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SHORT COMMUNICATION

# Serum hs-CRP varies with dietary cholesterol, but not dietary fatty acid intake in individuals free of any history of cardiovascular disease

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The objective of this study was to investigate whether serum high-sensitivity C-reactive protein (hs-CRP) concentration varies with dietary fatty acid intake in Iranian adults free of any history of cardiovascular disease (CVD). This cross-sectional study involved 8105 adults (3142 men) aged 35–65 years. Dietary intake was assessed using 24-h dietary recalls. The relationship between anthropometric, cardiometabolic risk factors and dietary data and serum hs-CRP was assessed using SPSS software. Median crude dietary saturated fat decreased across hs-CRP quarters ($P = 0.009$ for linear trend), whereas energy-adjusted total fat ($P = 0.017$), trans-fat ($P = 0.016$), monounsaturated fatty acids ($P = 0.030$) and cholesterol ($P = 0.005$) monotonically increased, with some evidence of statistical interactions by gender. In conclusion, serum hs-CRP concentrations were associated with some components of dietary fatty acid intake in our population of individuals without CVD, suggesting that dietary fat intake could be associated with subclinical inflammation.

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## INTRODUCTION

Recent evidence supports a key role for inflammation in all stages of the development of atherosclerosis.[1] Circulating markers of inflammation, such as C-reactive protein (CRP), tumor necrosis factor-α and some interleukins (IL-6 and IL- 1), are associated with high risk of cardiovascular events.[2] There is growing evidence that the influence of diet on cardiovascular disease (CVD) is mediated through mechanisms that include subclinical inflammation.[3] High-sensitivity CRP (hs-CRP) is a biomarker of low-grade inflammation, which has been shown to improve the prediction of the future risk of CVD and type 2 diabetes.[4] It has been reported that saturated fatty acids (SFAs) are more prone to be stored in the adipose tissue than monounsaturated fatty acids (MUFAs) and thereby increase the inflammatory milieu of this tissue.[5] Studies on the association of saturated fatty acids with hs-CRP[3,5] are conflicting. Some have reported positive associations between fatty acids and hs-CRP,[5] whereas others have indicated no significant association.[6,7] Clarifying this relationship has relevance for public health strategies to improve CVD risk stratification and reduction.

In the current study, we have examined the association between dietary intake of trans fatty acids, MUFAs, SFAs, poly unsaturated fatty acids (PUFAs), total fat and the serum concentrations of hs-CRP in a large Iranian adult population who were free of any overt CVD or other inflammatory diseases.

## MATERIALS AND METHODS

The Mashhad Stroke Heart Atherosclerosis Disorder (MASHAD) is an ongoing urban population-based, observational cohort study that was initiated by investigators of Mashhad University of Medical Sciences, using a stratified-cluster random sampling method.[8] The age range of participants was 35 to 64 years, and none had a past history of cardiovascular event (unstable angina, myocardial infarction and stroke), heart failure, peripheral vascular disease including transient ischemic attack or amaurosis fugax, or a history of any previous cardiovascular intervention or surgery. Individuals with any major comorbidity such as cancer, autoimmune, infectious and inflammatory diseases were excluded. The study protocol was approved by the Ethical Committee in Research of Mashhad University of Medical Sciences, and all study participants provided a written informed consent.

For all individuals, anthropometric parameters including weight, height and waist circumference were measured using standard protocols.[9] Fasting blood samples (after an overnight fast) were collected from each subject, and then centrifuged for 15 min to obtain serum. Sera were kept at − 80 °C until analyzed. Blood pressure was measured using a stethoscope and mercury sphygmomanometer calibrated by the Iranian Institute of Standards and Industrial Research; with Korotkoff phase 1 and phase 5 sounds marked the systolic and diastolic blood pressure,

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respectively. Fasting blood glucose and lipid profile were measured using an auto-analyzer (Eppendorf, Hamburg, Germany). Biochemical analysis comprising total cholesterol and triglycerides was carried out using enzymatic-based methods (Pars Azmon Inc., Tehran, Iran). Low-density lipoprotein cholesterol was calculated from the serum total cholesterol, triglycerides and high-density lipoprotein cholesterol concentrations expressed in mg/dl using the Friedewald formula. hs-CRP was measured by using an auto-analyzer (Eppendorf).[10]

### Dietary assessment

Dietary information was collected using a questionnaire for 24-h recall, administered by a trained dietary interviewer during a face-to-face interview, to collect information on food and beverage items consumed over the previous 24-h period.[11] Individual nutritional intakes were assessed using Dietplan6 software (Forestfield Software Ltd., Horsham, UK). An adjustment was made for total energy intake through the residual method as an alternative to using nutrient densities to control for confounding by total energy intake and to remove extraneous variation due to total energy intake.

### Statistical analysis

SPSS software (version 11.5, Chicago, IL, USA) was used for statistical analysis. The Kolmogorov–Smirnov tests were used to assess the normal distribution of continuous variables. Data are expressed as mean ± s.d. for normally distributed variables and median and 25th–75th percentiles for skewed variables. Changes in cardiometabolic profiles and dietary intake were investigated using the analysis of variance and Kruskal–Wallis tests. The association of dietary fat with hs-CRP was assessed by measuring the differences in dietary fat intake across subgroups of participants defined by increasing quarters of hs-CRP. A *P*-value $\leqslant 0.05$ was considered statistically significant.

## RESULTS

Of the 8105 participants, 39.8% ($n = 3142$) were men. The mean age was 48.3 years overall, 49.1 years in men and 47.9 years in women ($P = 0.001$). Compared with men, women were more likely to have a high body mass index ($P = 0.001$), higher waist girth ($P = 0.001$), higher high-density lipoprotein cholesterol, low-density lipoprotein cholesterol and total cholesterol (all $P = 0.001$), and a higher rate of metabolic syndrome ($P = 0.001$) based on the International Diabetes Federation (IDF) criteria but lower triglycerides, systolic blood pressure and lower smoking rate (all $P < 0.001$), Table 1). The distribution of the same characteristics across quarters of hs-CRP is shown in Table 2, with significant differences (all $P < 0.001$) in a linear manner (all $P < 0.001$ for linear trends), always reflecting monotonically increasing trend (decreasing for high-density lipoprotein cholesterol) across increasing quarters of hs-CRP, with, in most cases, evidence of statistically significant gender*hs-CRP interactions.

The association of fatty acid intake with hs-CRP is summarized in Table 3. Crude saturated fat and MUFA, energy-adjusted total fat, MUFA and cholesterol intake were significantly different across quarters of hs-CRP (all $P < 0.05$). Furthermore, median crude saturated fats monotonically decrease across hs-CRP quarters ($P = 0.009$ for linear trend), whereas energy-adjusted total fat ($P = 0.017$), trans-fat ($P = 0.016$), MUFA ($P = 0.030$) and cholesterol ($P = 0.005$) monotonically increased. Significant but weak correlations were apparent between continuous hs-CRP levels and all components of fatty acid intake. These correlations were negative for crude total fat, saturated fat and trans-fat but positive for the other components (Table 3).

## DISCUSSION

This study investigated the association between dietary fat intake and serum hs-CRP concentrations in Iranian adults. The main findings were a weak correlation between various components of fat intake with the hs-CRP level suggesting that the effect, if any, of fat intake on subclinical inflammation in people with no history of CVD is likely to be small.

Consistent with our findings, data from the National Health and Nutrition Examination Survey (NHANES 99-00) have reported SFA consumption to be correlated with increased hs-CRP in US adults.[3] Anti-inflammatory effects of Dietary Approaches to Stop Hypertension diet (DASH), which is low in cholesterol, saturated fat, total fat and increased consumption of fruits and vegetables, have been reported in the literature.[12] However, Muka *et al.*[13] reported that SFA intake was not significantly related with elevated hs-CRP ($\geqslant$1.0 mg/l) in Japanese women. The authors suggested that the absence of association could be due to the low baseline rate of elevated hs-CRP concentrations (5.6%). Moreover, a Swedish study of an elderly population failed to identify a significant relationship between levels of myristic, palmitic or stearic acids, measured in serum cholesteryl esters, and hs-CRP.[7] However, these findings were limited to a small sample of older individuals. In line with these latter findings, in an Italian population, an increased SFA intake was not significantly associated with changes in hs-CRP.

**Table 1.** General characteristics of participants overall and by gender

| *Variables* | *Overall* | *Men* | *Women* | P-*value*[a] |
|---|---|---|---|---|
| Age (years) | 48.35 ± 8.27 | 49.07 ± 8.46 | 47.88 ± 8.10 | < 0.001 |
| Body mass index (kg/m$^2$) | 27.89 ± 4.67 | 26.42 ± 4.12 | 28.87 ± 4.76 | < 0.001 |
| Waist circumference (cm) | 95.02 ± 12.66 | 93.48 ± 12.05 | 96.04 ± 12.94 | < 0.001 |
| Fasting blood glucose (mg/dl) | 91.67 ± 38.68 | 90.91 ± 37.27 | 92.16 ± 39.57 | 0.185 |
| HDL cholesterol (mg/dl) | 41.56 ± 10.54 | 38.23 ± 9.45 | 43.75 ± 10.64 | < 0.001 |
| LDL cholesterol (mg/dl) | 117.97 ± 35.65 | 114.83 ± 35.59 | 120.03 ± 35.55 | < 0.001 |
| Total cholesterol (mg/dl) | 190.83 ± 40.05 | 186.34 ± 40.20 | 193.79 ± 39.67 | < 0.001 |
| Triglycerides (mg/dl) | 143.55 ± 95.25 | 151.02 ± 105.70 | 138.61 ± 87.32 | < 0.001 |
| Systolic blood pressure (mmHg) | 120.77 ± 22.11 | 121.18 ± 21.80 | 120.49 ± 22.31 | < 0.001 |
| Diastolic blood pressure (mmHg) | 78.44 ± 13.92 | 79.24 ± 14.03 | 77.91 ± 13.82 | 0.123 |
| Current smoking (%) | 21.7 | 28.2 | 18.2 | < 0.001 |
| Diabetes mellitus (%) | 8.6 | 8.4 | 9.1 | 0.232 |
| Hypertension (%) | 23.5 | 25.1 | 23.5 | 0.312 |
| Metabolic syndrome (%) | 36.1 | 27.1 | 44 | < 0.001 |

Abbreviations: HDL, high-density lipoprotein; LDL, low-density lipoprotein. Values expressed as mean ± s.d. [a]*P*-values from analysis of the variance (ANOVA) for groups comparison.

**Table 2.** Clinical and biochemical features across quarters of hs-CRP

| *Variables* | *hs-CRP quarters* | | | | P-*value*[a] | P-*trend*[b] | P-*inter*[c] |
|---|---|---|---|---|---|---|---|
| | *1* | *2* | *3* | *4* | | | |
| N | *1970* | *2001* | *1940* | *1953* | | | |
| *Median CRP (25th–75th percentiles), mg/l* | *0.71 (0.57–0.81)* | *1.20 (1.00–1.38)* | *2.13 (1.86–2.51)* | *5.9 (4.1–7.4)* | | | |
| Sex (men) | 934 (47%) | 834 (42.7%) | 714 (36.8%) | 625 (32%) | $<0.001$ | $<0.001$ | — |
| Age (years) | $47\pm8$ | $47\pm8$ | $49\pm8$ | $49\pm8$ | $<0.001$ | $<0.001$ | $<0.001$ |
| Body mass index (kg/m$^2$) | $25\pm4$ | $27\pm4$ | $28\pm4$ | $29\pm5$ | $<0.001$ | $<0.001$ | $<0.001$ |
| Waist circumference (cm) | $90\pm11$ | $94\pm11$ | $96\pm11$ | $98\pm13$ | $<0.001$ | $<0.001$ | $<0.001$ |
| Fasting blood glucose (mg/dl) | $83\pm26$ | $88\pm34$ | $93\pm39$ | $100\pm48$ | $<0.001$ | $<0.001$ | $<0.01$ |
| HDL cholesterol (mg/dl) | $41\pm10$ | $42\pm11$ | $41\pm9$ | $41\pm9$ | $<0.001$ | $<0.001$ | 0.095 |
| LDL cholesterol (mg/dl) | $111\pm32$ | $115\pm33$ | $121\pm36$ | $123\pm37$ | $<0.001$ | $<0.001$ | 0.075 |
| Total cholesterol (mg/dl) | $181\pm35$ | $188\pm37$ | $195\pm42$ | $198\pm41$ | $<0.001$ | $<0.001$ | $<0.001$ |
| Triglycerides (mg/dl) | 107 (74–155) | 117 (83–171) | 132 (90–185) | 131 (95–186) | $<0.001$ | $<0.001$ | $<0.001$ |
| Systolic blood pressure | $117\pm19$ | $120\pm20$ | $122\pm21$ | $122\pm25$ | $<0.001$ | $<0.001$ | $<0.001$ |
| Diastolic blood pressure | $76\pm12$ | $78\pm13$ | $79\pm13$ | $79\pm15$ | $<0.001$ | $<0.001$ | $<0.001$ |
| Current smoking (%) | 20.1% | 22.2% | 22.1% | 24.2% | $<0.05$ | $<0.01$ | 0.003 |
| Diabetes mellitus (%) | 3.9% | 6.9% | 10.1% | 14.9% | $<0.001$ | $<0.001$ | 0.177 |
| Hypertension (%) | 17.3% | 23.6% | 26.9% | 28.7% | $<0.001$ | $<0.001$ | 0.054 |
| Metabolic syndrome (%) | 22.2% | 32.6% | 44.3% | 50.1% | $<0.001$ | $<0.001$ | $<0.001$ |

Abbreviations: HDL, high-density lipoprotein; hs-CRP, high-sensitivity C-reactive protein; LDL, low-density lipoprotein. Values expressed as mean ± s.d. for normally distributed data and median and 25th–75th percentiles for non-normally distributed data. [a]*P*-values from analysis of the variance (ANOVA) and Kruskal–Wallis tests *for groups comparison.* [b]*P*-values for linear trend across quarters of hs-CRP. [c]*P*-values for the interaction between sex and quarters of hs-CRP.

**Table 3.** Fatty acid intake in relation with hs-CRP levels

| *Variables* | *hs-CRP quarters* | | | | P-*value*[a] | P-*trend*[b] | P-*Inter*[c] | *Correlation coefficient (P-value)* |
|---|---|---|---|---|---|---|---|---|
| | *1* | *2* | *3* | *4* | | | | |
| N | *1970* | *2001* | *1940* | *1953* | | | | |
| *Median CRP (25th–75th percentiles), mg/l* | *0.71 (0.57–0.81)* | *1.20 (1.00–1.38)* | *2.13 (1.86–2.51)* | *5.9 (4.1–7.4)* | | | | |
| *Crude intake* | | | | | | | | |
| Total fat | 67 (47–92) | 65 (46–91) | 65 (46–90) | 66 (45–92) | 0.162 | 0.070 | 0.367 | −0.028 (0.011) |
| Saturated fat | 17 (12–24) | 17 (11–24) | 17 (12–24) | 16 (11–23) | $<0.05$ | 0.009 | 0.321 | −0.037 (0.001) |
| Trans fat (g) | 0.8 (0.4–1.3) | 0.7 (0.4–1.2) | 0.7 (0.4–1.2) | 0.7 (0.4–1.3) | 0.691 | 0.065 | 0.241 | −0.019 (0.231) |
| MUFA | 18 (12–25) | 17 (11–24) | 17 (12–24) | 17 (11–24) | $<0.05$ | 0.112 | 0.158 | 0.027 (0.023) |
| PUFA | 22 (15–33) | 22 (14–32) | 21 (15–32) | 22 (14–32) | 0.281 | 0.314 | 0.025 | 0.025 (0.015) |
| Cholesterol | 187 (103–316) | 176 (99–313) | 185 (100–323) | 186 (103–322) | 0.416 | 0.556 | 0.211 | 0.003 (0.351) |
| *Total energy-adjusted* | | | | | | | | |
| Total fat | 67 (55–80) | 67 (56–80) | 67 (56–80) | 69 (57–80) | $<0.05$ | 0.017 | 0.024 | 0.029 (0.019) |
| Saturated fat | 16 (12–20) | 16 (12–20) | 16 (12–20) | 16 (12–20) | 0.902 | 0.721 | 0.777 | 0.002 (0.632) |
| Trans fat (g) | 0.8 (0.5–1.2) | 0.9 (0.6–1.2) | 0.9 (0.6–1.2) | 0.9 (0.6–1.2) | 0.068 | 0.016 | 0.371 | 0.013 (0.412) |
| MUFA | 19 (16–23) | 19 (16–23) | 19 (16–23) | 20 (16–23) | $<0.052$ | 0.030 | 0.321 | 0.028 (0.018) |
| PUFA | 23 (17–30) | 24 (17–30) | 23 (17–30) | 24 (17–30) | 0.256 | 0.107 | 0.003 | 0.021 (0.325) |
| Cholesterol | 178 (115–297) | 178 (114–286) | 180 (121–303) | 189 (121–315) | $<0.05$ | 0.005 | 0.274 | 0.032 (0.001) |

Abbreviations: CRP, C-reactive protein; MUFA, monounsaturated fatty acid; PUFA, poly unsaturated fatty acid. Values expressed as mean ± s.d. for normally distributed data and median and 25th–75th percentiles for non-normally distributed data. [a]*P*-values from analysis of the variance (ANOVA) and Kruskal–Wallis tests *for groups comparison.* [b]*P*-values for linear trend across quarters of hs-CRP. [c]*P*-values for the interaction between sex and quarters of hs-CRP.

The authors hypothesized that, in dysmetabolic subjects, the role of dietary factors such as SFA on inflammation could be less evident than in healthy subjects.[14]

The suggested associations between SFA and serum hs-CRP, if any, could be supported by biological mechanisms. It seems that SFAs stimulate inflammatory signaling pathways by a process that involves Toll-like receptor-4[15] and subsequently nuclear factor kB, increasing the expression of a number of inflammatory genes.[16] A novel mechanism by which SFA might greatly amplify macrophage inflammation through a Toll-like receptor-4-independent pathway has been proposed, which is dependent on the uptake and metabolic processing of SFA into ceramide.[17]

It has been reported that the intake of peanuts that are rich in MUFA is related with improved postprandial profiles of inflammatory markers and lipids.[18,19] In addition, it has been suggested that the Mediterranean diet, high in MUFAs and PUFAs, has an inverse correlation with inflammatory markers including CRP.[12,20] However, inconsistent findings have been reported in studying this association, with some reporting no significant differences in subjects with a MUFA-rich diet.[21–23] These studies claimed that

central adiposity is associated with increased CRP levels, and it is possible that participants with central adiposity may not show improvements in inflammatory markers without weight loss. Furthermore, recently Muke and coworkers in a prospective study analyzing 4707 participants found that high intakes of PUFAs (mainly n-6 PUFAs) were correlated with lower levels of CRP, which might reflect reduced chronic systemic inflammation.[13] Julia *et al.*[24] stated that the inverse relation found between total n-3 PUFAs and CRP was mostly driven by long-chain n-3 PUFAs.[13]

### Limitations and strengths

The cross-sectional nature and the 24-h recall methodology are limitations of our study, although this approach has been widely used in previous surveys. Under-reporting of energy intake may be a problem when obese subjects are under investigation.[25] A major strength of the present study is the large number of participants. The differences in mean dietary intake between under reporters and those who give valid records reduced by energy adjustment through the residual model.

## CONCLUSION

The most obvious finding from this study is that hs-CRP concentrations are associated with some categories of dietary fatty acids in an Iranian population without the overt history of CVD, suggesting that hs-CRP concentrations could be modulated by dietary fatty acid intake. Furthermore, as the fatty acid intake has been a topic of interest in relation with CVD risk, understanding the effects of SFA, MUFA and PUFA on subclinical inflammation could bring new insights into this field.

## CONFLICT OF INTEREST

The authors declare no conflict of interest.

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