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Bayesian Estimation Under Informative Sampling: Investigating The Association Between Depression And Inflammation

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BAYESIAN ESTIMATION UNDER INFORMATIVE SAMPLING: INVESTIGATING THE ASSOCIATION BETWEEN DEPRESSION AND INFLAMMATION

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Survey data is often collected using complex sampling designs so that the probability of being included in the study is related to the outcome of interest (i.e. informative sample). Recently, a novel fully-Bayesian method has been developed for modeling data under informative sampling. Initial results indicate that this novel construction reduces bias in variance estimates compared to other pseudo-Bayesian techniques. The performance of this method has yet to be compared to traditional Frequentist approaches, which typically rely on Taylor series linearization (TSL) or resampling techniques for standard error (SE) estimation. Here, we modeled the relationship between depression and inflammation using data from the National Health and Nutrition Examination Survey using both a the fully-Bayesian method and a Frequentist method, specifically weighted least squares regression with TSL variance estimation. Although fully-Bayesian and the standard Frequentist approach generated similar parameter estimates, the fully-Bayesian model tended to produce smaller SEs than the Frequentist method. These findings suggest that the fully-Bayesian method performs equivalently to traditional Frequentist methods and may even provide better variance estimates than those computed by TSL. The current findings also replicate previous findings that the relationship between inflammation and depression is likely influenced by alcohol use, smoking, and Body Mass Index (BMI), but must be interpreted cautiously due to the high level of missing data.

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BACKGROUND

Statistical Methods for Analyzing Informative Samples

For practical reasons, survey data are frequently collected from samples drawn from multi-stage, clustered sampling designs that result in an unequal probability of selection. When these selection, or inclusion, probabilities are related to the values of the response variable of interest, we describe the sample as "informative". Informative sampling provides crucial information about the greater population and could result in biased parameter estimates if ignored (Sugden & Smith, 1984). Correctly accounting for informative sampling is essential for drawing accurate conclusions regarding epidemiological data with this type of sampling design.

To account for informative sampling, model-based approaches typically assign a sampling weight that is inversely proportional to the marginal inclusion probability for each observation. In weighted least squares (WLS) regression, sampling weights are incorporated into the maximal likelihood estimation computation and the solution yields a maximal pseudo-likelihood estimation of the population parameters (Binder, 1983). The likelihood contribution of each observation in the sample is adjusted by its associated sampling weight, thereby creating an adjusted joint likelihood that accounts for the imbalance of information in the sample and provides unbiased point estimates (Binder, 1983; Pfeffermann, 1993). Extant statistical programs (i.e. SAS, STATA, SUDAAN) use Taylor series linearization (TSL) or Balanced Repeated Replication (BRR) methods to estimate sampling errors of estimators based on complex sample designs. Other methods for variance estimation, such as Fay's BRR, Jackknife, and Hadamard matrix methods are also available.

In addition to the aforementioned Frequentist methods, there are a variety of pseudo-Bayesian model-based approaches to account for informative sampling. Bayesian survey inference requires specification of a prior distribution for the population values of the response variables. Inferences for the finite population are then based on the posterior distribution, or the distribution of nonsampled values of the response variable given the sampled values of the response variable. Current Bayesian approaches require a particular form of the likelihood and focus solely on domain-level estimation of mean and total statistics instead of on inference of parameters from the generating model (Dong, Elliott, & Raghunathan, 2014; Kunihama, Herring, Halpern, & Dunson, 2014; Rao & Wu, 2010). Alternatively, another approach by Savitsky and Toth (2016) exponentiates each unit likelihood contribution by its sampling weight to produce a pseudo-likelihood. This pseudo-likelihood, along with the prior distributions for the model parameters, is used to estimate the pseudo-posterior distributions used for model inference (Savitsky & Toth, 2016).

While these methods provide asymptotically unbiased parameter estimates, they are not fully-Bayesian and are limited by assumed and fixed inclusion probabilities. To date there is no fully-Bayesian method for model estimation under informative sampling. León-Novelo and Savtisky (unpublished) propose a novel fully-Bayesian method to adjust for informative sampling. Instead of treating the inclusion probabilities as fixed, the fully-Bayesian approach specifies a joint distribution for the response and inclusion probabilities. Unlike pseudo-posterior approaches

(Pfeffermann, Moura, & Silva, 2006; Savitsky & Toth, 2016) the fully-Bayesian approach proposed by León-Novelo and Savtisky (unpublished) treats the inclusion probabilities as a random variable and defines a likelihood by jointly modeling the response of the observed sample and the inclusion probability. Together, the joint likelihood and the prior distributions complete a Bayesian model. Initial results indicate that, compared to the pseudo posterior method, the point estimates of regression coefficients generated by the fully-Bayes approach are more robust against inclusion probabilities with high variance. These results also show that the fully-Bayes approach accurately estimate uncertainty under informative sampling while existing pseudo-Bayes underestimates it (León-Novelo & Savtisky, unpublished). Moreover, preliminary evidence indicates that the fully-Bayes approach produces point estimates that are robust, even in cases where the distribution of the inclusion probability is misspecified.

The current study aims to highlight the differences in between the fully-Bayesian method and the pseudo-likelihood approach on assessing relationships with informatively sampled survey data by modeling the relationship between inflammation and depression.

Inflammation and Depression

The World Health Organization estimates that more than 300 million people of all ages suffer from depression, and it is the leading cause of disability worldwide (World Health Organization, 2018). The global prevalence of depression and depressive symptoms has continued to increase in the past few decades, with an 18.4% increase between 2005 and 2015 (Vos et al., 2016), making depression a pressing, current public health concern.

Levels of inflammation are noticeably higher in about a third of depressed patients (Raison & Miller, 2011; Rethorst, Bernstein, & Trivedi, 2014), and this subset of patients appears to be more resistant to treatment. Patients with Major Depressive Disorder (MDD) who have heightened plasma inflammatory markers, proinflammatory gene expression, or polymorphisms in inflammation-related genes are less responsive to antidepressant medications (Baune et al., 2010; Carvalho et al., 2013; Eller, Vasar, Shlik, & Maron, 2008; O'Brien, Scully, Fitzgerald, Scott, & Dinan, 2007; Raison et al., 2013; Su et al., 2014; Wong, Dong, Maestre-Mesa, & Licinio, 2008; Yu, Chen, Hong, Chen, & Tsai, 2003). Further, an inhibitor of the proinflammatory cytokine tumor necrosis factor (TNF)-α substantially reduced depressive symptoms in a subset of medication resistant MDD patients with high baseline levels of inflammation (Raison et al., 2013), suggesting inflammation may be a promising clinical target specifically for patients with co-occurring depression and inflammation.

C-reactive protein (CRP) is a marker of systemic inflammation that has been extensively studied in population-based and clinical samples, likely due to its low cost and accessibility compared to cytokines. Increased levels of CRP are associated with increased depression symptom severity (Ekinci & Ekinci, 2017; Howren, Lamkin, & Suls, 2009; Khandaker, Pearson, Zammit, Lewis, & Jones, 2014); poorer response to antidepressant medications (Strawbridge et al., 2015); severity of overall depression, suicidal thoughts, disinterest, and cognitive symptoms

in women (Köhler-Forsberg et al., 2017); and lifetime history of major depression and recurrent depressive episodes in men (Ford & Erlinger, 2004).

Recently, monocyte-high density lipoprotein cholesterol ratio (MHR) has emerged as a promising new marker of inflammation and oxidative stress, and has been identified as a stable predictor of cardiovascular disease (Canpolat et al., 2016; Cetin et al., 2016; Ganjali et al., 2018). Peripheral monocytes are the primary producers of pro-inflammatory cytokines and contribute significantly to systemic inflammation (Ingersoll, Platt, Potteaux, & Randolph, 2011; Kurihara, Warr, Loy, & Bravo, 1997). While only a specific subpopulation of monocytes produces inflammatory cytokines (Yang, Zhang, Yu, Yang, & Wang, 2014), monocyte count has been has been associated with a variety of cardiovascular conditions (Afiune Neto, Mansur, Avakian, Gomes, & Ramires, 2006; Maekawa et al., 2002) and has been shown to be a more reliable risk factor for CVD than CRP, inflammatory cytokine interleukin (IL)-6, hypertension, and cigarette smoking (Chapman, Beilby, McQuillan, Thompson, & Hung, 2004). Increased blood monocyte count is also associated with reduced high density lipoprotein (HDL) cholesterol levels (Ganda et al., 2013). The anti-inflammatory and anti-oxidant effects of HDL are well documented (Navab et al., 2007). HDL protects endothelial cells against the unfavorable effects of low-density lipoprotein (LDL) (Hessler, Robertson, & Chisolm, 1979; Li et al., 2000), and prohibits oxidation of LDL molecules (Parthasarathy, Barnett, & Fong, 1990). HDL has also been shown to suppress cytokine expression in endothelial cells and monocytes in vitro and in animals (Cheng et al 2012). Thus, the balance of blood monocytes and HDL, as measured by MHR, may serve as an informative marker for systemic inflammation.

Research Objectives

Estimating the relationship between inflammation and depression in a large-scale, informative sample provides a realistic context for the application of this novel fully-Bayesian method. Thus, we will use this context to demonstrate the application of the fully-Bayesian construction for model estimation and compare these models to ones generated by a common Frequentist approach. This will allow us to fully investigate the adequacy of this novel Bayesian method in comparison to a standard approach.

Specifically, the primary objective of the current study is to evaluate the performance of a fully-Bayesian method in estimating regression coefficients under informative sampling. To accomplish this objective, we will use fully-Bayesian estimation to generate separate regressions quantifying the relationship between unique inflammatory markers (CRP and MHR) and depressive symptoms in an informative sample. The point estimates and their respective standard errors generated by the fully-Bayesian construction will be qualitatively compared to the ones generated by the standard weighted regression estimation, which utilizes WLS parameter estimation and TSL for variance estimation.

While some research suggests that inflammation is associated with specific depressive-symptom clusters (i.e. somatic and atypical symptoms), these studies have been limited to the use of cytokines and CRP as inflammatory markers (Case & Stewart, 2014; Duivis, Vogelzangs,

Kupper, de Jonge, & Penninx, 2013; Stewart, Rand, Muldoon, & Kamarck, 2009). Therefore, a secondary objective is to utilize non-informative or vague priors with the fully-Bayesian method to investigate an exploratory relationship between MHR and subtypes of depression, specifically somatic and non-somatic depression.

Public Health Significance

Accurately estimating the relationship between variables in large-scale epidemiological data sets typically requires controlling for a variety of covariates and accounting for informative sampling. While classical weighted regression methods (i.e. pseudo-likelihood estimation of regression parameters and TSL or resampling methods for estimation of the standard error of regression parameters) generate asymptotically unbiased model estimates, they cannot take advantage of some of the key benefits offered by Bayesian methods. Specifically, Bayesian methods allow for the incorporation of existing information about the distribution of covariates into parameter estimates and accurate inference from small sample sizes. (Berry, 2006; Bonangelino et al., 2011; Lilford, Thornton, & Braunholtz, 1995). A fully-Bayesian construction of a weighted regression under informative sampling would allow public health scientists to account for prior information about covariates, thereby generating more accurate and robust estimates of the standard error of model parameters, even in studies with small sample sizes.

This study will benefit public health research methodology by providing analysts with more accurate estimation of model parameter standard errors under informative sampling than currently offered by other common Frequentist methods. Future public health scientists will be able to use this method to incorporate prior information about variables of interest into their models, run studies with smaller sample sizes, and more accurately estimate the certainty of effect sizes of an exposure of interest. Taken together, this method will allow researchers to use informatively sampled data to more accurately estimate the relationship between depressive symptoms, as well as specific symptom clusters, and inflammation. Further, this method could be used to identify traits (i.e. race, medication, cardiovascular health, etc.) that attenuate or modulate the bidirectional relationship between depression and inflammation.

METHODS

Survey Design and Study Sample

The National Health and Nutrition Examination Survey (NHANES) is a national survey designed to gather nutrition and health information representation of the civilian, non-institutionalized U.S. population. Detailed information of sampling methods is described on the NHANES website (Center for Disease Control and Prevention, 2013c). Briefly, NHANES data are collected through a complex, multistage, probability sampling design. The NHANES has fixed sample-size targets for subpopulations of interest (e.g. minorities, low income groups, pregnant

women), and intentionally over-samples these subpopulations to increase the reliability and precision of health status indicator estimates for these groups. In order to account for informative sampling, each observation in the NHANES is assigned a sampling weight, which is calculated based on the inclusion probability and non-response (Center for Disease Control and Prevention, 2013b).

The study population of interest is healthy adults (over the age of 18) without any existing inflammatory conditions. While it is very difficult to account for all medical conditions, we excluded participants endorsing a history of or current inflammatory health conditions that likely influence marker levels: cardiovascular disease (coronary heart disease, angina, myocardial infarction, stroke, congestive heart failure) (Casas, Shah, Hingorani, Danesh, & Pepys, 2008), current chronic bronchitis (Gan, Man, Senthilselvan, & Sin, 2004), emphysema (Omori et al., 2009), rheumatoid arthritis (Sokka & Pincus, 2009), human immunodeficiency virus (Tien et al., 2010), hepatitis C (Kessel et al., 2007), and current liver condition (Abraham et al., 2009). Further, respondents with CRP values >=10 were also excluded, as values above this threshold indicate an acute infection (Pearson et al., 2003).

Because there is no standard method for addressing missing data in an informative sample (Berg, Kim, & Skinner, 2016) and because it is outside the scope of this project, we decided to limit our analyses to respondents with complete data. A total of 31,034 individuals who participated in the NHANES during the three cycles conducted from 2005 to 2010. Of those individuals, 15,760 completed all items on the Patient Health Questionnaire (PHQ-9) (Kroenke MD & Spitzer MD, 2002), a questionnaire used to assess for depressive symptoms over the past two weeks, during the Mobile Examination Centers (MEC) examination. Participants with missing CRP (n=689) and MHR (n=136) data were also excluded, resulting in a sample of 14,935. An additional (n=8,092) were excluded for either missing data on or endorsing at least one of the exclusionary health conditions. Individuals missing education (n= 6), alcohol use (n=1,669), body mass index (BMI, n=24), and diabetes (n=4) data were also excluded. The remaining individuals also had data for covariates of interest, i.e. sex, race/ethnicity, gender, smoking status, medication information, leaving us with a final complete case sample of n=5,142.

Data Collection

Detailed descriptions of data collection methods are available at the study website (Center for Disease Control and Prevention, 2013a). Briefly, approximately 5,000 people were recruited each survey year. Individuals were selected and consented to participate completed a computer-assisted interview conducted by trained personnel in their homes. Additional interviews (such as the depression assessment) and all medical examinations (including the blood draw) were conducted at MEC after the home interview.

Depressive symptoms. The PHQ-9 asks respondents to consider, using a 4-point scale (0=not at all, 1= several days, 2= more than half the days, and 3=nearly every day), how frequently they experienced the following 9 symptoms of major depressive disorder: (1) anhedonia, (2) depressed mood, (3) sleep disturbance, (4) fatigue, (5) appetite changes, (6) low self-esteem, (7)

concentration problems, (8) psychomotor retardation/agitation, and (9) suicidal ideation. Total scores range from 0-27, with higher values indicating more severe depression. The PHQ-9 initially categorizes depression scores into four categories: 0-4 as no depression, 5-9 as minimal, 10-14 as mild, 15-19 as moderate, and 20-27 as severe (Kroenke MD & Spitzer MD, 2002).

The PHQ-9 Total (sum of all items) and two subscale scores (Somatic and Nonsomatic) were then computed. The PHQ-9 Somatic Subscale score was computed by summing the sleep disturbance, fatigue, appetite changes, and psychomotor retardation/agitation items (Items 3, 4, 5, and 8), and the PHQ-9 Nonsomatic subscale score was computed by summing the remaining 5 times (Items 1, 2, 6, 7, and 9).

Inflammatory markers. Whole blood samples were collected from respondents who were asked to abstain from food, beverages (other than water), and certain over-the counter-medications for at least nine hours prior to their MEC examination. Documentation for standard laboratory procedures used to collect Complete Blood Count, CRP, and Cholesterol samples can be found on the NHANES website (Center for Disease Control and Prevention, 2007, 2009, 2011).

Covariates. The following variables were included as covariates in our model due to their potential relationship with inflammation: age, race/ethnicity (Mexican American, other Hispanic, non-Hispanic White, non-Hispanic Black, and Other Race), sex (Male, Female), education (less than 9th grade, some high school, high school or GED, some college, college graduate or above), diabetes (diabetic, non-diabetic, borderline), use of lipid lowering medication (Yes, No), and use of selective serotonin reuptake inhibitor (SSRI) or serotonin and norepinephrine reuptake inhibitor (SNRI) medications (Yes, No) (Aiello et al., 2009; Biondi-Zoccai, Abbate, Liuzzo, & Biasucci, 2003; Creider, Hegele, & Joy, 2012; Kraus et al., 2007; Tynan et al., 2012). We also included BMI, current smoking status, and alcohol consumption as potential confounders (Hamer, Molloy, de Oliveira, & Demakakos, 2009; Miller & Blackwell, 2006; Miller, Freedland, Carney, Stetler, & Banks, 2003). The smoking assessment for respondents 20 and older was not the same as that of individuals 18-19 years. We classified respondents 20 years and older as current smokers if they reported having had at least 100 cigs in a lifetime and currently smoke at least some days out of the week. We classified respondents aged 18-19 as current smokers if they are smoking 15 or more cigarettes in the last 30 days. Alcohol status was split into 3 levels based on gender and age. We first calculated average number of alcohol drinks per day for every individual in the sample. From this variable, we created three groups: abstainer (0 drinks/day); moderate user (.1-2 drinks/day in men and .1-1 drinks/day in women) and heavier user (>2 drinks/day in men, >1 drinks/day in women). The 2009-2010 NHANES cycle included a special questionnaire for alcohol use for individuals ages 12-19. For this subsample, alcohol use categories were defined as abstainers (no alcohol in the past month), moderate user (>=1 drink in the past month) and heavier user (>=2 days in the past month where they had >=5 drinks).

Data Analysis

Selected methods.

Classical Method. A typical approach to handling informativeness is to use the weights to generate point estimates and use information about the sampling design (weights, strata, and primary sampling units (PSUs)) to estimate the standard errors via TSL or resampling methods. This method is standard in many statistical packages, such as SAS, STATA and SUDAAN, and has been used to estimate the relationship between inflammatory markers and depression in NHANES data previously (see Case et al. (2014), Ford et al. (2004), Hickman et al. (2014), Merikangas et al. (2011) for examples). The Classical method solves a WLS regression, where the inverse of inclusion probability is assigned to the diagonal of the weight matrix. The set of normal equations is solved by using a modified sweep routine that produces a generalized inverse and a solution for point estimates of regression coefficients (Pringle & Rayner, 1971). Although there are a variety of ways to estimate standard errors for the estimated regression coefficients, TSL will be utilized in this study. The TSL method obtains a first-order linear approximation for the ratio estimator and then uses the variance estimate for this approximation to estimate the variance of the estimate itself (Fuller, 1975; Woodruff, 1971). Because the NHANES uses a stratified and clustered sampling design, the TSL variance estimation method requires both stratum and PSUs information to compute variance estimates.

Fully-Bayesian Method. First, we constructed a joint distribution for the response and inclusion probabilities for the population, $p(y_i, \pi_i \mid \boldsymbol{x}_i, \boldsymbol{\theta}, \boldsymbol{\kappa})$, where i is each unit in the sample, y_i is the response value, π_i is the marginal inclusion probability, $\boldsymbol{x}_i$ is a vector of predictors, $\boldsymbol{\theta}$ is a vector of parameters or the distribution of y_i, and $\boldsymbol{\kappa}$ is a vector of parameters of the log normal distribution of π_i conditional on y_i. To do so, we specified a conditional distribution of the inclusion probabilities for all units in the population, $p(\pi_i \mid y_i, \kappa)$. Next, we specified prior distributions for our model parameters. We used the likelihood of the joint distribution along with the associated priors to generate a posterior distribution, from which we inferred point estimates and their respective 95% Bayesian credible intervals (95% BCI).

We specified normal, vague prior distributions for regression coefficients and truncated, positive Cauchy distribution, with location and scale parameters of 0 and 1, respectively, for the standard deviation. The appropriate distribution was investigated for each of the markers (CRP and MHR) response variables by qualitatively evaluating the posterior distribution.

General approach for each aim. To evaluate the fully-Bayesian model estimation under informative sampling, we generated regression models utilizing both Classical and fully-Bayesian approaches and compare model estimates qualitatively. The first set of regressions modeled depression and a set of covariates (i.e., age, race/ethnicity, sex, education, diabetes, use of lipid lowering medication, and use of SSRI or SNRI) as the predictor variables and the marker as the response variable; these regressions will be referred to as the adjusted models. Because BMI, smoking status, and alcohol use are potential mediators of the depression inflammation relationship (Hamer et al., 2009; Miller & Blackwell, 2006; Miller et al., 2003), we then added these variables to our models, to create fully-adjusted models for each inflammatory marker. The effect of confounds were assessed by determining the percent change in the effect size (i.e. coefficient estimate); any percent change greater than 10% was noted as supporting evidence for

a confounding effect. We qualitatively compared the pointwise estimates and 95% BCI for each of the model parameters generated by the fully-Bayesian method to the pointwise estimates and 95% confidence intervals (95% CI) generated with the Classical method.

Data Handling.

Total PHQ-9 depression scores were highly zero-inflated, with only 7.24% of the respondents in our final endorsed mild to severe depression (Total PHQ-9 scores >=10). Somatic and nonsomatic symptom scores were equally zero-inflated. Depression was therefore dichotomized, so that respondents who endorsed at least minimal depression (Total PHQ-9 scores >=5) were categorized as having current depression, and respondents that did not meet this threshold were categorized as having no current depression. Since we are also interested in the relationship between inflammation and symptom subtypes amongst individuals with at least subclinical depression, we decided to use only the depressed subsample to model the relationship between the two inflammatory markers and somatic and nonsomatic symptom severity.

For ease of interpretation, monocyte counts were converted from units of 10^3 cells/μL to 10^9 cells/L and HDL cholesterol units were converted from mg/dL to mg/L. The MHR was calculated as the ratio of monocyte count to HDL-C level. CRP and MHR values were also highly skewed and underwent a logarithmic transformation so that model assumptions would be met.

Gender, ethnicity, level of education, and alcohol use were coded as categorical indicators with male, non-Hispanic White, high school graduate or GED, and abstinence set as reference categories, respectively. Non-smokers, nondiabetics, no use of lipid-lowering medications, and no use of SSRI/SNRI medications were also set as reference categories for their respective dichotomous variables.

RESULTS

Descriptive Statistics. The average Total PHQ-9 scores for the full sample (combined depressed and non-depressed) was 3.05; the depressed group had a mean total depression symptom score of 8.82 compared to a mean of 1.29 in the non-depressed group. Median high-sensitivity serum CRP for the full sample was 1.60 mg/L, which was below the cut off for high risk of CVD (>3.0 mg/L). Values for MHR ranged from 0.10 to 8.57, with a median value of 1.03 in the full sample. The full sample was mostly male (52.41%), Non-Hispanic White (47.76), had some college education (31.25%), and had an average age of 35.41. Other descriptive information about the sample can be found in Table 1.

Classical vs. Fully-Bayesian model estimates. Figure 1 displays the coefficient estimates derived from the fully-Bayesian and Classical methods for fully-adjusted models predicting CRP, along with their respective 95% BCI and 95% CI. Figure 2 shows the same information for fully-adjusted models predicting MHR. As shown, there is considerable overlap between 95% BCI and CI for each coefficient. Figure 3 displays the differences between the magnitude of

fully-Bayesian estimators vs. the magnitude of Classical estimators for each coefficient. Overall, point estimates generated by WLS, or the Classical method, were larger in magnitude than point estimates generated by the fully-Bayes method. The mean and median differences between the magnitude of coefficient estimates in the fully-adjusted models using presence of depression (mean: -0.003, median: -0.007), somatic symptom severity (mean: -0.007, median: -0.004), and nonsomatic symptom severity (mean:-0.006, median: -0.003) to predict transformed CRP were all negative. Similarly, fully-adjusted models using presence of depression (mean: -0.002, median: -0.002), somatic symptom severity (mean: -0.003, median: -0.003), and nonsomatic symptom severity (mean:-0.003, median: -0.002) to predict transformed MHR were also all negative.

Classical vs. Fully-Bayesian variance estimates. Figure 4 displays the difference between the lengths of fully-Bayes 95% BCIs and the lengths of Classical 95% CIs for each variable in the fully-adjusted models. Negative values indicate that TSL variance estimates are larger than fully-Bayesian variance estimates. The plots indicate that fully-Bayes SE estimates were generally smaller than those generated by the Classical method; this trend was more consistent for models predicting MHR. Specifically, fully-adjusted models predicting the relationship between presence of depression and CRP, somatic symptom severity and CRP, and nonsomatic symptom severity and CRP had 5, 5, and 7 variables respectively with fully-Bayes variance estimates that were larger than TSL variance estimates. The mean (and median) values of differences between interval lengths for coefficients in the fully-adjusted models using presence of depression, somatic symptom severity, and nonsomatic symptom severity to predict transformed CRP values are -0.013 (-0.008), -0.013 (-0.011), and -0.010 (-0.002) respectively. In contrast, the fully-adjusted models predicting MHR had fewer variables with fully-Bayes variance estimates that were larger than TSL variance estimates. The mean (median) values of differences between interval lengths for coefficients in the fully-adjusted models using presence of depression, somatic symptom severity, and nonsomatic symptom severity to predict transformed MHR values are -0.005 (-0.005), -0.014 (-0.014), and -0.013 (-0.011), respectively. Table 2 through Table 7 show all the differences in 95% CBI and 95% CI estimates.

Presence of depression and inflammation: full sample. When adjusting for covariates only, presence of depressive symptoms was reliably associated with increased CRP levels in both the Classical ($\beta = 0.070$, 95% CI = [0.025, 0.114]) and Fully-Bayesian ($\beta = 0.047$, 95% BCI = [0.008, 0.084]) models. This small effect disappeared when confounders were included in the model (Classical: $\beta = 0.024$, 95% CI = [-0.017, 0.064], Fully-Bayesian: $\beta = 0.005$, 95% BCI = [-0.027, 0.039]). After adjusting for current smoking status, alcohol use, and BMI, the estimate for the effect of presence of depression was reduced by 65.71% in the Classical model and 89.36% in the Fully-Bayesian method, suggesting that the relationship between the presence of depressive symptoms and CRP is potentially confounded by these factors. Results for these models are summarized in Table 8.

Findings were similar for MHR, such that, when adjusting for covariates only, presence of depressive symptoms was reliably associated with increased MHR in both Classical ($\beta = 0.021$, 95% CI = [0.004, 0.038]) and Fully-Bayesian ($\beta = 0.022$, 95% BCI = [0.010, 0.033]) models.

Similarly, when potential confounders were included, this association was no longer significant in the Classical model (β = 0.010, 95% CI = [-0.006, 0.025]), but remained reliable in the Fully-Bayesian model (β = 0.012, 95% BCI = [0.000, 0.024]). As with CRP, the effect size for presence of depressive symptoms had a percent change decrease of 52.38% using Classical estimation methods and 45.45% using Fully-Bayesian estimation methods, suggesting that BMI, smoking status, and alcohol use are also potential confounders between MHR and depression. Estimates for these models are listed in Table 9.

Somatic symptom severity and inflammation: depressed sample. In the depressed subsample, somatic symptom severity was significantly and reliably associated with increased CRP levels in the covariate adjusted models using both Classical (β = 0.029, 95% CI = [0.011, 0.046]) and Fully-Bayesian (β = 0.021, 95% BCI = [0.006, 0.035]) model estimation methods. The positive association between somatic symptom severity and CRP levels was still significant/reliable after adjusting for BMI, alcohol use, and current smoking status (Classical: β = 0.016, 95% CI = [0.000, 0.032], Fully-Bayesian: β = 0.012, 95% BCI = [0.000, 0.023]) although the effect size was substantially diminished. After adjusting for potential confounders, the coefficient for somatic symptom severity was reduced by 43.83% in the Classical model and 42.13% in the Fully-Bayesian method. Results for these models are summarized in Table 10.

While there was a significant/reliable positive association between somatic symptom severity and MHR in the covariate adjusted model (Classical: β = 0.006, 95% CI = [0.001, 0.012], Fully-Bayesian: β = 0.006, 95% BCI = [0.001, 0.010]), this relationship was absent when the model was fully-adjusted for confounders (Classical: β = 0.002, 95% CI = [-0.003, 0.008], Fully-Bayesian: β = 0.003, 95% BCI = [-0.002, 0.008]). The coefficient estimate for somatic symptom severity was reduced by 64.55% and 43.38% in the Classical and Fully-Bayesian methods, respectively. Table 11 displays the estimates for these models.

Nonsomatic symptom severity and inflammation: depressed sample. Nonsomatic symptom severity was not significantly or reliably associated with CRP levels in either the adjusted (Classical: β = -0.007, 95% CI = [-0.022, 0.007], Fully-Bayesian: β = -0.008, 95% BCI = [-0.020, 0.005]) or fully-adjusted models (Classical: β = -0.005, 95% CI = [-0.016, 0.007], Fully-Bayesian: β = -0.004, 95% BCI = [-0.015, 0.006]). Similarly, no significant or reliable association was detected between MHR and nonsomatic symptom severity in either the (Classical: β = 0.002, 95% CI = [-0.004, 0.007], Fully-Bayesian: β = 0.002, 95% BCI = [-0.002, 0.006]) or fully-adjusted models (Classical: β = 0.002, 95% CI = [-0.003, 0.007], Fully-Bayesian: β = 0.002, 95% BCI = [-0.002, 0.006]), suggesting that there is no relationship between non-specific inflammation and nonsomatic symptom severity in individuals endorsing some depressive symptoms. Results for these analyses with CRP and MHR are summarized in Table 12 and Table 13, respectively.

Notable Covariates. Several covariates were significant or reliable across all fully-adjusted models using the presence of depression and both types of depressive symptom clusters as predictors. Here, we note all such variables and their fully-Bayesian estimators, but Classical estimators can also be found on Tables 2-7 and are summarized graphically in Figures 2-3. Gender was reliably related to CRP levels such that women had consistently higher levels of

CRP after controlling for other covariates, potential confounders, presence of depression (β = 0.193, 95% BCI = [0.166, 0.221]), somatic symptom severity (β = 0.143, 95% BCI = [0.086, 0.197]), and nonsomatic symptom severity (β = 0.156, 95% BCI = [0.097, 0.204]). Individuals with a college education or higher had reliably lower levels of CRP in models that used presence of depression (β = -0.169, 95% BCI = [-0.213, -0.124]), somatic symptom severity (β =-0.226, 95% BCI = [-0.332, -0.119]), and nonsomatic symptom severity (β = -0.248, 95% BCI = [-0.352, 0.148]). BMI was also consistently associated with increased levels of CRP after controlling for presence of depression (β = 0.043, 95% BCI = [0.041, 0.045]), as well as somatic (β = 0.04, 95% BCI = [0.040, 0.048]), and nonsomatic (β = 0.041, 95% BCI = [0.041, 0.048]) symptoms in the depressed subgroup.

In addition to gender and BMI, MHR was reliably associated with being non-Hispanic Black, a current smoker, and both moderate and heavier alcohol use. Opposite of CRP, females had reliably lower levels of MHR after controlling for potential confounders, depression presence (β = -0.126, 95% BCI = [-0.144, -0.123]), somatic symptom (β = -0.13, 95% BCI = [-0.158, -0.112]), and nonsomatic symptoms (β = -0.133, 95% BCI = [-0.159, -0.110]. As with CRP, BMI was positively associated with MHR in the full-adjusted models when controlling for current depression (β = 0.008, 95% BCI = [0.007, 0.009]), somatic symptom severity (β = 0.007, 95% BCI = [0.005, 0.008]), and nonsomatic symptom severity (β = 0.007, 95% BCI = [0.005, 0.008]). Non-Hispanic Black respondents have consistently lower MHR compared to non-Hispanic White respondents after adjusting for depression (β = -0.113, 95% BCI = [-0.126, -0.100]), somatic symptoms (β = -0.111, 95% BCI = [-0.141, -0.082]), and nonsomatic symptoms (β = -.112, 95% BCI = [-0.145, -0.083]). Current smokers had reliably higher MHR when accounting for presence of depression (β = 0.052, 95% BCI = [0.039, 0.064]), as well as somatic (β = 0.47, 95% BCI = [0.023, 0.072]), and nonsomatic symptoms (β = 0.047, 95% BCI = [0.024, 0.070]) in our depressed subsample. Finally, both moderate alcohol and heavier alcohol use was associated with lower average MHR, such that moderate and heavy alcohol users had reliably lower mean MHR than abstinent respondents when adjusting for depression (Moderate: β = -0.036, 95% CI = [-0.047, -0.025], Heavier: β = -.057, 95% BCI = [-0.073, -0.040]), somatic symptoms (Moderate: β = -0.025, 95% CI = [-0.046, -0.003], Heavier: β = -0.042, 95% BCI = [-0.075, -0.008]), and nonsomatic symptoms (Moderate: β = -0.012, 95% CI = [-0.050, -0.001], Heavier: β = -0.042, 95% BCI = [-0.077, -0.007]).

DISCUSSION

The present study aimed to utilize fully-Bayesian method to estimate a model using real informatively sampled data. As a motivating example, we used NHANES data to model the relationship between two inflammatory markers and depression, as well as depressive symptom clusters. To assess the validity of our method, we qualitatively compared the fully-Bayesian point and standard error estimates to estimates generated from a common Frequentist method for modeling informatively sampled data. Specifically, we compared our fully-Bayes estimators to coefficient estimates generated by WLS and variance estimates generated by TSL. To our

knowledge, this is the first study to compare the performance of these estimation methods and MHR as a nonspecific inflammatory marker associated with depression.

Our results indicate that the fully-Bayesian method produces similar estimates to that of the classical, Frequentist method. Point estimates and 95% CBIs generated from our posterior distributions generally agreed with the results of respective Classical models. Although most point estimates were relatively congruent across both estimation methods, WLS point estimates tended to be larger in effect size than those generated by the fully-Bayes construction. Although we cannot speak to the true bias of these point estimates without a simulation, these current findings suggest that the fully-Bayesian point estimates tend to be more conservative and have smaller effect sizes than point estimates generated by WLS methods. Researchers should keep this trend in mind when using these methods to analyze other complex survey data.

Of notable interest are the differences in variance estimates between both methods. Specifically, the fully-Bayes method generated 95% CBI lengths that were narrower than the 95% CI lengths generated by TSL, particularly for models predicting MHR. These findings suggest that the fully-Bayesian method provides more accurate variance estimates than TSL. One possible explanation for the discrepancy in differences of variance estimates between models predicting CRP and models predicting MHR is distribution of transformed CRP and MHR values: transformed MHR values were more closely normally distributed than transformed CRP values, which may have resulted in more accurate variance estimates.

Regardless, our findings indicate that fully-Bayesian methods for analyzing informatively sampled data benefit researchers primarily by strengthening the confidence in effect sizes, as opposed to improving the estimate of the effect sizes themselves. While the Classical method employed in this study requires information about both the stratum and PSUs to calculate standard error via TSL, the fully-Bayesian approach does not require the specification of stratum or PSU for variance estimation. By treating the inclusion probabilities as a random variable and jointly modeling the response of the observed sample and the inclusion probability, we can generate accurate estimates without requiring the analyst to use TSL or resampling methods. Furthermore, this novel technique allows analysts to benefit from the other well-documented advantages of Bayesian methods over Frequentist approaches, such as the incorporation of informative prior distributions and applications to smaller sample sizes. Future research may consider comparing the performance of the fully-Bayesian method against variance estimates generate by resampling techniques, in both real samples and simulated data.

From an epidemiological perspective, the present study revealed a weak relationship between presence of depression and inflammation. The relationship between presence of depression and inflammation, as measured by CRP and MHR, was likely confounded by smoking status, alcohol use and BMI. The only relationship that remained statistically reliable after adjusting for potential confounders was that between MHR and presence of depression; these findings were only robust under fully-Bayesian estimation, albeit they bordered nonreliability when confounders were included in the model. The incongruency in variance estimates between Classical and fully-Bayesian methods in this model highlight the importance of accurate standard error estimation methods for detecting weak associations in informatively sampled data.

Although other studies using similar samples report a relationship between CRP and depression severity, they frequently do not adequately control for inflammatory health conditions or limit their sample to patients with diagnosed Major Depressive Disorder (Ford & Erlinger, 2004; Köhler-Forsberg et al., 2017; Rethorst, Bernstein, & Trivedi, 2014). The current findings build on previous research by focusing specifically on the relationship between inflammation and presence of depressive symptoms in healthy adults only. We also explored the relationship between MHR and depressive symptoms in healthy adults, which, to our knowledge, has not been done previously. These results provide preliminary evidence of a weak relationship between MHR and presence of depression. Exploring this relationship in a sample with higher incidence of moderate and severe depression would more clearly elucidate the association between MHR and depression in healthy adults.

Partially consistent with our expectations, inflammation was not associated with nonsomatic symptom severity in participants endorsing at least minimal depression, even before controlling for potential confounders. Conversely, increased somatic symptom severity in respondents endorsing some depression was related to increased CRP levels, but not MHR, even after adjusting for confounders. One must note that these effect sizes were small, but confidence and credible intervals were narrow, suggesting that the relationship between CRP and somatic symptom severity was weak, but robust. Our results are partially consistent with findings by Case & Stewart (2014), where the positive relationship between somatic symptoms and CRP remained after adjusting for the same confounders (BMI, smoking, alcohol use). Although the authors had stronger effect sizes, this could be attributed to differences in the way somatic symptom scores were calculated, less stringent exclusion criteria, and the inclusion of respondents endorsing none to minimal depressive symptoms in their analyses, which were excluded from our analyses to satisfy the assumptions of WLS regression. Our results indicate that while MHR may be a potential inflammatory marker related to depression overall, it may not be related to specific depressive symptom clusters.

There are several limitations in the current study. The lack of respondents reporting mild to severe depression was a primary limitation. The proportion of respondents with moderate to severe depression was relatively small before excluding for inflammatory medical conditions and, after subsampling, continued to be severely skewed and zero-inflated. Unlike other studies that dichotomize depression with PHQ Total>=10 (Shiue, 2015; Wirth, Shivappa, Burch, Hurley, & Hébert, 2017), we chose to dichotomize total depression scores at a threshold that was well below the clinical cutoff to adequately satisfy the assumptions of WLS regression. While this allowed us to generate unbiased estimates, it also forced us to a) dilute our depressed group by including respondents with minimal depression and b) assume that the relationship between depressive symptom severity and inflammation is monotonic. Some preliminary data exists for differences in cholesterol between individuals with severe and moderate depression only (Tedders et al., 2011), suggesting that the relationship between at least MHR may not be monotonic.

An additional limitation to our study was the cross-sectional design, which did not allow us to adequately assess the mediational relationship of our potential confounders. Furthermore, much

of the data we used to determine presence of exclusionary medical condition only required respondents to report if they had any history of a specific medical condition. Unable to determine the actual health state of respondents, we were forced to exclude all participants who had any history of a condition, regardless of their status at the time the CRP and MHR samples were collected. This may have introduced biased in our estimates, as we may have been unknowingly excluding healthy adults from our sample. Future research should include a longitudinal design and ensure that questions capture the current state of respondents. Finally, our study did not address missing data, which again severely cut down our sample size. Although there are many methods to address missing data in complex survey designs, more research is required to determine which imputation methods most effectively mitigate bias in model-based designs using informatively sampled data (Berg, Kim, & Skinner, 2016).

The current study serves as a guide for the application of a fully-Bayesian estimation in informative sampling. This novel method generates estimates that are comparable to traditional frequentist approaches, while providing epidemiologists and analyses with computational benefits of a Bayesian framework. Furthermore, our findings, although weak in effect size, contribute to the large body of epidemiological research examining the relationship between inflammation and depressive symptomology. Future research should focus on identifying inflammatory markers related to depression that are not strongly influenced by confounders.

Table 1. Characteristics of respondents

	Depression (n= 1,198)	**No Depression (n= 3,944)**	**Total Sample (N=5,142)**
	Median (SD)	Median (SD)	Median (SD)
CRP, mg/L	1.90 (6.73)	1.50 (5.77)	1.90 (6.02)
MHR, 10^3/mg	1.05 (0.61)	1.02 (0.56)	1.03 (0.57)
	Mean (SD)	Mean (SD)	Mean (SD)
$\log_{10}$(CRP)	0.25 (0.58)	0.17 (0.56)	0.19 (0.57)
$\log_{10}$(MHR)	0.01 (0.21)	0.00 (0.20)	0.01 (0.20)
Total PHQ-9 scores (range: 0-27)	8.82 (4.10)	1.29 (1.35)	3.05 (3.93)
Somatic Symptoms (range: 0-12)	4.86 (2.31)	1.07 (0.93)	1.84 (2.21)
Nonsomatic Symptoms (range: 0-15)	3.96 (2.75)	0.36 (0.725)	1.20 (2.12)
BMI	29.09 (7.23)	28.15 (6.44)	28.37 (6.64)
Age, years	35.01 (10.11)	35.52 (9.99)	35.41 (10.02)
	n (%)	n (%)	n (%)
Male (reference)	522 (43.57)	2173 (55.10)	2,695 (52.41)
Race/Ethnicity			
Mexican American	233 (19.45)	844 (21.40)	1,077 (20.95)
Other Hispanic	131 (10.93)	327 (8.29)	458 (8.91)
Non-Hispanic White (reference)	553 (46.16)	1,903 (48.25)	2,456 (47.76)
Non-Hispanic Black	218 (18.20)	696 (17.65)	914 (17.78)
Other Race	63 (5.26)	174 (4.41)	237 (4.61)
Education level			
Less than 9^{th} grade	84 (7.01)	273 (6.92)	357 (6.94)
9^{th}-12^{th} grade (no diploma)	242 (20.20)	528 (13.29)	770 (14.97)
High School Graduate/GED (reference)	184 (15.36)	870 (22.06)	1,164 (22.64)
Some College or Associates Degree	393 (32.8)	1,214 (30.78)	1,607 (31.25)
College Graduate or Above	184 (15.36)	1,059 (26.85)	1,244 (24.19)
Lipid-lowering medication use	62 (5.17)	163 (4.13)	225 (4.38)
SSRI/SNRI medication use	164 (13.70)	168 (4.26)	332 (6.46)
Current Smoker	447 (37.31)	971 (24.62)	1,418 (27.58)
Current Alcohol Use			
Abstainer (reference)	479 (39.98)	1459 (36.99)	1,938 (37.69)
Moderate User	547 (45.66)	2067 (52.41)	2,614 (50.84)
Heavier User	172 (14.36)	418 (10.60)	590 (11.47)

Table 2. Presence of Depression and log10(CRP): Lengths of confidence and credible intervals.

	L_{Bayes}	$L_{Classical}$	L_{Δ}
Adjusted			
Intercept	0.364	0.163	0.201
Presence of Depression	0.076	0.089	-0.013
Gender	0.061	0.078	-0.017
Age	0.003	0.004	0.000
Ethnicity			
Mexican-American	0.084	0.100	-0.016
Other Hispanic	0.103	0.160	-0.058
Non-Hispanic Black	0.086	0.106	-0.020
Other Ethnicity	0.141	0.146	-0.005
Education			
Less than 9th	0.141	0.171	-0.029
9^{th} through 12^{th}	0.107	0.127	-0.020
Some college	0.085	0.078	0.007
College/+	0.088	0.096	-0.008
Lipid Lowering Medication	0.154	0.141	0.013
Depression Medication	0.124	0.098	0.026
Diabetes	0.154	0.207	-0.052
Fully Adjusted			
Intercept	-2.029	0.173	-0.059
Presence of Depression	0.002	0.065	-0.016
Gender	-0.141	0.055	-0.025
Age	-0.002	0.003	-0.001
Ethnicity			
Mexican- American	-0.041	0.079	-0.008
Other Hispanic	-0.016	0.104	-0.044
Non-Hispanic Black	-0.115	0.073	-0.015
Other Ethnicity	-0.003	0.123	-0.008
Education			
Less than 9th	0.001	0.113	-0.036
9^{th} through 12^{th}	0.000	0.088	-0.002
Some college	-0.023	0.075	0.001
College/+	-0.053	0.083	0.001
Lipid Lowering Medication	-0.019	0.134	0.006
Depression Medication	-0.003	0.113	0.019
Diabetes	0.021	0.137	-0.066
Current Smoker	0.053	0.066	0.002
Heavier Alcohol Use	-0.064	0.094	-0.019
Moderate Alcohol Use	-0.038	0.057	-0.016
BMI	0.008	0.004	-0.002

Note: L_{Bayes} refers to the length of the fully-Bayesian 95% BCI, $L_{Classical}$ refers to the length of the Classical 95% CI, and L_{Δ} refers to the difference between L_{Bayes} and $L_{Classical}$.

Table 3. Presence of Depression and $\log_{10}$(MHR): Lengths of confidence and credible intervals.

	L_{Bayes}	$L_{Classical}$	L_{Δ}
Adjusted			
Intercept	0.048	0.051	-0.003
Presence of Depression	0.023	0.034	-0.010
Gender	0.021	0.024	-0.004
Age	0.001	0.001	0.000
Ethnicity			
Mexican-American	0.029	0.034	-0.006
Other Hispanic	0.040	0.048	-0.008
Non-Hispanic Black	0.030	0.038	-0.008
Other Ethnicity	0.048	0.060	-0.012
Education			
Less than 9th	0.048	0.058	-0.010
9^{th} through 12^{th}	0.036	0.040	-0.004
Some college	0.029	0.039	-0.010
College/+	0.030	0.046	-0.015
Lipid Lowering Medication	0.052	0.053	-0.001
Depression Medication	0.043	0.043	0.000
Diabetes	0.057	0.064	-0.007
Fully Adjusted			
Intercept	0.066	0.075	-0.009
Presence of Depression	0.024	0.031	-0.007
Gender	0.021	0.024	-0.003
Age	0.001	0.001	0.000
Ethnicity			
Mexican- American	0.029	0.030	-0.002
Other Hispanic	0.035	0.040	-0.005
Non-Hispanic Black	0.026	0.035	-0.009
Other Ethnicity	0.052	0.062	-0.009
Education			
Less than 9th	0.046	0.053	-0.007
9^{th} through 12^{th}	0.032	0.036	-0.004
Some college	0.028	0.037	-0.009
College/+	0.029	0.042	-0.013
Lipid Lowering Medication	0.048	0.057	-0.010
Depression Medication	0.040	0.037	0.003
Diabetes	0.050	0.061	-0.011
Current Smoker	0.025	0.029	-0.004
Heavier Alcohol Use	0.033	0.033	0.000
Moderate Alcohol Use	0.023	0.025	-0.002
BMI	0.002	0.002	-0.001

Note: L_{Bayes} refers to the length of the fully-Bayesian 95% BCI, $L_{Classical}$ refers to the length of the Classical 95% CI, and L_{Δ} refers to the difference between L_{Bayes} and $L_{Classical}$.

Table 4. Somatic Symptom Severity and $\log_{10}$(CRP): Lengths of confidence and credible intervals.

	L_{Bayes}	$L_{Classical}$	L_{Δ}
Adjusted			
Intercept	0.314	0.338	-0.024
Somatic Symptoms	0.029	0.036	-0.006
Gender	0.124	0.155	-0.031
Age	0.007	0.009	-0.002
Ethnicity			
Mexican-American	0.183	0.150	0.033
Other Hispanic	0.225	0.286	-0.062
Non-Hispanic Black	0.189	0.225	-0.036
Other Ethnicity	0.295	0.252	0.043
Education			
Less than 9th	0.300	0.308	-0.008
9^{th} through 12^{th}	0.187	0.230	-0.044
Some college	0.161	0.196	-0.035
College/+	0.213	0.231	-0.018
Lipid Lowering Medication	0.298	0.390	-0.091
Depression Medication	0.201	0.153	0.049
Diabetes	0.314	0.416	-0.102
Fully Adjusted			
Intercept	0.357	0.455	-0.098
Somatic Symptoms	0.023	0.031	-0.008
Gender	0.111	0.145	-0.034
Age	0.006	0.007	-0.001
Ethnicity			
Mexican- American	0.155	0.131	0.024
Other Hispanic	0.206	0.298	-0.093
Non-Hispanic Black	0.146	0.148	-0.002
Other Ethnicity	0.237	0.207	0.030
Education			
Less than 9th	0.232	0.263	-0.031
9^{th} through 12^{th}	0.157	0.149	0.009
Some college	0.146	0.158	-0.013
College/+	0.174	0.171	0.002
Lipid Lowering Medication	0.272	0.309	-0.037
Depression Medication	0.166	0.119	0.048
Diabetes	0.263	0.283	-0.019
Current Smoker	0.117	0.142	-0.025
Heavier Alcohol Use	0.176	0.216	-0.039
Moderate Alcohol Use	0.120	0.153	-0.033
BMI	0.008	0.008	-0.001

Note: L_{Bayes} refers to the length of the fully-Bayesian 95% BCI, $L_{Classical}$ refers to the length of the Classical 95% CI, and L_{Δ} refers to the difference between L_{Bayes} and $L_{Classical}$.

Table 5. Somatic Symptom Severity and $\log_{10}$(MHR): Lengths of confidence and credible intervals.

	L_{Bayes}	$L_{Classical}$	L_{Δ}
Adjusted			
Intercept	0.104	0.131	-0.027
Somatic Symptoms	0.009	0.010	-0.001
Gender	0.045	0.046	-0.001
Age	0.002	0.003	-0.001
Ethnicity			
Mexican-American	0.067	0.085	-0.018
Other Hispanic	0.075	0.097	-0.022
Non-Hispanic Black	0.064	0.068	-0.004
Other Ethnicity	0.100	0.094	0.006
Education			
Less than 9th	0.101	0.118	-0.017
9^{th} through 12^{th}	0.069	0.084	-0.015
Some college	0.062	0.073	-0.011
College/+	0.073	0.094	-0.020
Lipid Lowering Medication	0.104	0.146	-0.041
Depression Medication	0.065	0.103	-0.038
Diabetes	0.103	0.183	-0.080
Fully Adjusted			
Intercept	0.147	0.138	0.009
Somatic Symptoms	0.010	0.011	-0.001
Gender	0.046	0.045	0.000
Age	0.002	0.003	-0.001
Ethnicity			
Mexican- American	0.064	0.083	-0.020
Other Hispanic	0.079	0.095	-0.015
Non-Hispanic Black	0.058	0.065	-0.006
Other Ethnicity	0.098	0.096	0.001
Education			
Less than 9th	0.102	0.112	-0.010
9^{th} through 12^{th}	0.062	0.078	-0.016
Some college	0.060	0.072	-0.012
College/+	0.070	0.090	-0.019
Lipid Lowering Medication	0.102	0.144	-0.042
Depression Medication	0.061	0.096	-0.035
Diabetes	0.104	0.157	-0.053
Current Smoker	0.048	0.056	-0.008
Heavier Alcohol Use	0.067	0.083	-0.015
Moderate Alcohol Use	0.044	0.054	-0.011
BMI	0.003	0.003	0.000

Note: L_{Bayes} refers to the length of the fully-Bayesian 95% BCI, $L_{Classical}$ refers to the length of the Classical 95% CI, and L_{Δ} refers to the difference between L_{Bayes} and $L_{Classical}$.

Table 6. Nonsomatic Symptom Severity and $\log_{10}$(CRP): Lengths of confidence and credible intervals.

	L_{Bayes}	$L_{Classical}$	L_{Δ}
Adjusted			
Intercept	0.302	0.356	-0.054
Nonsomatic Symptoms	0.024	0.028	-0.004
Gender	0.130	0.151	-0.021
Age	0.007	0.009	-0.002
Ethnicity			
Mexican-American	0.188	0.157	0.032
Other Hispanic	0.211	0.283	-0.072
Non-Hispanic Black	0.186	0.223	-0.037
Other Ethnicity	0.295	0.250	0.045
Education			
Less than 9th	0.300	0.328	-0.029
9^{th} through 12^{th}	0.194	0.234	-0.041
Some college	0.162	0.198	-0.036
College/+	0.204	0.232	-0.028
Lipid Lowering Medication	0.308	0.410	-0.102
Depression Medication	0.196	0.155	0.041
Diabetes	0.290	0.401	-0.111
Fully Adjusted			
Intercept	0.345	0.459	0.032
Nonsmatic Symptoms	0.021	0.023	-0.072
Gender	0.107	0.141	-0.037
Age	0.006	0.007	0.045
Ethnicity			
Mexican- American	0.165	0.134	-0.029
Other Hispanic	0.190	0.292	-0.041
Non-Hispanic Black	0.150	0.146	-0.036
Other Ethnicity	0.252	0.210	-0.028
Education			
Less than 9th	0.247	0.272	-0.102
9^{th} through 12^{th}	0.170	0.151	0.041
Some college	0.144	0.156	-0.111
College/+	0.179	0.172	0.032
Lipid Lowering Medication	0.262	0.323	-0.072
Depression Medication	0.173	0.119	-0.037
Diabetes	0.283	0.278	0.045
Current Smoker	0.117	0.144	-0.029
Heavier Alcohol Use	0.175	0.214	-0.041
Moderate Alcohol Use	0.125	0.161	-0.036
BMI	0.007	0.009	-0.028

Note: L_{Bayes} refers to the length of the fully-Bayesian 95% BCI, $L_{Classical}$ refers to the length of the Classical 95% CI, and L_{Δ} refers to the difference between L_{Bayes} and $L_{Classical}$.

Table 7. Nonsomatic Symptom Severity and $\log_{10}$(MHR): Lengths of confidence and credible intervals.

	L_{Bayes}	$L_{Classical}$	L_{Δ}
Adjusted			
Intercept	0.108	0.140	-0.033
Nonsomatic Symptoms	0.008	0.011	-0.003
Gender	0.043	0.044	-0.001
Age	0.002	0.003	-0.001
Ethnicity			
Mexican-American	0.064	0.086	-0.022
Other Hispanic	0.075	0.096	-0.021
Non-Hispanic Black	0.067	0.065	0.002
Other Ethnicity	0.105	0.091	0.014
Education			
Less than 9th	0.099	0.117	-0.019
9^{th} through 12^{th}	0.069	0.085	-0.015
Some college	0.059	0.074	-0.016
College/+	0.077	0.093	-0.016
Lipid Lowering Medication	0.112	0.147	-0.035
Depression Medication	0.069	0.106	-0.037
Diabetes	0.102	0.176	-0.074
Fully Adjusted			
Intercept	0.138	0.134	0.004
Nonsomatic Symptoms	0.007	0.010	-0.003
Gender	0.049	0.045	0.004
Age	0.002	0.003	-0.001
Ethnicity			
Mexican- American	0.066	0.083	-0.018
Other Hispanic	0.070	0.093	-0.023
Non-Hispanic Black	0.062	0.063	-0.001
Other Ethnicity	0.098	0.095	0.002
Education			
Less than 9th	0.101	0.112	-0.011
9^{th} through 12^{th}	0.070	0.078	-0.008
Some college	0.058	0.073	-0.015
College/+	0.072	0.089	-0.017
Lipid Lowering Medication	0.098	0.146	-0.047
Depression Medication	0.066	0.097	-0.030
Diabetes	0.106	0.155	-0.049
Current Smoker	0.046	0.057	-0.011
Heavier Alcohol Use	0.070	0.081	-0.011
Moderate Alcohol Use	0.049	0.053	-0.004
BMI	0.003	0.003	0.000

Note: L_{Bayes} refers to the length of the fully-Bayesian 95% BCI, $L_{Classical}$ refers to the length of the Classical 95% CI, and L_{Δ} refers to the difference between L_{Bayes} and $L_{Classical}$.

Table 8. Presence of Depression and $\log_{10}$(CRP): Model Estimates.

	Classical			**Fully-Bayesian**		
		95% Confidence Interval			95% Credible Interval	
	Coefficient Estimate	Lower Bound	Upper Bound	Coefficient Estimate	Lower Bound	Upper Bound
Adjusted						
Intercept	-1.060	-1.142	-0.979	-0.695	-0.871	-0.507
Presence of Depression	0.070	0.025	0.114	0.047	0.008	0.084
Gender	0.140	0.100	0.179	0.186	0.155	0.216
Age	0.004	0.003	0.006	0.005	0.003	0.006
Ethnicity						
Mexican- American	0.097	0.047	0.148	0.081	0.040	0.125
Other Hispanic	0.036	-0.044	0.116	0.006	-0.044	0.059
Non-Hispanic Black	0.097	0.044	0.150	0.083	0.041	0.127
Other Ethnicity	-0.096	-0.169	-0.023	-0.073	-0.144	-0.003
Education						
Less than 9th	-0.039	-0.125	0.046	-0.046	-0.116	0.025
9^{th} through 12^{th}	0.014	-0.049	0.078	-0.015	-0.070	0.037
Some college	-0.039	-0.078	0.000	-0.068	-0.110	-0.025
College/+	-0.140	-0.188	-0.092	-0.169	-0.213	-0.124
Lipid Lowering Medication	-0.040	-0.110	0.031	-0.026	-0.101	0.053
Depression Medication	0.078	0.029	0.127	0.051	-0.010	0.115
Diabetes	0.237	0.133	0.340	0.187	0.112	0.266
Fully Adjusted						
Intercept	-2.270	-2.386	-2.155	-2.223	-2.312	-2.139
Presence of Depression	0.024	-0.017	0.064	0.005	-0.027	0.039
Gender	0.177	0.137	0.217	0.193	0.166	0.221
Age	0.002	0.000	0.004	0.002	0.001	0.004
Ethnicity						
Mexican- American	0.068	0.024	0.111	0.050	0.010	0.089
Other Hispanic	0.038	-0.035	0.112	0.014	-0.039	0.065
Non-Hispanic Black	0.014	-0.030	0.059	0.008	-0.027	0.046
Other Ethnicity	-0.054	-0.120	0.011	-0.047	-0.106	0.017
Education						
Less than 9th	-0.024	-0.098	0.050	-0.025	-0.080	0.033
9^{th} through 12^{th}	0.002	-0.043	0.048	-0.018	-0.062	0.027
Some college	-0.034	-0.071	0.003	-0.063	-0.102	-0.027
College/+	-0.047	-0.088	-0.006	-0.082	-0.124	-0.041
Lipid Lowering Medication	-0.067	-0.131	-0.003	-0.061	-0.128	0.006
Depression Medication	0.050	0.003	0.098	0.023	-0.031	0.082
Diabetes	0.047	-0.055	0.149	0.034	-0.035	0.103
Current Smoker	0.103	0.071	0.135	0.083	0.049	0.115
Heavier Alcohol Use	0.065	0.008	0.121	0.052	0.004	0.098
Moderate Alcohol Use	-0.004	-0.041	0.032	0.003	-0.026	0.030
BMI	0.044	0.041	0.047	0.043	0.041	0.045

Table 9. Presence of Depression and $\log_{10}$(MHR): Model Estimates.

	Classical			**Fully-Bayesian**		
		95% Confidence Interval			95% Credible Interval	
	Coefficient Estimate	Lower Bound	Upper Bound	Coefficient Estimate	Lower Bound	Upper Bound
Adjusted						
Intercept	-1.822	-1.848	-1.797	-1.824	-1.848	-1.800
Presence of Depression	0.021	0.004	0.038	0.022	0.010	0.033
Gender	-0.138	-0.150	-0.126	-0.126	-0.136	-0.115
Age	-0.002	-0.003	-0.001	-0.002	-0.003	-0.001
Ethnicity						
Mexican- American	-0.015	-0.032	0.002	-0.016	-0.030	-0.002
Other Hispanic	-0.014	-0.038	0.010	-0.019	-0.038	0.002
Non-Hispanic Black	-0.096	-0.115	-0.077	-0.095	-0.110	-0.080
Other Ethnicity	-0.018	-0.048	0.012	-0.024	-0.048	0.000
Education						
Less than 9th	0.010	-0.019	0.038	0.006	-0.019	0.029
9^{th} through 12^{th}	0.004	-0.016	0.024	0.000	-0.018	0.018
Some college	-0.019	-0.038	0.001	-0.019	-0.034	-0.005
College/+	-0.065	-0.088	-0.042	-0.061	-0.075	-0.045
Lipid Lowering Medication	0.035	0.009	0.061	0.033	0.007	0.058
Depression Medication	-0.005	-0.026	0.017	-0.004	-0.025	0.018
Diabetes	0.033	0.001	0.065	0.029	0.002	0.059
Fully Adjusted						
Intercept	-2.043	-2.080	-2.005	-2.023	-2.057	-1.991
Presence of Depression	0.010	-0.006	0.025	0.012	0.000	0.024
Gender	-0.139	-0.152	-0.127	-0.133	-0.144	-0.123
Age	-0.002	-0.003	-0.002	-0.002	-0.003	-0.002
Ethnicity						
Mexican- American	-0.021	-0.037	-0.006	-0.020	-0.035	-0.006
Other Hispanic	-0.017	-0.037	0.003	-0.018	-0.036	-0.002
Non-Hispanic Black	-0.119	-0.136	-0.101	-0.113	-0.126	-0.100
Other Ethnicity	-0.021	-0.052	0.010	-0.027	-0.053	-0.001
Education						
Less than 9th	0.013	-0.013	0.040	0.011	-0.013	0.033
9^{th} through 12^{th}	0.000	-0.018	0.018	-0.002	-0.018	0.015
Some college	-0.014	-0.032	0.005	-0.013	-0.028	0.000
College/+	-0.038	-0.058	-0.017	-0.035	-0.049	-0.020
Lipid Lowering Medication	0.027	-0.002	0.055	0.023	-0.001	0.047
Depression Medication	-0.009	-0.027	0.010	-0.008	-0.029	0.012
Diabetes	-0.006	-0.036	0.025	0.003	-0.020	0.029
Current Smoker	0.057	0.042	0.071	0.052	0.039	0.064
Heavier Alcohol Use	-0.069	-0.086	-0.053	-0.057	-0.073	-0.040
Moderate Alcohol Use	-0.041	-0.054	-0.029	-0.036	-0.047	-0.025
BMI	0.009	0.008	0.010	0.008	0.007	0.009

Table 10. Somatic Symptom Severity and $\log_{10}$(CRP): Model Estimates.

	Classical			**Fully-Bayesian**		
		95% Confidence Interval			95% Credible Interval	
	Coefficient Estimate	Lower Bound	Upper Bound	Coefficient Estimate	Lower Bound	Upper Bound
Adjusted						
Intercept	-1.016	-1.185	-0.847	-1.035	-1.198	-0.884
Somatic Symptoms	0.029	0.011	0.046	0.021	0.006	0.035
Gender	0.145	0.068	0.223	0.158	0.098	0.222
Age	0.003	-0.002	0.007	0.004	0.000	0.007
Ethnicity						
Mexican-American	0.031	-0.044	0.106	0.043	-0.048	0.135
Other Hispanic	-0.048	-0.192	0.095	-0.022	-0.128	0.096
Non-Hispanic Black	-0.015	-0.127	0.098	0.013	-0.083	0.106
Other Ethnicity	-0.113	-0.239	0.013	-0.069	-0.223	0.072
Education						
Less than 9th	-0.056	-0.210	0.098	-0.046	-0.191	0.108
9^{th} through 12^{th}	-0.019	-0.134	0.096	-0.060	-0.150	0.037
Some college	-0.059	-0.157	0.039	-0.040	-0.120	0.041
College/+	-0.209	-0.324	-0.093	-0.226	-0.332	-0.119
Lipid Lowering Medication	0.022	-0.173	0.217	0.016	-0.131	0.168
Depression Medication	0.011	-0.065	0.088	0.013	-0.087	0.114
Diabetes	0.326	0.118	0.535	0.246	0.090	0.405
Fully Adjusted						
Intercept	-2.211	-2.438	-1.983	-2.218	-2.403	-2.046
Somatic Symptoms	0.016	0.000	0.032	0.012	0.000	0.023
Gender	0.146	0.074	0.219	0.143	0.086	0.197
Age	0.001	-0.003	0.004	0.002	-0.001	0.005
Ethnicity						
Mexican- American	-0.015	-0.081	0.050	-0.001	-0.082	0.073
Other Hispanic	-0.006	-0.155	0.144	0.023	-0.076	0.130
Non-Hispanic Black	-0.063	-0.137	0.011	-0.038	-0.110	0.036
Other Ethnicity	-0.080	-0.183	0.024	-0.029	-0.148	0.089
Education						
Less than 9th	-0.023	-0.155	0.108	-0.014	-0.134	0.098
9^{th} through 12^{th}	-0.040	-0.114	0.034	-0.052	-0.132	0.026
Some college	-0.063	-0.142	0.016	-0.038	-0.106	0.040
College/+	-0.144	-0.229	-0.058	-0.166	-0.249	-0.076
Lipid Lowering Medication	-0.041	-0.195	0.114	-0.045	-0.187	0.084
Depression Medication	0.023	-0.036	0.083	0.010	-0.074	0.093
Diabetes	0.066	-0.076	0.207	0.017	-0.119	0.145
Current Smoker	0.028	-0.043	0.099	0.045	-0.015	0.103
Heavier Alcohol Use	0.049	-0.059	0.157	0.036	-0.051	0.125
Moderate Alcohol Use	0.017	-0.060	0.093	0.019	-0.042	0.078
BMI	0.046	0.041	0.050	0.044	0.040	0.048

Table 11. Somatic Symptom Severity and $\log_{10}$(MHR): Model Estimates.

	Classical			**Fully-Bayesian**		
		95% Confidence Interval			95% Credible Interval	
	Coefficient Estimate	Lower Bound	Upper Bound	Coefficient Estimate	Lower Bound	Upper Bound
Adjusted						
Intercept	-1.832	-1.897	-1.766	-1.833	-1.887	-1.783
Somatic Symptoms	0.006	0.001	0.012	0.006	0.001	0.010
Gender	-0.134	-0.157	-0.111	-0.126	-0.148	-0.103
Age	-0.002	-0.003	0.000	-0.002	-0.003	-0.001
Ethnicity						
Mexican-American	-0.037	-0.080	0.005	-0.036	-0.068	-0.001
Other Hispanic	-0.024	-0.072	0.025	-0.037	-0.074	0.001
Non-Hispanic Black	-0.105	-0.139	-0.070	-0.105	-0.137	-0.072
Other Ethnicity	0.006	-0.041	0.053	-0.008	-0.056	0.044
Education						
Less than 9th	-0.005	-0.065	0.054	-0.007	-0.059	0.042
9^{th} through 12^{th}	0.004	-0.038	0.046	-0.005	-0.040	0.030
Some college	-0.030	-0.066	0.007	-0.017	-0.047	0.014
College/+	-0.081	-0.128	-0.034	-0.064	-0.102	-0.029
Lipid Lowering Medication	-0.001	-0.074	0.072	-0.002	-0.055	0.049
Depression Medication	-0.005	-0.056	0.047	-0.013	-0.047	0.019
Diabetes	0.063	-0.029	0.154	0.045	-0.004	0.099
Fully Adjusted						
Intercept	-2.034	-2.103	-1.964	-2.014	-2.090	-1.942
Somatic Symptoms	0.002	-0.003	0.008	0.003	-0.002	0.008
Gender	-0.142	-0.165	-0.120	-0.135	-0.158	-0.112
Age	-0.002	-0.003	0.000	-0.002	-0.003	-0.001
Ethnicity						
Mexican- American	-0.041	-0.083	0.001	-0.036	-0.067	-0.004
Other Hispanic	-0.016	-0.063	0.032	-0.026	-0.067	0.012
Non-Hispanic Black	-0.115	-0.148	-0.083	-0.111	-0.141	-0.082
Other Ethnicity	-0.002	-0.050	0.046	-0.004	-0.055	0.043
Education						
Less than 9th	0.002	-0.055	0.058	-0.002	-0.052	0.050
9^{th} through 12^{th}	0.001	-0.038	0.040	-0.003	-0.034	0.028
Some college	-0.023	-0.059	0.013	-0.011	-0.041	0.018
College/+	-0.052	-0.097	-0.007	-0.041	-0.076	-0.006
Lipid Lowering Medication	-0.019	-0.091	0.053	-0.017	-0.069	0.033
Depression Medication	-0.002	-0.050	0.046	-0.013	-0.043	0.018
Diabetes	0.020	-0.059	0.099	0.014	-0.038	0.067
Current Smoker	0.052	0.024	0.081	0.047	0.023	0.072
Heavier Alcohol Use	-0.063	-0.104	-0.021	-0.042	-0.075	-0.008
Moderate Alcohol Use	-0.037	-0.064	-0.010	-0.025	-0.046	-0.003
BMI	0.008	0.007	0.010	0.007	0.005	0.008

Table 12. Nonsomatic Symptom Severity and $\log_{10}$(CRP): Model Estimates.

	Classical			**Fully-Bayesian**		
		95% Confidence Interval			95% Credible Interval	
	Coefficient Estimate	Lower Bound	Upper Bound	Coefficient Estimate	Lower Bound	Upper Bound
Adjusted						
Intercept	-0.879	-1.057	-0.702	-0.926	-1.080	-0.778
Nonsomatic Symptoms	-0.007	-0.022	0.007	-0.008	-0.020	0.005
Gender	0.166	0.091	0.242	0.175	0.108	0.239
Age	0.003	-0.001	0.008	0.004	0.001	0.008
Ethnicity						
Mexican-American	0.031	-0.047	0.110	0.039	-0.057	0.132
Other Hispanic	-0.039	-0.181	0.102	-0.022	-0.130	0.081
Non-Hispanic Black	0.008	-0.104	0.119	0.026	-0.066	0.120
Other Ethnicity	-0.120	-0.245	0.005	-0.076	-0.221	0.074
Education						
Less than 9th	-0.050	-0.214	0.115	-0.045	-0.193	0.107
9^{th} through 12^{th}	-0.020	-0.137	0.097	-0.060	-0.159	0.035
Some college	-0.060	-0.159	0.038	-0.046	-0.123	0.039
College/+	-0.231	-0.347	-0.115	-0.248	-0.352	-0.148
Lipid Lowering Medication	0.015	-0.190	0.220	0.014	-0.135	0.173
Depression Medication	0.019	-0.058	0.097	0.025	-0.073	0.123
Diabetes	0.336	0.136	0.537	0.247	0.104	0.393
Fully Adjusted						
Intercept	-2.139	-2.369	-1.909	-2.162	-2.345	-2.000
Nonsomatic Symptoms	-0.005	-0.016	0.007	-0.004	-0.015	0.006
Gender	0.156	0.085	0.226	0.151	0.097	0.204
Age	0.001	-0.003	0.005	0.002	0.000	0.005
Ethnicity						
Mexican- American	-0.014	-0.081	0.053	-0.002	-0.084	0.081
Other Hispanic	0.001	-0.146	0.147	0.026	-0.069	0.121
Non-Hispanic Black	-0.051	-0.124	0.023	-0.028	-0.103	0.047
Other Ethnicity	-0.086	-0.191	0.019	-0.033	-0.151	0.101
Education						
Less than 9th	-0.020	-0.156	0.116	-0.009	-0.129	0.118
9^{th} through 12^{th}	-0.041	-0.116	0.034	-0.053	-0.136	0.034
Some college	-0.062	-0.140	0.016	-0.040	-0.110	0.033
College/+	-0.151	-0.237	-0.065	-0.174	-0.264	-0.084
Lipid Lowering Medication	-0.045	-0.207	0.116	-0.045	-0.176	0.086
Depression Medication	0.029	-0.031	0.088	0.019	-0.068	0.105
Diabetes	0.070	-0.069	0.209	0.014	-0.126	0.157
Current Smoker	0.037	-0.035	0.109	0.052	-0.007	0.110
Heavier Alcohol Use	0.042	-0.065	0.149	0.035	-0.057	0.118
Moderate Alcohol Use	0.004	-0.076	0.084	0.013	-0.046	0.079
BMI	0.046	0.042	0.050	0.044	0.041	0.048

Table 13. Nonsomatic Symptom Severity and $\log_{10}$(MHR): Model Estimates.

	Classical			Fully-Bayesian		
		95% Confidence Interval			95% Credible Interval	
	Coefficient Estimate	Lower Bound	Upper Bound	Coefficient Estimate	Lower Bound	Upper Bound
Adjusted						
Intercept	-1.810	-1.880	-1.740	-1.818	-1.872	-1.764
Nonsomatic Symptoms	0.002	-0.004	0.007	0.002	-0.002	0.006
Gender	-0.130	-0.152	-0.108	-0.122	-0.144	-0.101
Age	-0.002	-0.003	0.000	-0.002	-0.003	0.000
Ethnicity						
Mexican-American	-0.037	-0.080	0.006	-0.036	-0.069	-0.005
Other Hispanic	-0.023	-0.071	0.025	-0.037	-0.075	0.000
Non-Hispanic Black	-0.102	-0.134	-0.069	-0.103	-0.137	-0.070
Other Ethnicity	0.003	-0.042	0.049	-0.009	-0.063	0.042
Education						
Less than 9th	-0.005	-0.064	0.053	-0.006	-0.054	0.045
9^{th} through 12^{th}	0.003	-0.040	0.045	-0.005	-0.040	0.029
Some college	-0.031	-0.068	0.006	-0.016	-0.045	0.013
College/+	-0.085	-0.131	-0.038	-0.066	-0.104	-0.027
Lipid Lowering Medication	-0.002	-0.075	0.071	-0.006	-0.061	0.051
Depression Medication	-0.006	-0.059	0.047	-0.013	-0.048	0.021
Diabetes	0.065	-0.023	0.153	0.046	-0.004	0.098
Fully Adjusted						
Intercept	-2.029	-2.097	-1.962	-2.007	-2.075	-1.937
Nonsomatic Symptoms	0.002	-0.003	0.007	0.002	-0.002	0.006
Gender	-0.141	-0.164	-0.119	-0.133	-0.159	-0.110
Age	-0.002	-0.003	0.000	-0.002	-0.003	-0.001
Ethnicity						
Mexican- American	-0.041	-0.083	0.001	-0.037	-0.070	-0.005
Other Hispanic	-0.016	-0.062	0.031	-0.027	-0.060	0.010
Non-Hispanic Black	-0.115	-0.147	-0.084	-0.112	-0.145	-0.083
Other Ethnicity	-0.003	-0.051	0.044	-0.007	-0.055	0.043
Education						
Less than 9th	0.001	-0.055	0.057	-0.004	-0.054	0.047
9^{th} through 12^{th}	0.000	-0.039	0.039	-0.004	-0.038	0.031
Some college	-0.023	-0.060	0.013	-0.011	-0.041	0.017
College/+	-0.053	-0.097	-0.008	-0.042	-0.080	-0.008
Lipid Lowering Medication	-0.019	-0.092	0.053	-0.018	-0.067	0.031
Depression Medication	-0.003	-0.051	0.045	-0.015	-0.047	0.019
Diabetes	0.021	-0.057	0.098	0.014	-0.037	0.068
Current Smoker	0.053	0.024	0.081	0.047	0.024	0.070
Heavier Alcohol Use	-0.064	-0.105	-0.024	-0.042	-0.077	-0.007
Moderate Alcohol Use	-0.038	-0.064	-0.012	-0.026	-0.050	-0.001
BMI	0.008	0.007	0.010	0.007	0.005	0.008

Figure 1. Point estimates for models predicting $\log_{10}$(CRP). Estimates for the coefficients of the fully-adjusted models predicting transformed CRP, and their respective standard errors, are plotted for both the fully-Bayesian and Classical estimation methods. Depression, Nonsomatic Symptoms, and Somatic Symptoms correspond to fully-adjusted models using presence of depression, nonsomatic symptom severity, and somatic symptom severity, respectively, as predictors.

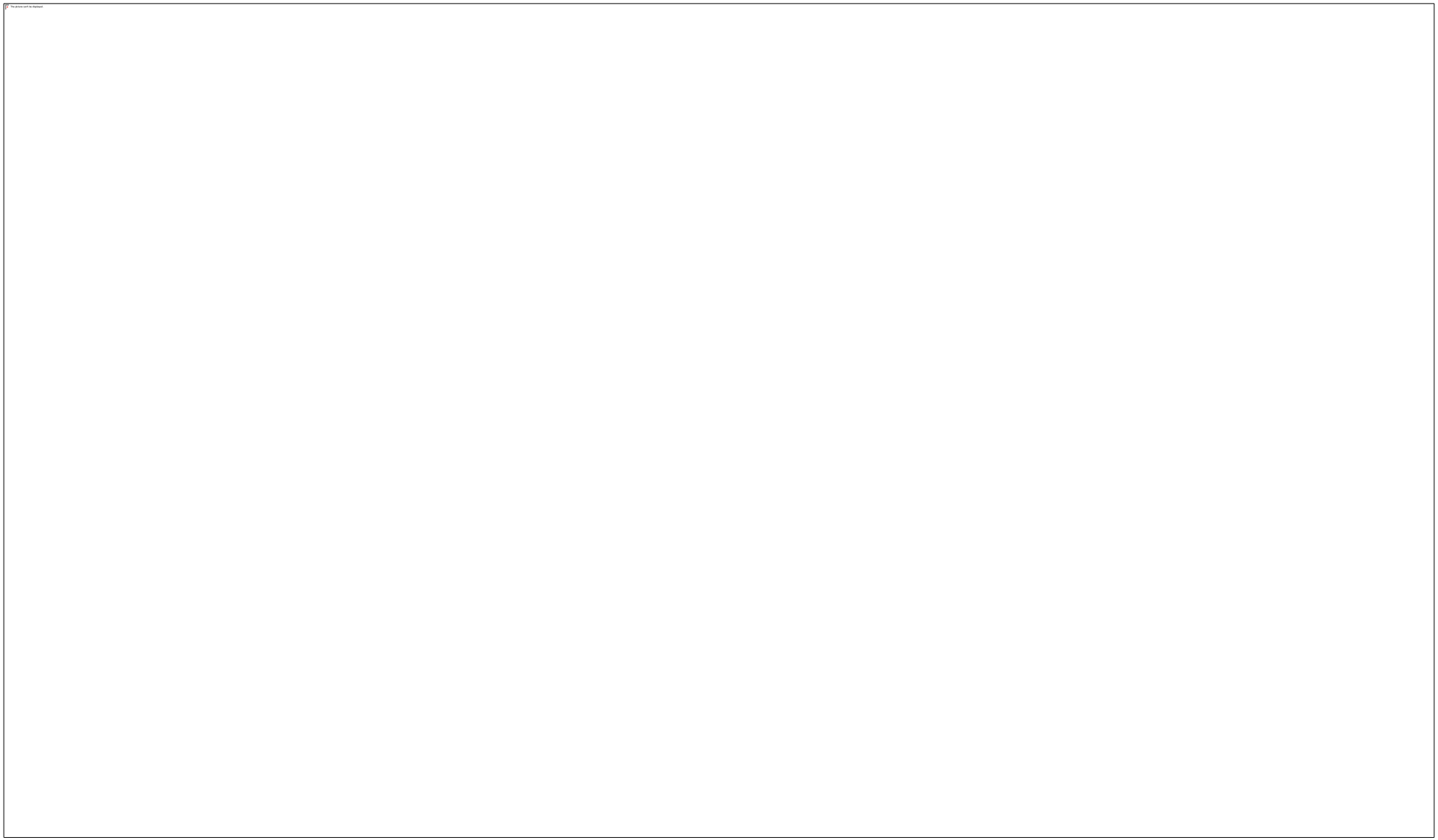

Figure 2. Point estimates for models predicting $\log_{10}$(MHR). Estimates for the coefficients of the fully-adjusted models predicting transformed MHR, and their respective standard errors, are plotted for both the fully-Bayesian and Classical estimation methods. Depression, Nonsomatic Symptoms, and Somatic Symptoms correspond to fully-adjusted models using presence of depression, nonsomatic symptom severity, and somatic symptom severity, respectively, as predictors.

Figure 3. Differences Between Fully-Bayesian and Classical Coefficient Estimates. Both figures show the difference between the absolute values of the coefficients estimated from the fully-Bayesian (Bayes) and Classical (WLS) methods. Negative values indicate that point estimates generated by WLS are larger than in magnitude than point estimates generated by the fully-Bayes method. A) Difference between coefficient estimates for fully-adjusted models predicting transformed CRP values. B) Difference between coefficient estimates for fully-adjusted models predicting transformed MHR values.

Figure 4. Difference between length of fully-Bayesian 95% BCI and Classical 95% CI. Both figures show the difference between the lengths of the fully-Bayesian 95% credible intervals (L_{Bayes}) and the 95% confidence intervals estimated using TSL (L_{TSL}) for each variable, except intercepts, in the fully-adjusted models. Negative values indicate that the length of the Classical 95% CI is larger than the length of the fully-Bayesian 95% BCI, whereas positive values indicate that the length of the Classical 95% CI is smaller than the length of the fully-Bayesian 95% BCI. A) Differences between interval lengths for coefficients in the fully-adjusted models using presence of depression, somatic symptom severity, and nonsomatic symptom severity to predict transformed CRP. The mean difference values are -0.013, -0.013, and -0.010 respectively. B) The mean difference between interval lengths for coefficients in the fully-adjusted models using presence of depression, somatic symptom severity, and nonsomatic symptom severity to predict transformed CRP values is -0.013, -0.013, and -0.010 respectively.

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