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Research paper

Demographic, behavioral, dietary, and clinical predictors of high-sensitivity C-reactive protein: The National Health and Nutrition Examination Surveys (NHANES)☆

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ABSTRACT

Aims: High-sensitivity C-reactive protein (hs-CRP), a marker of inflammation, is associated with atherosclerosis, and recent studies indicate that therapies targeting inflammation are associated with reductions in cardiovascular risk. However, factors predictive of elevated hs-CRP in the general population have not been elucidated.
Methods: In this cross-sectional study, multivariable logistic regression analysis was used to identify independent predictors of elevated hs-CRP (≥3 mg/L) utilizing the National Health and Nutrition Examination Survey (NHANES) 2015–2016 cycle. The model was verified using the independent NHANES 2017–2018 cycle. Candidate variables comprised demographic, behavioral, dietary, and clinical factors. The study included 5412 adults from the 2015–2016 cohort and 5856 adults from the 2017–2018 cohort.
Results: Significant independent predictors of elevated hs-CRP included: older age (OR 1.09 per decade; 95 % CI 1.03–1.14; P = 0.024), female sex (OR 1.57; 95 % CI 1.36–1.80; P = 0.003), Black vs White race (OR 1.31; 95 % CI 1.10–1.56; P = 0.037), increased BMI (OR 1.12 per kg/m^2; 95 % CI 1.10–1.14; P < 0.001), elevated white blood cell count (OR 1.21 per 1000 white blood cells/μL; 95 % CI 1.15–1.28; P = 0.002), and self-reported poor vs excellent health (OR 1.73; 95 % CI 1.04–2.22; P = 0.012). The model had excellent discrimination with a c-statistic of 0.77 in the 2015–2016 cycle and 0.76 in the 2017–2018 cycle.
Conclusion: Older age, female sex, Black race, increased BMI, higher white blood cell count, and self-reported poor health were independent predictors of elevated hs-CRP levels. Additional studies are needed to determine if behavioral modifications can lower hs-CRP and whether this translates to reduced risk for cardiovascular disease and other conditions associated with chronic inflammation.

1. Introduction

Cardiovascular disease (CVD) is the leading cause of death in the United States, accounting for 859,125 deaths in 2017 [1]. Of these, 365,914 deaths were due to coronary heart disease (CHD), primarily due to atherosclerosis [1,2]. Inflammation is a key contributor to atherosclerosis and therapies that target inflammation have been shown to induce regression of atherosclerotic plaques, which in turn decreases the number of atherosclerotic cardiovascular (CV) events [3–6]. Understanding demographic, behavioral, dietary and clinical factors associated with chronic inflammation may provide insights into additional potential therapeutic targets to decrease inflammation and reduce the burden of CVD and other conditions associated with chronic inflammation, including cancer, Alzheimer's disease, and frailty [7,8].

High sensitivity C-reactive protein (hs-CRP), a circulating acute-phase protein used as a marker of inflammation, has been shown to have a moderate association with CVD [9]. Previous analysis of Norwegian patients with a history of coronary events found that elevated hs-

☆ The authors affirm that this is original work and that no part of this study has been published previously or is under consideration for publication elsewhere. However, an abstract describing portions of this analysis was presented virtually at the European Society of Cardiology Preventive Cardiology conference on April 7, 2022.

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CRP was associated with smoking, higher BMI, and anxiety [10]. However, factors predictive of elevated hs-CRP in the general United States (US) population have not been elucidated.

The objective of the current study, therefore, was to identify independent predictors of hs-CRP levels in an ambulatory adult population in the US. We hypothesized that in addition to demographic factors, potentially modifiable behavioral, dietary, and clinical factors would be associated with elevated hs-CRP. Identification of such factors could then serve as the basis for future studies designed to target specific risk factors or to identify subjects who might be suitable candidates for novel anti-inflammatory therapies.

2. Methods

2.1. Study population and design

This cross-sectional study was conducted utilizing the two most recent fully completed cycles (2015–2016 and 2017–2018) of the National Health and Nutrition Examination Survey (NHANES) [11]. NHANES includes demographic information, clinical diagnoses, objective examination findings, laboratory data, and interviews assessing participants' behavioral and dietary choices. NHANES datasets are publicly available de-identified surveys with informed consent obtained from all participants. NHANES samples are scientifically selected to ensure representativeness of the larger US population, with oversampling of specific subgroups to increase statistical precision [12].

The study included 5412 adults aged 18 or older from the NHANES 2015–2016 cohort and 5856 adults from the NHANES 2017–2018 cohort. The primary outcome measure was hs-CRP level, which was dichotomized at the clinical threshold of 3 mg/dL indicative of low-grade chronic inflammation [13]. Candidate variables used for univariate analysis comprised demographic, behavioral, and clinical factors, including standard CV risk factors. To simplify interpretation, age was modeled as a tripartite categorical variable (18–39, 40–64, 65 and older). Metabolic activity score was measured in minutes per week and categorized as “low” for a score under 500, “moderate” for 500–1000, and “high” for a score of 1000 or higher.

Dietary and nutritional data were available for a subsample of participants. The total number of subjects who answered dietary and nutritional surveys were 4760 (88.0 %) and 4982 (85.1 %) for the 2015–2016 and 2017–2018 cycles, respectively. Dietary intake data were obtained through participant recall of the prior 24 h. Due to skewed distributions of individual food items, they were recoded as categorical variables. To assess overall diet quality, we used the 2015 Healthy Eating Index (HEI) from the US Department of Health and Human Services as the standard of comparison and used HEI as a candidate variable to model hs-CRP [14].

2.2. Statistical analysis

Both the outcome (hs-CRP) and prediction variables had some missing values. All variables except for triglycerides and LDL-cholesterol, which were only collected on a subset of patients examined during a morning session, were imputed using multiple imputation. The variables were imputed multivariately using chained equation algorithm as implemented in MICE package in R software [15,16] Outlier predictor variables were Winsorized at 1st and 99th percentile [17]. All analyses were performed with inverse-probability weighting to account for the survey data structure using *survey* package in R. The weights of the smallest subpopulation that included all the variables were used as recommended.

From the univariate analysis, variables with P-value <0.05 for 2015–2016 were offered into the multivariable models. Multivariable generalized linear models with quasi-binomial distribution were used to identify independent predictors of elevated hs-CRP ($\geq$3 mg/dL) utilizing the NHANES 2015–2016 cycle (derivation set). The models were verified utilizing the independent NHANES 2017–2018 cycle (validation set). Three models were created: 1) demographic, clinical, and laboratory multivariable regression model, 2) nutritional multivariable regression model, 3) dietary multivariable regression model. It was necessary to develop separate models based on diet and nutrition because only a subsample of subjects answered the dietary and nutrition questions. The dietary model tested specific food items (i.e. fruits, vegetables, meat, etc.) and the nutritional model tested specific dietary nutrients (i.e. total calories, grams of sugar, fiber, vitamin E, etc.). In these models, hs-CRP was treated as a binary outcome variable, with values $<$3 mg/dL considered normal (low) and values of 3 mg/dL or higher considered elevated. In the models, odds ratios >1 indicate factors associated with increased risk of elevated hs-CRP and odds ratios <1 indicate factors associated with lower risk. Model discrimination was assessed using area under the receiver-operator characteristic curve (c-statistic).

3. Results

3.1. Participants

The final study sample for the derivation set included 5412 adults aged $\geq$18 years (2613 men [48 %]); 2799 women [52 %]; mean [SD] age, 48.1 [18.5] years. For the validation set, the sample included 5856 participants (2840 men [48 %]; 3016 women [52 %]; mean [SD] 49.9 [18.9] years). As shown in Table 1, the two cohorts were similar with

Table 1
Sample characteristics.

	2015–2016	2017–2018	P-value
n	5412	5856	
Male (%)	2613 (48.3)	2840 (48.5)	0.83
Age (mean (SD))	48.10 (18.53)	49.89 (18.78)	0.85
Race (%)			0.97
White	1731 (32.0)	2032 (34.7)	
Black	1138 (21.0)	1343 (22.9)	
Hispanic	1686 (31.2)	1335 (22.8)	
Other	857 (15.8)	1146 (19.6)	
Ratio family income to poverty (%)			0.63
0–1	1242 (22.9)	1143 (19.5)	
1–2	1509 (27.9)	1647 (28.1)	
2–3	923 (17.1)	989 (16.9)	
3+	1738 (32.1)	2077 (35.5)	
BMI, kg/m^2	29.39 (7.04)	29.71 (7.42)	0.26
Waist circumference, cm	99.80 (16.90)	100.42 (17.42)	0.54
Systolic BP, mm Hg	124.89 (18.47)	126.30 (20.28)	0.94
Diastolic BP, mm Hg	69.58 (11.90)	72.04 (12.60)	0.01
HDL cholesterol, mg/dL	53.9 (16.6)	53.2 (14.7)	0.05
Total cholesterol, mg/dL	188.8 (39.5)	186.6 (39.8)	0.10
White blood count, 1000 cells/μL	7.00 [5.80, 8.50]	7.00 [5.70, 8.50]	0.51
Platelet count, 1000 cells/μL	239.42 (59.59)	242.83 (62.35)	0.05
General health			0.68
Fair/poor	1345 (24.9)	1447 (24.7)	
Good	2267 (41.9)	2472 (42.2)	
Excellent/very good	1800 (33.3)	1937 (33.1)	
Arthritis	1342 (24.8)	1704 (29.1)	0.57
Congestive heart failure	190 (3.5)	204 (3.5)	0.95
Coronary heart disease	220 (4.1)	266 (4.5)	0.35
Angina	120 (2.2)	163 (2.8)	0.15
Heart attack	226 (4.2)	273 (4.7)	0.63
Liver condition	239 (4.4)	307 (5.2)	0.11
COPD	174 (3.2)	297 (5.1)	0.09
Cancer	494 (9.1)	593 (10.1)	0.72
MET score, min/week	1200 [0, 4320]	1320 [0, 4800]	0.01
Statin	978 (18.1)	1200 (20.5)	0.86
Metformin	496 (9.2)	606 (10.3)	0.65
Smoking, current	994 (18.4)	1021 (17.4)	0.44

BMI: body mass index, HDL: high density lipoprotein, BP: blood pressure, COPD: chronic obstructive pulmonary disease, MET: metabolic activity.

respect to all baseline characteristics. The distribution of hs-CRP in the two samples is shown in Fig. 1.

Dietary information was available on 4760 participants in 2015–2016 cycle and 4982 participants in 2017–2018 cycle. As shown in Supplemental Table 1, dietary characteristics were similar across the two cohorts.

3.2. Univariate analysis

In univariate analysis of the 2015–2016 cycle, numerous factors were significantly associated with elevated hs-CRP (Table 2). Notably, individuals with elevated hs-CRP were older, more likely to be female, and more likely to be Black or Hispanic. They also had had lower self-reported health, lower income, and more comorbidities than those with lower hs-CRP levels. Significant laboratory predictors of high hs-CRP included lower HDL cholesterol and higher white blood cell and platelet counts. The results were mostly consistent across the two cycles.

In subsamples of patients with dietary and nutritional information, predictors of elevated hs-CRP included having a lower HEI score and lower intake of total calories, fiber, protein, carbohydrate, total fat, monounsaturated fat, and polyunsaturated fat. The results were mostly consistent across the two cycles (Supplemental Table 2).

3.3. Multivariable logistic regression models

Univariate predictors significant at the ≤0.05 level were offered into the multivariate models. In the primary model (Table 3), independent predictors of elevated hs-CRP included: older age (OR 1.32 age: 40–64 vs <40; 95 % CI 1.04–1.69; P = 0.035, OR 1.45 age: 65+ vs <40; 95 % CI 1.02–2.05; P = 0.043), female sex (OR 1.57; 95 % CI 1.36–1.80; P = 0.003), Black vs White race (OR 1.31; 95 % CI 1.10–1.56; P = 0.037), increased BMI (OR 1.12 per kg/m^2; 95 % CI 1.10–1.14; P < 0.001), elevated white blood cell count (OR 1.21 per 1000 white blood cells/μL; 95 % CI 1.15–1.28; P = 0.002), and self-reported poor vs excellent health (OR 1.73; 95 % CI 1.04–2.22; P = 0.012). The model achieved a c-statistic of 0.77 (0.76–0.78) in the 2015–2016 derivation cycle and 0.76 (0.75–0.77) in the 2017–2018 validation cycle (Fig. 2).

In the nutritional model (Table 4), after adjusting for age, sex, race, and activity level based on metabolic activity score, the only significant predictor of elevated hs-CRP was lower fiber intake (OR 0.88 per 10 g; 95 % CI 0.80–0.97; P = 0.035). In the dietary model (Table 5), after adjusting for the same variables, the only significant predictor was lower fruit intake (OR 0.67; 95 % CI 0.40–0.934; P = 0.045). Fruit intake was treated dichotomously (any vs none) based on participants' 24-hour dietary recall. The dietary and nutrition models had poor discrimination with c-statistics of 0.60 in the 2015–2016 cohort and 0.61 in the 2017–2018 cohort for both models. In a sensitivity analysis, adding dietary factors to the main model did not improve discrimination.

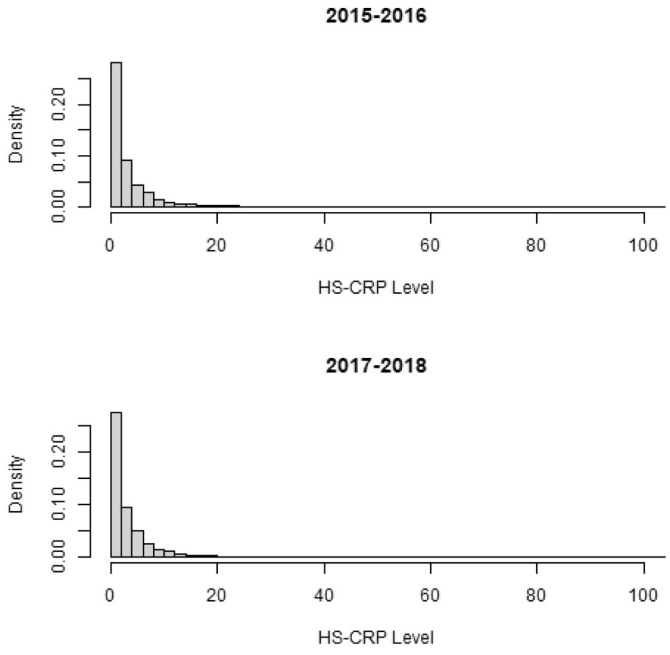

Fig. 1. hs-CRP distribution.

4. Discussion

The main findings of our analysis were that older age, female sex, Black race, increased BMI, higher white blood cell count, and self-reported poor health were independent predictors of elevated hs-CRP levels in the US adult population. The demographic, clinical, and laboratory multivariable regression model provided good discriminatory power with a c-statistic of 0.77. Model validation was confirmed with a c-statistic value of 0.76 in the 2017–2018 cycle. In addition, numerous dietary and nutritional predictors were associated with hs-CRP levels in univariate but not multivariable models. Protective dietary factors associated with lower hs-CRP levels included higher fruit and fiber intake. However, both dietary and nutritional models had poor discrimination and did not improve discrimination when integrated into the main model.

4.1. Interpretation

The objective of this study was to determine independent correlates of elevated hs-CRP levels with a particular interest in factors that might be modifiable, and thus subject to potential intervention. Of the factors we identified, only BMI is potentially modifiable, and even that is challenging, with studies showing only 20 % success rates at long term weight loss [18].

Our study is consistent with prior findings associating obesity with higher hs-CRP [19]. With the continuing rise in obesity in the US, the attributable risk of obesity to elevated hs-CRP levels and downstream illnesses mechanistically linked to chronic inflammation is likely enormous [20]. Conditions known to be linked to elevated hs-CRP include CVD, cancer, cognitive decline comprising both all-cause dementia and Alzheimer's disease, and frailty [8,21–24]. While primary prevention of obesity through healthy diet and exercise is critical to reversing the obesity epidemic, it is also important to note that several studies have shown that weight loss, whether medical or surgical, is associated with significant reductions in hs-CRP [25,26]. Further, bariatric surgery has been associated with reduced risk for cardiovascular events, and has potential for improving neurocognitive outcomes as well, although additional research is needed [27,28]. Studies have also shown that glucagon-like peptide-1 (GLP-1) agonists have a favorable effect on cardiovascular outcomes in patients with type 2 diabetes mellitus, which may be mediated in part through weight reduction and decreased inflammation [29]. With the recent FDA approval of the GLP-1 agonist semaglutide for weight loss in June 2021, additional studies are needed to assess the effects of this agent on chronic inflammation and its potential for reducing risk for CVD, cancer, cognitive decline, and frailty. Our findings, in the context of prior research, support increased efforts to prevent and treat obesity as a potent means to reduce the prevalence of major chronic illnesses mediated in part by chronic inflammation.

Our study also showed that several non-modifiable demographic factors, including older age, female sex, and Black race, were associated with increased hs-CRP. Previous studies have noted the association between female sex and higher hs-CRP levels, and other studies have suggested that this may be related to sex hormones and differences in amount of subcutaneous fat compared to males [30,31]. Conversely, a study in a large healthy Chinese population found that male sex was associated with higher CRP [32]. This discrepancy may be due to genetic or cultural differences in the study populations, or to unmeasured

Table 2
Univariate predictors of hs-CRP.

	2015–2016			2017–2018		
	hs-CRP < 3 mg/dL	hs-CRP ≥ 3 mg/dL	P-val	hs-CRP < 3 mg/dL	hs-CRP ≥ 3 mg/dL	P-val
n	3643	1769		3984	1872	
Male (%)	1900 (52.2)	713 (40.3)	<0.001	2080 (52.2)	760 (40.6)	0.0028
Age (mean (SD))	47.12 (19.03)	50.13 (17.27)	0.0301	49.44 (19.31)	50.84 (17.55)	0.0292
Race (%)			0.0033			0.4076
White	1172 (32.2)	559 (31.6)		1367 (34.3)	665 (35.5)	
Black	731 (20.1)	407 (23.0)		854 (21.4)	489 (26.1)	
Hispanic	1067 (29.3)	619 (35.0)		877 (22.0)	458 (24.5)	
Other	673 (18.5)	184 (10.4)		886 (22.2)	260 (13.9)	
Ratio family income to poverty (%)			0.0086			0.0256
0–1	806 (22.1)	436 (24.6)		724 (18.2)	419 (22.4)	
1–2	1003 (27.5)	506 (28.6)		1090 (27.4)	557 (29.8)	
2–3	601 (16.5)	322 (18.2)		655 (16.4)	334 (17.8)	
3 +	1233 (33.8)	505 (28.5)		1515 (38.0)	562 (30.0)	
BMI, kg/m^2	27.48 (5.68)	33.32 (7.88)	<0.001	27.86 (6.03)	33.64 (8.50)	<0.001
Waist circumference, cm	95.36 (14.83)	108.96 (17.20)	<0.001	96.23 (15.61)	109.35 (17.71)	<0.001
Systolic BP, mm Hg	123.89 (18.57)	126.95 (18.10)	0.0001	125.29 (20.11)	128.44 (20.48)	0.0002
Diastolic BP, mm Hg	69.50 (11.52)	69.72 (12.66)	0.0480	71.57 (12.47)	73.02 (12.83)	0.0060
HDL cholesterol, mg/dL	55.7 (16.9)	50.1 (15.2)	<0.001	54.6 (15.0)	50.3 (13.7)	<0.001
Total cholesterol, mg/dL	187.8 (39.5)	190.9 (39.5)	0.0713	185.0 (39.8)	189.8 (39.4)	0.0006
White blood count, 1000 cells/μL	6.70 [5.60, 8.00]	7.80 [6.50, 9.40]	<0.001	6.70 [5.50, 8.00]	7.80 [6.40, 9.40]	<0.001
Platelet count, 1000 cells/μL	232.90 (55.74)	252.82 (64.81)	<0.001	235.21 (57.72)	259.05 (68.46)	<0.001
General health			<0.001			<0.001
Fair/poor	747 (20.5)	598 (33.8)		831 (20.9)	616 (32.9)	
Good	1514 (41.6)	753 (42.6)		1666 (41.8)	806 (43.1)	
Excellent/very good	1382 (37.9)	418 (23.6)		1487 (37.3)	450 (24.0)	
Arthritis	767 (21.1)	575 (32.5)	<0.001	1049 (26.3)	655 (35.0)	0.0007
Congestive heart failure	101 (2.8)	89 (5.0)	0.0030	112 (2.8)	92 (4.9)	0.0004
Coronary heart disease	143 (3.9)	77 (4.4)	0.5172	176 (4.4)	90 (4.8)	0.0795
Angina	69 (1.9)	51 (2.9)	0.1831	104 (2.6)	59 (3.2)	0.0045
Heart attack	129 (3.5)	97 (5.5)	0.0422	176 (4.4)	97 (5.2)	0.0259
Liver condition	131 (3.6)	108 (6.1)	0.0013	184 (4.6)	123 (6.6)	0.1128
COPD	84 (2.3)	90 (5.1)	0.0005	162 (4.1)	135 (7.2)	0.0009
Cancer	322 (8.8)	172 (9.7)	0.9334	389 (9.8)	204 (10.9)	0.7234
MET Score	1300 [40, 4800]	960 [0, 3840]	0.0085	1440.00 [80.00, 5040.00]	960.00 [0.00, 4725.00]	0.0013
Statin	627 (17.2)	351 (19.8)	0.4517	831 (20.9)	369 (19.7)	0.4854
Metformin	288 (7.9)	08 (11.8)	0.0072	374 (9.4)	232 (12.4)	0.0283
Smoking, current	635 (17.4)	359 (20.3)	0.0479	636 (16.0)	385 (20.6)	0.0422

BMI: body mass index, HDL: high density lipoprotein BP: blood pressure, COPD: chronic obstructive pulmonary disease, MET: metabolic activity.

Table 3
Demographic, clinical, and laboratory multivariable regression model.

hs-CRP predictors	OR	LCL	UCL	P-value
Female vs male	1.57	1.26	1.95	0.007
Age: 40–64 vs <40	1.32	1.04	1.69	0.035
Age: 65+ vs <40	1.45	1.02	2.05	0.043
Black vs White	1.32	1.02	1.72	0.043
Hispanic vs White	1.07	0.75	1.51	0.591
Other vs White	1.00	0.65	1.55	0.974
BMI	1.12	1.09	1.14	0.001
WBC, per 1000 WBC/μL	1.24	1.15	1.33	0.002
Platelets, per 10,000 cells/μL	1.02	0.99	1.05	0.124
Liver disease	1.74	0.86	3.53	0.089
Good health vs poor	0.78	0.57	1.07	0.087
Excellent health vs poor	0.57	0.38	0.86	0.022

BMI: body mass index, WBC: white blood cells.

confounding factors, such as BMI or smoking, which were not assessed in the Chinese study. Additionally, associations between increased hs-CRP and Black race have been demonstrated in prior studies [33–35]. Higher hs-CRP in Black Americans may be a downstream biomarker of health disparities due to social and structural determinants of health. Furthermore, our findings of a racial disparity in hs-CRP levels may in part explain some of the increased cardiovascular burden and younger age of onset of CVD in the Black population [36]. Lastly, our study is in line with previous studies showing an association between older age and elevated hs-CRP [37,38]. This is an important finding because elevated hs-CRP in older adults is associated with CVD, frailty, and mortality

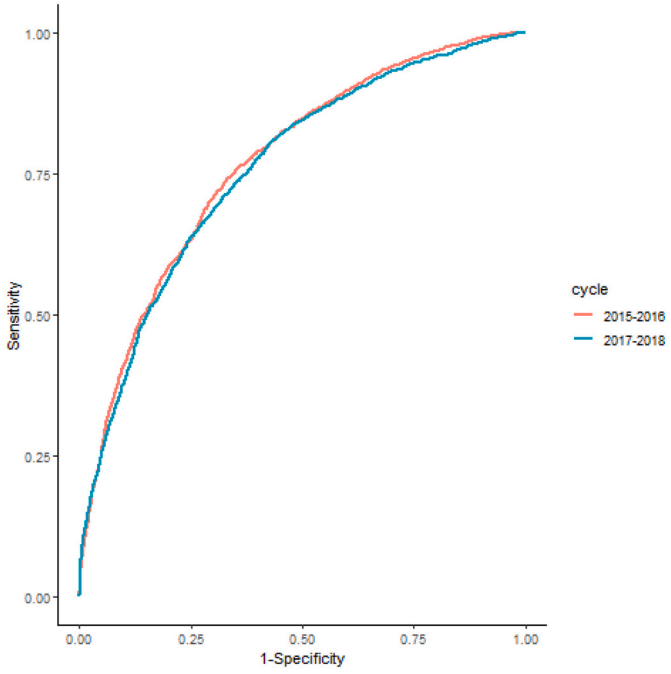

Fig. 2. Demographic, clinical, and laboratory multivariable regression model.

Table 4
Nutritional multivariable regression model.

hs-CRP predictors	OR	LCL	UCL	P-value
Female vs male	1.57	1.31	1.88	<0.001
Age: 40–64 vs <40	1.37	1.14	1.65	0.004
Age: 65+ vs <40	1.29	1.02	1.64	0.039
Black vs White	1.26	1.04	1.53	0.024
Hispanic vs White	1.44	1.17	1.78	0.004
Other vs White	0.87	0.67	1.14	0.280
Fiber, per 10 g	0.87	0.80	0.95	0.007

Table 5
Dietary multivariable regression model.

hs-CRP predictors	OR	LCL	UCL	P-value
Female vs male	1.67	1.40	1.99	<0.001
Age: 40–64 vs <40	1.39	1.14	1.70	0.006
Age: 65+ vs <40	1.36	1.06	1.76	0.023
Black vs White	1.31	1.08	1.59	0.014
Hispanic vs White	1.43	1.16	1.77	0.005
Other vs White	0.87	0.66	1.14	0.272
Fruit: <1 serving vs none	0.72	0.49	1.06	0.083
Fruit: >1 serving vs none	0.66	0.49	0.89	0.013

[39,40]. Further studies are warranted to determine if reducing hs-CRP in an older population through novel anti-inflammatory treatments could affect incidence and prevalence of these conditions.

Our findings are also notable for the lack of independent associations of behavioral and dietary factors with hs-CRP levels. We had anticipated that certain behavioral factors, including physical activity, tobacco use, and alcohol consumption, as well as a healthy dietary pattern with high intake of fruits and vegetables and avoidance of fast foods, would have impact on hs-CRP. However, although regular exercise, the Healthy Eating Index, and several dietary components were associated with hs-CRP by univariate analysis, none remained significant in the fully adjusted model. A possible explanation for this somewhat surprising observation is that any effect of these factors on hs-CRP may be too small to be detected in NHANES due to relatively modest sample size, cross-sectional design, and somewhat crude metrics for assessing physical activity and diet. It seems probable that dietary factors do have a role, as a recent systematic review on cardiovascular outcomes determined that fruit, vegetables, and fiber intake likely have a protective association [41].

4.2. Generalizability

The study has strong external validity to the broader US population due to the consistency in results across the two wholly independent study samples and excellent representation of African American and Hispanic Americans in NHANES. Asians are under-represented in NHANES and generalizability to non-US populations is uncertain.

4.3. Limitations

A limitation inherent to the cross-sectional observational nature of this study is that no causal inferences can be made. Nutritional and dietary data were obtained from a 24-hour recall, which may not accurately reflect the participant's regular diet. More rigorous and in depth collection of dietary data in future iterations of NHANES or other studies should be performed to better inform associations between dietary factors and inflammation. Other factors not included in the NHANES datasets could also affect hs-CRP levels.

5. Conclusion

Elevated hs-CRP in the US adult population is largely driven by demographic factors, but elevated BMI is a potentially modifiable factor, treatment of which could reduce risk for cardiovascular disease and other conditions associated with chronic inflammation.

CRediT authorship contribution statement

Zachary Randall, BS: Interpretation of data. Drafted the manuscript. Manuscript revisions.
Adam M. Brouillard MD, MBA: Conception and design of the study. Interpretation of data. Critically revised the manuscript.
Elena Deych, MS: Design of the study. Analysis of the data. Critically revised the manuscript.
Michael W. Rich, MD: Conception and design of the study. Interpretation of data. Critically revised the manuscript.

Declaration of competing interest

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

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ahjo.2022.100196.

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