (i.e., identification with American culture) was included in the model. Life events were not associated with CRP or sTNFR2.

Conclusions—This study shows that acculturative stress is associated with inflammation markers in a Chinese immigrant population. These findings suggest that immigrant health trajectories may be adversely influenced by psychological distress associated with the demands of acculturation. Replication in other immigrant samples is needed to fully establish the biological correlates and clinical consequences of acculturation stress.

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Keywords

migration; acculturation; stress; Asian; inflammation

INTRODUCTION

Asian Americans are the fastest growing racial group in the United States (US) (1), and Chinese Americans represent the largest subgroup of US Asians. Over 76% of Chinese Americans are foreign-born (1), and it is now well-documented that with increasing duration of US residence comes elevated risk for various conditions, including increased risk for type 2 diabetes (2, 3), breast cancer (4, 5), and cardiovascular disease (6, 7). Though behavioral changes among immigrants (e.g., diet) have been extensively studied, less understood is the psychosocial impact of the immigration experience upon biomarkers of health. Indeed, in addition to the significant behavioral and lifestyle changes that ensue, Chinese immigrants are also likely to experience considerable stress (e.g., language barriers, separation from family, discrimination). And stress may arise from the acculturation process, which can involve significant cultural instability and social isolation as documented in various populations (8), including Chinese immigrants (9). Social isolation in particular has been linked to poor health outcomes (10, 11) and the up-regulation of genes that confer susceptibility to inflammatory disease (12, 13). In addition, empirical data suggest that acculturative stress can diminish health and well-being (8, 14).

The negative effects of acculturative stress on health might occur via inflammatory pathways. Extensive empirical data indicate that stress is associated with higher levels of inflammatory markers such as C-reactive protein (CRP) and TNF- α (15-19). Experimental studies have demonstrated that stressful experiences can directly stimulate production of pro-inflammatory cytokines (15, 20-22). Higher levels of inflammation, in turn, can contribute to increased risk for developing cardiovascular disease (23), diabetes (24-26), and selected cancers (27-29). Thus, the objective of the present study was to examine associations between psychosocial stress, specifically acculturative stress, and inflammatory markers in a sample of foreign-born Chinese American women. It was hypothesized that greater acculturative stress and negative life events would be associated with higher levels of inflammation in this immigrant population.

METHODS**Participants**

Participants for this analysis were drawn from a study of 436 healthy premenopausal foreign-born Chinese American women who were recruited between October, 2005, and April, 2008, to participate in a study of breast density. Inclusion criteria for the parent study included Chinese heritage, migration from Asia 20 years earlier, and being of mammography screening age. Exclusion criteria included: postmenopausal status; history of breast augmentation/reduction, prophylactic mastectomy, or any cancer except non-melanoma skin cancer; current pregnancy; and current breastfeeding or breastfeeding within the last 9 months. The study was approved by the Fox Chase Cancer Center Institutional

Review Board and all participants provided written informed consent. Of the 436 participants, 422 provided blood samples for assessment of inflammatory markers. One participant reported taking non-steroidal anti-inflammatory medications and was excluded from analysis. We also excluded 14 participants who had a CRP level > 10 mg/L or sTNFR2 level that was greater than 3 standard deviations above the mean (30), due to the possibility of other sources of inflammation such as that resulting from infection, leaving a sample of 407 women for analysis. Characteristics of study participants are presented in Table 1.

Procedures

Participants were recruited through Chinese community organizations, local medical practices, newspaper advertisements, and other contacts in the Chinese community in the Philadelphia region. Bilingual research staff administered interviews to obtain information on sociodemographic background, participant health behaviors, current medications, level of acculturation, and psychosocial stress.

All participants provided blood samples in the morning. Trained personnel collected 1-2 tubes of blood in 10 mL red top tubes. Samples were labeled with a study ID number and transported to the Biosample Repository Core Facility (BRCF) at Fox Chase Cancer Center for processing and storage. Blood samples were centrifuged, and aliquots of serum were stored at -80°C until analysis. At the time of blood draw, participant weight and standing height were measured using standard protocols (31) as previously reported (32). All measurements were taken and recorded in duplicate, with the mean value used in analyses.

Measures

Demographic characteristics including participants' age, education, length of US residence, marital status, smoking status, and current medications were assessed. Weight and height were used to compute body mass index (BMI), which is defined as an individual's weight (in kilograms) divided by the square of their height (in meters).

Acculturation was measured using an adapted, 11-item measure of the General Ethnicity Questionnaire-American (GEQ-A) version (33), which has demonstrated high validity and reliability in prior studies of Chinese Americans (34-37). The GEQ-A assesses acculturation in various life domains (including language use and proficiency, social affiliation, cultural activities, and cultural pride) and provides an overall score of identification with American culture. All items on the scale are scored on a five-point Likert type scale, with higher scores representing greater endorsement of American culture. The mean of all items is used to quantify overall acculturation to American culture. In the present study, internal reliability was high with an alpha coefficient of 0.91.

Acculturative stress was assessed using the Migration-Acculturation Stressor Scale (MASS) (38). The MASS is a 22-item scale that measures the severity of various acculturative stressors, including cultural, social, and functional difficulties, and has been found to have good psychometric properties in US Chinese populations (9, 35, 37, 39). For each item, participants were asked if they had encountered that difficulty in the past year, with answers ranging from 1 (no) to 5 (very much). Examples of MASS items include difficulties with language, housing, unfamiliar environment, making friends, cultural differences, missing

family, missing their homeland, loneliness, and racial discrimination. Total level of acculturative stress was calculated by summing across all items (39). Internal reliability was high in the present study (Cronbach's $\alpha = 0.84$).

To assess non-migration specific life events that may affect study participants, we administered the Life Experiences Survey (LES) (40). The LES is a commonly used instrument that assesses the presence and impact of both positive and negative life experiences over the past year and has been shown to have good reliability and validity (40). Participants rated each life event on a 7-point scale ranging from -3 (extremely negative) to +3 (extremely positive). If an event did not occur, the item was coded as 0. Standard procedures (40, 41) were used to calculate scores based on events reported on the LES. Briefly, every event that occurred was coded as one "life change unit." Positive and negative life events were differentiated based on whether respondents reported a positive (1 to 3) or negative (-1 to -3) impact for the event, and then summed to compute the number of positive and negative events each. Positive and negative impact rating scores were calculated by summing the impact ratings of positive and negative events, respectively, with the absolute value of impact ratings used for negative events. Results using the number of positive and negative events were similar to results based on their respective impact rating scores; therefore, only results for impact rating scores are presented.

Inflammatory markers

Inflammatory markers measured in this study include C-reactive protein (CRP) and soluble tumor necrosis factor receptor 2 (sTNFR2). CRP is an acute phase protein that serves as a reliable marker of systemic inflammation (42). Due to the sensitivity of TNF- α to sample processing conditions (43), we assessed sTNFR2, one of the receptors through which TNF- α signals and whose expression is induced by TNF- α . sTNFR2 is a more stable protein than TNF in circulation and has served as a useful inflammatory marker in previous studies of diabetes risk (44) and breast cancer (45).

Serum levels of CRP and sTNFR2 were assessed in singleton using fluorescent-bead-based immunoassays on a BioPlex 200 Luminex system. Bead-based assays for CRP and sTNFR2 are commercially available (Millipore, Billerica, MA) and were performed following manufacturer's protocol. The sensitivity of the fluorescent CRP assay, with a lower limit of quantification (LoQ) of 0.0024 mg/L, is on par with automated high-sensitivity CRP assays used in clinical settings (46). The LoQ of the sTNFR2 assay was 84.5 pg/mL. None of the samples tested were below the LoQ for either marker. A randomly selected 10% of samples were re-assayed in both the same and separate batches to evaluate within and between-batch reproducibility of all assays. The intraclass correlation coefficient was 0.99 for CRP and 0.69 for sTNFR2.

Data analyses

Descriptive analyses were used to characterize the study measures. Because inflammatory markers were highly skewed, the data were log-transformed for analyses to normalize their distributions. Preliminary analyses were conducted to examine potential sociodemographic correlates of inflammatory markers using Pearson correlation or one-way analysis of

variance (ANOVA), depending upon the nature of the variable. Multivariable regression analysis was used to estimate associations of log-transformed inflammatory markers with psychosocial stress measures after adjustment for potential confounders (e.g., age, BMI) and level of acculturation (GEQ-A, length of US residence). Results are presented as unstandardized regression coefficients (B) with 95% confidence intervals (CI), along with standardized betas (β). Separate models were run for each inflammatory marker. Analyses were conducted using SPSS Statistics version 21.0 (IBM, Armonk, NY) and reported *p*-values correspond to two-tailed tests.

RESULTS

Participants were on average 44 years of age (see Table 1) and had lived in the US for a mean of 7.5 years. The majority of women was non-smoking (99.3%) and currently married (92.1%). Only 17% had obtained some college education, with the remainder reporting a high school education level (34.6%) or lower (48.2%). Average body mass index (BMI) was 23.4 kg/m².

Participants reported a mean of 1.12 negative life events (range = 0-20) and 0.51 positive life events (range = 0-8) in the 12-month period prior to the study. The mean impact rating score for negative life events was 2.12 compared with 0.94 for positive life events. The mean level of acculturative stress in this sample was 36.42 (SD = 10.63).

Demographic correlates of stress and inflammatory markers

As presented in Table 2, correlational analyses indicated that older age was associated with higher levels of CRP ($r = 0.17, p < 0.001$) and sTNFR2 ($r = 0.10, p = 0.044$). BMI was positively correlated with higher levels of CRP ($r = 0.28, p < 0.001$). Longer residence in the US was associated with lower levels of sTNFR2 ($r = -0.11, p = 0.024$). Greater acculturation, as measured by the GEQ-A, was significantly associated with positive event impact ratings ($r = 0.24, p < 0.001$) and negative event impact ratings ($r = 0.23, p < 0.001$), but not with either of the inflammatory markers.

With respect to the stress measures, higher levels of acculturative stress were associated with shorter length of US residence ($r = -0.35, p < 0.001$), and positively correlated with both positive ($r = 0.19, p < 0.001$) and negative life event impact ratings ($r = 0.36, p < 0.001$). Acculturative stress was also positively associated with CRP ($r = 0.11, p = 0.028$), but the association between acculturative stress and sTNFR2 did not reach statistical significance ($r = 0.10, p = 0.053$).

Negative event impact ratings were correlated with positive event impact ratings ($r = 0.27, p < 0.001$) and older age ($r = 0.11, p = 0.021$). Positive event impact ratings were associated with shorter length of US residence ($r = -0.11, p = 0.022$) and with higher levels of CRP ($r = 0.11, p = 0.026$).

Finally, analyses of variance revealed no significant differences in inflammatory markers by education level or marital status ($p > 0.14$). Thus, education and marital status were not included in subsequent analyses.

Associations between psychosocial stress variables and inflammatory markers

Based on bivariate analyses described above, age and BMI were included as covariates in regression models of stress, acculturation, and inflammatory markers. In multiple regression analyses (Table 3), acculturative stress was positively associated with higher levels of CRP ($B = 0.07$, 95% $CI = 0.01-0.13$, $p = 0.031$). Because the inflammatory markers were log transformed, this coefficient may also be interpreted in terms of percent change.

Specifically, every one-unit increase in acculturative stress was associated with a 7% increase in CRP. This association remained statistically significant after adjusting for the acculturation variables of GEQA score and length of US residence ($B = 0.08$, 95% $CI = 0.02-0.14$, $p = 0.014$). Thus, in fully adjusted models, every one unit increase in acculturative stress was associated with an 8% increase in CRP. Negative and positive life events were not significantly associated with CRP in the multivariable models.

Acculturative stress was also positively associated with higher levels of sTNFR2 ($B = 0.02$, 95% $CI = 0.004-0.03$, $p = 0.012$), such that every one unit increase in acculturative stress was associated with a 2% increase in sTNFR2, but this association was no longer statistically significant when acculturation was included in the model.

DISCUSSION

This is one of the first studies to demonstrate that greater acculturative stress is associated with higher levels of circulating inflammatory markers in a cohort of foreign-born women. While chronic stress and job stress have been extensively examined in relation to inflammatory markers (47-49), few studies have explored how stress associated with the immigration experience may be independently associated with biomarkers of health. Prior studies have documented substantial acculturative stress among immigrants (8, 9), but they have not routinely incorporated biomarkers that may have relevance for the increased disease risk commonly observed among immigrant populations.

One potential pathway that may underlie the association between acculturative stress and greater inflammation may be via depressive symptoms. Prior studies have reported that lower levels of acculturation (50) and higher levels of acculturative stress are linked to depressive symptoms (51, 52). Depressive symptoms, in turn, have been consistently associated with greater levels of inflammation (53-55), including studies in ethnic/racial minority subgroups, notably African Americans (56, 57). In light of studies suggesting that rates of depression are higher in older Chinese immigrants relative to older Americans of other ethnicities (58), further investigation of the associations among acculturative stress, depressive symptoms, and inflammatory markers is warranted in Chinese immigrant populations.

Another potential pathway to consider is diet. Although the relationship between stress and dietary patterns is complex, greater stress has been associated with unhealthy dietary behaviors, including increased fat intake (59-61), higher consumption of snack-type foods (62, 63), and lower vegetable intake (64). Poorer dietary patterns, in turn, have been associated with higher levels of inflammatory markers (65, 66). In light of prior findings that greater acculturative stress was associated with diets marked by higher percent energy from fat among less acculturated Chinese women (35), future studies should explore whether diet

serves as a behavioral mediator of the association between acculturative stress and inflammation in immigrant populations.

The findings from the present study are relevant in light of statistics documenting that chronic disease risk among Chinese immigrants rises to converge with or even exceed risk in US whites (3, 67-69). For example, in a large cohort study, diabetes prevalence was 12-13% in Chinese participants vs. 4-7% in white participants (70). Notably, this elevated risk of chronic disease is observed despite relatively low prevalence of obesity among Chinese Americans (68, 71, 72). Similarly, rates of breast cancer incidence are also relatively lower among Chinese women residing in China (73) and increase after immigration to the US (5, 69, 74). Although much of the increase in chronic disease risk has been attributed to lifestyle changes (e.g., diet), our findings suggest that acculturative stress is associated with inflammatory markers known to play a role in various chronic conditions, including diabetes (24-26, 44) and cancer (27, 28, 45). As a result, studies of immigrant health trajectories may need to consider not only behavioral and lifestyle changes, but also take into account the varied social experiences that immigrants encounter during the acculturation process, and how these experiences may affect health.

At present, it is difficult to determine whether certain aspects of acculturative stress are more closely associated with inflammatory markers relative to other components of acculturative stress. It is possible that the social isolation and loneliness that can accompany prolonged separation from family and friends may have a greater negative impact on health than other acculturation challenges, such as learning where to buy familiar foods and navigating an unfamiliar environment and culture. Indeed social stress has been reported to be reliably predictive of increased inflammation (75-77). Future research designed to disentangle the associations of inflammatory processes with specific subcomponents of acculturative stress may yield important insight for understanding immigrant health and offer potential opportunities for novel health-promoting interventions.

Contrary to our hypothesis, no association between life events and inflammatory markers was observed. The extant literature suggests that such associations are variably observed, with some studies reporting an association (78), whereas other studies have not (79). This variability may be due, in part, to the different measures used to characterize life events across studies and dissimilarities across study populations. In this sample of relatively low acculturated women, one possible explanation for the lack of association may be that the burden of acculturative stress (e.g., language difficulties, cultural and social isolation) is experienced daily and thus may have more of an impact than life events that are encountered less frequently. Indeed, other studies have noted that measures of daily stress are more closely related to immune outcomes (80) and clinical conditions (81) than measures of life events.

Interestingly, the association between acculturative stress and sTNFR2 was no longer statistically significant once acculturation, specifically length of US residence, was included in the model. Our data indicate that longer US residence was correlated with lower levels of acculturative stress, consistent with the notion that these types of stressors decrease over time. Longer US residence was also associated with lower levels of sTNFR2 and CRP,

thereby potentially attenuating the association between acculturative stress and inflammation. Other covariates in the regression model, such as BMI, also were consistently associated with both inflammatory markers, as would be expected based on extensive findings regarding obesity and inflammation (82).

Finally, it should be noted that overall levels of acculturative stress were modest in this sample of immigrant women. A closer examination of women's responses to individual items on the Migration-Acculturation Stress Scale reveals that women reported considerable difficulties with language, missing their native country, and missing their family members, but generally had few difficulties with other aspects of adjusting to their new environment, including food, housing, or climate. As prior studies have noted (83), greater length of time since immigration is generally associated with decreasing levels of acculturative stress. That the study participants had been residing in the US for, on average, 7 years may help to explain the relatively modest levels of acculturative stress in the present sample.

The present study has several limitations. First, the cross-sectional nature of the data precludes any causal inferences regarding the direction of the observed associations and limits our ability to determine cause and effect. Second, a single measure of inflammatory markers might not reflect inflammatory burden given intra-individual variability in circulating cytokine and CRP levels. Nevertheless, relative rankings of CRP levels appear to be maintained over time (84), and previous studies have found single measures of sTNFR2 and CRP to be predictive of various disease outcomes (27, 44, 85-91). Third, the use of convenience sampling in the community may have led to sampling bias. The present sample is comprised of relatively low acculturated female Chinese immigrants, potentially limiting the generalizability of findings to more highly acculturated immigrants and to male immigrants. A related concern may be selection bias, particularly if women experiencing high levels of acculturative or life event stress were more likely to forego study participation. Similarly, if women were motivated to participate in the study because others in their social network or community group had done so, this might lead to a more homogeneous sample in terms of sociodemographic, behavioral, and/or psychosocial similarities, thereby also decreasing generalizability of study findings. While future studies in diverse immigrant populations are needed to confirm the present findings, our cohort comprised entirely of foreign-born Chinese provides an extraordinary opportunity to begin to characterize psychobiologic pathways that may be contributing to immigrant health trajectories. As such, these findings represent a unique contribution to the literature on acculturative stress and inflammation in US Chinese immigrants.

In summary, our study suggests that acculturative stress is associated with higher levels of inflammation in an immigrant population that is undergoing significant health transition. These findings suggest that a greater characterization of the psychosocial impact of acculturation on clinically relevant biomarkers is warranted. Such findings could have implications for understanding how immigration to the US influences disease risk via myriad social, behavioral, and biologic factors, although replication of these results in other immigrant samples is needed. These and future findings are likely to yield significant insight regarding the complex pathways underlying the health trajectories observed among US

immigrants and will inform the development of future chronic disease prevention efforts in these unique and growing populations.

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References

1. Pew Research Center. The Rise of Asian Americans. 2012
2. Candib LM. Obesity and diabetes in vulnerable populations: reflection on proximal and distal causes. *Ann Fam Med*. 2007; 5:547–56. [PubMed: 18025493]
3. Misra A, Ganda OP. Migration and its impact on adiposity and type 2 diabetes. *Nutrition*. 2007; 23:696–708. [PubMed: 17679049]
4. Deapen D, Liu L, Perkins C, Bernstein L, Ross RK. Rapidly rising breast cancer incidence rates among Asian-American women. *International Journal of Cancer*. 2002; 99:747–50.
5. Ziegler RG, Hoover RN, Pike MC, Hildesheim A, Nomura AM, West DW, Wu-Williams AH, Kolonel LN, Horn-Ross PL, Rosenthal JF, Hyer MB. Migration patterns and breast cancer risk in Asian-American women. *Journal of the National Cancer Institute*. 1993; 85:1819–27. [PubMed: 8230262]
6. Dassanayake J, Gurrin L, Payne WR, Sundararajan V, Dharmage SC. Cardiovascular Disease Risk in Immigrants: What Is the Evidence and Where Are the Gaps? *Asia-Pacific Journal of Public Health*. 2011; 23:882–95. [PubMed: 21159699]
7. Lear SA, Humphries KH, Hage-Moussa S, Chockalingam A, Mancini GBJ. Immigration presents a potential increased risk for atherosclerosis. *Atherosclerosis*. 2009; 205:584–9. [PubMed: 19181317]
8. Berry JW, Kim U, Minde T, Mok D. Comparative studies of acculturative stress. *International Migration Review*. 1987:491–511.
9. Ying Y. Immigration satisfaction of Chinese Americans: An empirical examination. *Journal of Community Psychology*. 1996:3–16.
10. Cornwell EY, Waite LJ. Social disconnectedness, perceived isolation, and health among older adults. *J Health Soc Behav*. 2009; 50:31–48. [PubMed: 19413133]
11. Coyle CE, Dugan E. Social isolation, loneliness and health among older adults. *J Aging Health*. 2012; 24:1346–63. [PubMed: 23006425]
12. Cole SW. Social regulation of human gene expression: mechanisms and implications for public health. *Am J Public Health*. 2013; 103(Suppl 1):S84–92. [PubMed: 23927506]
13. Hawkley LC, Cole SW, Capitanio JP, Norman GJ, Cacioppo JT. Effects of social isolation on glucocorticoid regulation in social mammals. *Horm Behav*. 2012; 62:314–23. [PubMed: 22663934]
14. Chun KM, Chesla CA, Kwan CML. “So We Adapt Step by Step”: Acculturation experiences affecting diabetes management and perceived health for Chinese American immigrants. *Social Science & Medicine*. 2011; 72:256–64. [PubMed: 21147509]
15. Steptoe A, Hamer M, Chida Y. The effects of acute psychological stress on circulating inflammatory factors in humans: a review and meta-analysis. *Brain Behav Immun*. 2007; 21:901–12. [PubMed: 17475444]
16. McDade TW, Hawkley LC, Cacioppo JT. Psychosocial and behavioral predictors of inflammation in middle-aged and older adults: the Chicago health, aging, and social relations study. *Psychosom Med*. 2006; 68:376–81. [PubMed: 16738067]

17. Kiecolt-Glaser JK, McGuire L, Robles TF, Glaser R. Psychoneuroimmunology: psychological influences on immune function and health. *Journal of consulting and clinical psychology*. 2002; 70:537–47. [PubMed: 12090368]
18. Kiecolt-Glaser JK, Robles TF, Heffner KL, Loving TJ, Glaser R. Psycho-oncology and cancer: psychoneuroimmunology and cancer. *Ann Oncol*. 2002; 13(Suppl 4):165–9. [PubMed: 12401684]
19. Coussons-Read ME, Okun ML, Nettles CD. Psychosocial stress increases inflammatory markers and alters cytokine production across pregnancy. *Brain Behav Immun*. 2007; 21:343–50. [PubMed: 17029703]
20. Hamer M, Steptoe A. Association between physical fitness, parasympathetic control, and proinflammatory responses to mental stress. *Psychosom Med*. 2007; 69:660–6. [PubMed: 17724255]
21. Langhorst J, Cobelens PM, Kavelaars A, Heijnen CJ, Benson S, Rifaie N, Dobos GJ, Schedlowski M, Elsenbruch S. Stress-related peripheral neuroendocrine-immune interactions in women with ulcerative colitis. *Psychoneuroendocrinology*. 2007; 32:1086–96. [PubMed: 17933470]
22. Wirtz PH, von Kanel R, Emini L, Suter T, Fontana A, Ehlert U. Variations in anticipatory cognitive stress appraisal and differential proinflammatory cytokine expression in response to acute stress. *Brain Behav Immun*. 2007; 21:851–9. [PubMed: 17412556]
23. Wang TJ, Larson MG, Levy D, Benjamin EJ, Kupka MJ, Manning WJ, Clouse ME, D'Agostino RB, Wilson PWF, O'Donnell CJ. C-Reactive Protein Is Associated With Subclinical Epicardial Coronary Calcification in Men and Women: The Framingham Heart Study. *Circulation*. 2002; 106:1189–91. [PubMed: 12208790]
24. Marques-Vidal P, Bastardot F, von Kanel R, Paccaud F, Preisig M, Waeber G, Vollenweider P. Association between circulating cytokine levels, diabetes and insulin resistance in a population-based sample (CoLaus study). *Clin Endocrinol (Oxf)*. 2012
25. Mirza S, Hossain M, Mathews C, Martinez P, Pino P, Gay JL, Rentfro A, McCormick JB, Fisher-Hoch SP. Type 2-diabetes is associated with elevated levels of TNF-alpha, IL-6 and adiponectin and low levels of leptin in a population of Mexican Americans: a cross-sectional study. *Cytokine*. 2012; 57:136–42. [PubMed: 22035595]
26. Pradhan AD, Manson JE, Rifai N, Buring JE, Ridker PM. C-reactive protein, interleukin 6, and risk of developing type 2 diabetes mellitus. *JAMA*. 2001; 286:327–34. [PubMed: 11466099]
27. Il'yasova D, Colbert LH, Harris TB, Newman AB, Bauer DC, Satterfield S, Kritchevsky SB. Circulating levels of inflammatory markers and cancer risk in the health aging and body composition cohort. *Cancer Epidemiol Biomarkers Prev*. 2005; 14:2413–8. [PubMed: 16214925]
28. Siemes C, Visser LE, Coebergh JW, Splinter TA, Witteman JC, Uitterlinden AG, Hofman A, Pols HA, Stricker BH. C-reactive protein levels, variation in the C-reactive protein gene, and cancer risk: the Rotterdam Study. *Journal of Clinical Oncology*. 2006; 24:5216–22. [PubMed: 17114654]
29. Heikkila K, Harris R, Lowe G, Rumley A, Yarnell J, Gallacher J, Ben-Shlomo Y, Ebrahim S, Lawlor DA. Associations of circulating C-reactive protein and interleukin-6 with cancer risk: findings from two prospective cohorts and a meta-analysis. *Cancer Causes Control*. 2009; 20:15–26. [PubMed: 18704713]
30. Pearson TA, Mensah GA, Alexander RW, Anderson JL, Cannon RO 3rd, Criqui M, Fadl YY, Fortmann SP, Hong Y, Myers GL, Rifai N, Smith SC Jr, Taubert K, Tracy RP, Vinicor F. Centers for Disease C, Prevention, American Heart A. Markers of inflammation and cardiovascular disease: application to clinical and public health practice: A statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. *Circulation*. 2003; 107:499–511. [PubMed: 12551878]
31. Centers for Disease Control. cartographer NHANES Anthropometry and Physical Activity Monitor Procedures Manual. 2005
32. Tseng M, Byrne C. Adiposity, adult weight gain and mammographic breast density in US Chinese women. *International Journal of Cancer*. 2011; 128:418–25.
33. Tsai JL, Ying Y, Lee PA. The meaning of “Being Chinese” and “Being American”: Variation among Chinese American young adults. *Journal of Cross-Cultural Psychology*. 2000; 31:302–32.

34. Liu A, Berhane Z, Tseng M. Improved dietary variety and adequacy but lower dietary moderation with acculturation in Chinese women in the United States. *Journal of the American Dietetic Association*. 2010; 110:457–62. [PubMed: 20184998]
35. Tseng M, Fang CY. Stress is associated with unfavorable patterns of dietary intake among female Chinese immigrants. *Ann Behav Med*. 2011; 41:324–32. [PubMed: 21384248]
36. Tseng M, Fang CY. Socio-economic position and lower dietary moderation among Chinese immigrant women in the USA. *Public Health Nutr*. 2012; 15:415–23. [PubMed: 21806866]
37. Ying Y-W, Han M, Tseng M. Acculturation and post-migration stress in middle-aged Chinese immigrant women in Philadelphia: Variation between the Fujianese and the non-Fujianese women. *Journal of Human Behavior in the Social Environment*. 2012; 22:20–38. [PubMed: 22451743]
38. Ying Y-W. Variation in acculturative stressors over time: A study of Taiwanese students in the United States. *International Journal of Intercultural Relations*. 2005; 29:59–71.
39. Ying YW, Han M. The contribution of personality, acculturative stressors, and social affiliation to adjustment: A longitudinal study of Taiwanese students in the United States. *International Journal of Intercultural Relations*. 2006; 30:623–35.
40. Sarason IG, Johnson JH, Siegel JM. Assessing the impact of life changes: development of the Life Experiences Survey. *J Consult Clin Psychol*. 1978; 46:932–46. [PubMed: 701572]
41. Kornblith AB, Herndon JE 2nd, Zuckerman E, Viscoli CM, Horwitz RI, Cooper MR, Harris L, Tkaczuk KH, Perry MC, Budman D, Norton LC, Holland J. Social support as a buffer to the psychological impact of stressful life events in women with breast cancer. *Cancer*. 2001; 91:443–54. [PubMed: 11180093]
42. Gabay C, Kushner I. Acute-phase proteins and other systemic responses to inflammation. *New England Journal of Medicine*. 1999; 340:448–54. [PubMed: 9971870]
43. Tworoger SS, Hankinson SE. Collection, processing, and storage of biological samples in epidemiologic studies: sex hormones, carotenoids, inflammatory markers, and proteomics as examples. *Cancer Epidemiol Biomarkers Prev*. 2006; 15:1578–81. [PubMed: 16985015]
44. Fernandez-Real JM, Broch M, Ricart W, Casamitjana R, Gutierrez C, Vendrell J, Richart C. Plasma levels of the soluble fraction of tumor necrosis factor receptor 2 and insulin resistance. *Diabetes*. 1998; 47:1757–62. [PubMed: 9792545]
45. Gross AL, Newschaffer CJ, Hoffman-Bolton J, Rifai N, Visvanathan K. Adipocytokines, inflammation, and breast cancer risk in postmenopausal women: a prospective study. *Cancer Epidemiol Biomarkers Prev*. 2013; 22:1319–24. [PubMed: 23651666]
46. Roberts WL, Moulton L, Law TC, Farrow G, Cooper-Anderson M, Savory J, Rifai N. Evaluation of nine automated high-sensitivity C-reactive protein methods: implications for clinical and epidemiological applications. Part 2. *Clin Chem*. 2001; 47:418–25. [PubMed: 11238291]
47. Almadi T, Cathers I, Chow CM. Associations among work-related stress, cortisol, inflammation, and metabolic syndrome. *Psychophysiology*. 2013
48. Kunz-Ebrecht SR, Kirschbaum C, Steptoe A. Work stress, socioeconomic status and neuroendocrine activation over the working day. *Soc Sci Med*. 2004; 58:1523–30. [PubMed: 14759695]
49. Steptoe A, Cropley M, Griffith J, Kirschbaum C. Job strain and anger expression predict early morning elevations in salivary cortisol. *Psychosom Med*. 2000; 62:286–92. [PubMed: 10772410]
50. Swenson CJ, Baxter J, Shetterly SM, Scarbro SL, Hamman RF. Depressive symptoms in Hispanic and non-Hispanic White rural elderly: the San Luis Valley Health and Aging Study. *Am J Epidemiol*. 2000; 152:1048–55. [PubMed: 11117614]
51. Driscoll MW, Torres L. Acculturative stress and Latino depression: the mediating role of behavioral and cognitive resources. *Cultural diversity & ethnic minority psychology*. 2013; 19:373–82. [PubMed: 23914748]
52. Mui AC, Kang SY. Acculturation stress and depression among Asian immigrant elders. *Soc Work*. 2006; 51:243–55. [PubMed: 17076122]
53. Dinan TG. Inflammatory markers in depression. *Current opinion in psychiatry*. 2009; 22:32–6. [PubMed: 19122532]

54. Elovainio M, Keltikangas-Jarvinen L, Pulkki-Raback L, Kivimaki M, et al. Depressive symptoms and C-reactive protein: The Cardiovascular Risk in Young Finns Study. *Psychol Med*. 2006; 36:797–805. [PubMed: 16623962]
55. Howren MB, Lamkin DM, Suls J. Associations of depression with C-reactive protein, IL-1, and IL-6: a meta-analysis. *Psychosom Med*. 2009; 71:171–86. [PubMed: 19188531]
56. Cassidy-Bushrow AE, Peters RM, Johnson DA, Templin TN. Association of depressive symptoms with inflammatory biomarkers among pregnant African-American women. *Journal of reproductive immunology*. 2012; 94:202–9. [PubMed: 22386525]
57. Deverts DJ, Cohen S, DiLillo VG, Lewis CE, Kiefe C, Whooley M, Matthews KA. Depressive symptoms, race, and circulating C-reactive protein: the Coronary Artery Risk Development in Young Adults (CARDIA) study. *Psychosom Med*. 2010; 72:734–41. [PubMed: 20668285]
58. Mui A, Lee E. Correlates of Depression Among Chinese and Korean Immigrant Elders in the United States. *Ageing International*. 2013:1–15.
59. Hellerstedt WL, Jeffery RW. The association of job strain and health behaviours in men and women. *Int J Epidemiol*. 1997; 26:575–83. [PubMed: 9222783]
60. Ng DM, Jeffery RW. Relationships between perceived stress and health behaviors in a sample of working adults. *Health Psychol*. 2003; 22:638–42. [PubMed: 14640862]
61. Wardle J, Steptoe A, Oliver G, Lipsey Z. Stress, dietary restraint and food intake. *J Psychosom Res*. 2000; 48:195–202. [PubMed: 10719137]
62. Conner M, Fitter M, Fletcher W. Stress and snacking: A diary study of daily hassles and between-meal snacking. *Psychol Health*. 1999; 14:51–63.
63. O'Connor DB, O'Connor RC. Perceived changes in food intake in response to stress: the role of conscientiousness. *Stress Health*. 2004; 20:279–91.
64. O'Connor DB, Jones F, Conner M, McMillan B, Ferguson E. Effects of daily hassles and eating style on eating behavior. *Health Psychol*. 2008; 27:S20–31. [PubMed: 18248102]
65. Derosa G, Ferrari I, D'Angelo A, Salvadeo ST, Fogari E, Gravina A, Mereu R, Palumbo I, Maffioli P, Randazzo S, Cicero AG. Oral fat load effects on inflammation and endothelial stress markers in healthy subjects. *Heart Vessels*. 2009; 24:204–10. [PubMed: 19466522]
66. Davis NJ, Crandall JP, Gajavelli S, Berman JW, Tomuta N, Wylie-Rosett J, Katz SD. Differential effects of low-carbohydrate and low-fat diets on inflammation and endothelial function in diabetes. *Journal of Diabetes and its Complications*. 2011; 25:371–6. [PubMed: 22036100]
67. Kandula NR, Diez-Roux AV, Chan C, Daviglus ML, Jackson SA, Ni H, Schreiner PJ. Association of acculturation levels and prevalence of diabetes in the multi-ethnic study of atherosclerosis (MESA). *Diabetes Care*. 2008; 31:1621–8. [PubMed: 18458142]
68. Rajpathak SN, Wylie-Rosett J. High prevalence of diabetes and impaired fasting glucose among Chinese immigrants in New York City. *J Immigr Minor Health*. 2011; 13:181–3. [PubMed: 20533090]
69. Stanford JL, Herrinton LJ, Schwartz SM, Weiss NS. Breast cancer incidence in Asian migrants to the United States and their descendants. *Epidemiology*. 1995; 6:181–3. [PubMed: 7742407]
70. Boykin S, Diez-Roux AV, Carnethon M, Shrager S, Ni H, Whitt-Glover M. Racial/ethnic heterogeneity in the socioeconomic patterning of CVD risk factors: in the United States: the multi-ethnic study of atherosclerosis. *J Health Care Poor Underserved*. 2011; 22:111–27. [PubMed: 21317510]
71. Huxley R, James WP, Barzi F, Patel JV, Lear SA, Suriyawongpaisal P, Janus E, Caterson I, Zimmet P, Prabhakaran D, Reddy S, Woodward M. Ethnic comparisons of the cross-sectional relationships between measures of body size with diabetes and hypertension. *Obesity Reviews*. 2008; 9(Suppl 1):53–61. [PubMed: 18307700]
72. Shai I, Jiang R, Manson JE, Stampfer MJ, Willett WC, Colditz GA, Hu FB. Ethnicity, obesity, and risk of type 2 diabetes in women: a 20-year follow-up study. *Diabetes Care*. 2006; 29:1585–90. [PubMed: 16801583]
73. Parkin, DM.; Whelan, SL.; Ferlay, J.; Raymond, L.; Young, J., editors. *Cancer incidence in five continents*. Vol. VII. Lyon: International Agency for Research on Cancer; 1997.
74. Deapen D, Liu LJ, Perkins C, Bernstein L, Ross RK. Rapidly rising breast cancer incidence rates among Asian-American women. *International Journal of Cancer*. 2002; 99:747–50.

75. Almeida D, Neupert S, Banks S, Serido J. Do daily stress processes account for socioeconomic health disparities? *J Gerontol B Psychol Sci Soc Sci*. 2005; 60:34–9. [PubMed: 16251588]
76. Dickerson SS, Gable SL, Irwin MR, Aziz N, Kemeny ME. Social-evaluative threat and proinflammatory cytokine regulation: an experimental laboratory investigation. *Psychol Sci*. 2009; 20:1237–44. [PubMed: 19754527]
77. Dickerson SS, Kemeny ME. Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. *Psychol Bull*. 2004; 130:355–91. [PubMed: 15122924]
78. Paul K, Boutain D, Agnew K, Thomas J, Hitti J. The relationship between racial identity, income, stress and C-reactive protein among parous women: implications for preterm birth disparity research. *J Natl Med Assoc*. 2008; 100:540–6. [PubMed: 18507206]
79. Low CA, Matthews KA, Hall M. Elevated C-reactive protein in adolescents: Roles of stress and coping. *Psychosomatic medicine*. 2013
80. Fang CY, Miller SM, Bovbjerg DH, Bergman C, Edelson MI, Rosenblum NG, Bove BA, Godwin AK, Campbell DE, Douglas SD. Perceived stress is associated with impaired T-cell response to HPV16 in women with cervical dysplasia. *Ann Behav Med*. 2008; 35:87–96. [PubMed: 18347908]
81. Peralta-Ramirez MI, Jimenez-Alonso J, Godoy-Garcia JF, Perez-Garcia M. The effects of daily stress and stressful life events on the clinical symptomatology of patients with lupus erythematosus. *Psychosomatic medicine*. 2004; 66:788–94. [PubMed: 15385708]
82. Choi J, Joseph L, Pilote L. Obesity and C-reactive protein in various populations: a systematic review and meta-analysis. *Obes Rev*. 2013; 14:232–44. [PubMed: 23171381]
83. Alpass F, Flett R, Trlin A, Henderson A, North N, Skinner M, Wright S. Psychological Wellbeing in Three Groups of Skilled Immigrants to New Zealand. *Australian Journal of Rehabilitation Counselling*. 2007; 13:1–13.
84. Macy EM, Hayes TE, Tracy RP. Variability in the measurement of C-reactive protein in healthy subjects: implications for reference intervals and epidemiological applications. *Clin Chem*. 1997; 43:52–8. [PubMed: 8990222]
85. Danesh J, Wheeler JG, Hirschfield GM, Eda S, Eiriksdottir G, Rumley A, Lowe GD, Pepys MB, Gudnason V. C-reactive protein and other circulating markers of inflammation in the prediction of coronary heart disease. *New England Journal of Medicine*. 2004; 350:1387–97. [PubMed: 15070788]
86. Hu FB, Meigs JB, Li TY, Rifai N, Manson JE. Inflammatory markers and risk of developing type 2 diabetes in women. *Diabetes*. 2004; 53:693–700. [PubMed: 14988254]
87. Spranger J, Kroke A, Mohlig M, Hoffmann K, Bergmann MM, Ristow M, Boeing H, Pfeiffer AF. Inflammatory cytokines and the risk to develop type 2 diabetes: results of the prospective population-based European Prospective Investigation into Cancer and Nutrition (EPIC)-Potsdam Study. *Diabetes*. 2003; 52:812–7. [PubMed: 12606524]
88. Siemes C, Visser LE, Coebergh JW, Splinter TA, Witteman JC, Uitterlinden AG, Hofman A, Pols HA, Stricker BH. C-reactive protein levels, variation in the C-reactive protein gene, and cancer risk: the Rotterdam Study. *J Clin Oncol*. 2006; 24:5216–22. [PubMed: 17114654]
89. Elkind MS, Cheng J, Boden-Albala B, Rundek T, Thomas J, Chen H, Rabbani LE, Sacco RL. Tumor necrosis factor receptor levels are associated with carotid atherosclerosis. *Stroke*. 2002; 33:31–7. [PubMed: 11779885]
90. Shai I, Schulze MB, Manson JE, Rexrode KM, Stampfer MJ, Mantzoros C, Hu FB. A prospective study of soluble tumor necrosis factor-alpha receptor II (sTNF-RII) and risk of coronary heart disease among women with type 2 diabetes. *Diabetes Care*. 2005; 28:1376–82. [PubMed: 15920055]
91. Strackowski M, Kowalska I, Nikolajuk A, Adamska A, Karolczuk-Zarachowicz M, Karczewska-Kupczewska M, Kozłowska A, Gorska M. Plasma levels of soluble tumor necrosis factor-alpha receptors are related to total and LDL-cholesterol in lean, but not in obese subjects. *Cardiovascular Diabetology*. 2006; 5:14. [PubMed: 16803616]

Glossary

BMI	body mass index
CRP	C-reactive protein
GEQ-A	General Ethnicity Questionnaire-American
LES	Life Experiences Survey
MASS	Migration-Acculturation Stress Scale
sTNFR2	soluble tumor necrosis factor receptor 2
TNF-α	tumor necrosis factor-alpha

Table 1

Participant Characteristics (n=407)

Variable (SD)	No. of participants (%)	Mean
Age (years) (4.54)		43.8
Length of US residence (years) (4.80)		7.5
Body mass index (kg/m ²) (2.85)		23.4
Married	375 (92.1%)	
Education		
<8 years	196 (48.2%)	
9-12 years / technical school	141 (34.6%)	
At least some college	70 (17.2%)	
Smoking status	404 (99.3%) non-smokers	
General Ethnicity Questionnaire score (0.71)		2.13
Migration-Acculturation Stressor Scale score (10.63)		36.42
Number of positive life events (1.17)		0.51
Impact rating score for positive events (2.30)		0.94
Number of negative life events (2.07)		1.12
Impact rating score for negative events (4.11)		2.12
Inflammatory markers		
C-reactive protein (mg/L) (1.84)		1.54
Low (< 1 mg/L)	220 (54.1%)	
Average (1.0-3.0 mg/L)	136 (33.4%)	
High (> 3.0 mg/L)	51 (12.5%)	
Soluble TNF receptor 2 (pg/mL) (920.35)		4187.56

Table 2

Correlations among study variable

Variable	2	3	4	5	6	7	8	9
1. Age	0.04	0.19***	-0.01	0.07	0.04	0.11*	0.17***	0.10*
2. Length US residence	--	0.06	0.18**	-0.35***	-0.11*	-0.001	0.01	-0.11*
3. BMI		--	-0.06	-0.05	0.02	0.05	0.28***	0.09
4. GEQ-A			--	0.05	0.24***	0.23***	-0.06	-0.03
5. MASS				--	0.19***	0.36***	0.11*	0.10
6. Positive events impact rating					--	0.27***	0.11*	-0.01
7. Negative events impact rating						--	0.09	0.05
8. CRP							--	0.14**
9. sTNFR2								--

Note:

*
p < .05;**
p < .01;***
p < .001.

Table 3

Associations of stress and inflammatory markers in Chinese immigrant women

	CRP			sTNFr2		
	β	B (95% CI)	p	β	B (95% CI)	p
Model 1						
Age	0.14	0.06 (0.01 to 0.12)	0.029	0.06	0.006 (-0.01 to 0.02)	0.34
BMI	0.34	0.17 (0.11 to 0.22)	<0.001	0.13	0.01 (0.00 to 0.02)	0.049
Positive life events	0.07	0.03 (-0.02 to 0.07)	0.28	-0.07	-0.005 (-0.02 to 0.004)	0.26
Negative life events	-0.04	-0.02 (-0.07 to 0.03)	0.51	-0.05	-0.004 (-0.01 to 0.01)	0.47
Acculturative stress (MASS)	0.14	0.07 (0.01 to 0.13)	0.031	0.17	0.02 (0.004 to 0.03)	0.012
Model 2 ¹						
Age	0.12	0.06 (-0.002 to 0.11)	0.059	0.08	0.008 (-0.004 to 0.02)	0.21
BMI	0.33	0.16 (0.10 to 0.22)	<0.001	0.13	0.01 (0.00 to 0.02)	0.042
Acculturation level (GEQ-A)	-0.13	-0.07 (-0.13 to -0.003)	0.038	0.03	0.003 (-0.01 to 0.02)	0.66
Length of US residence	0.14	0.07 (0.004 to 0.13)	0.039	-0.16	-0.01 (-0.03 to -0.001)	0.030
Positive life events	0.11	0.04 (-0.01 to 0.09)	0.091	-0.09	-0.007 (-0.02 to 0.003)	0.16
Negative life events	-0.04	-0.02 (-0.07 to 0.04)	0.54	-0.03	-0.002 (-0.01 to 0.01)	0.70
Acculturative stress (MASS)	0.17	0.08 (0.02 to 0.14)	0.014	0.12	0.01 (-0.003 to 0.02)	0.11

Note:

¹Linear regression models adjusted for the variables listed in Model 1 (age, BMI) and acculturation variables (GEQ-A score and length of US residence).

β = standardized regression coefficients; B = unstandardized regression coefficients; CI = confidence interval.