HHS Public Access

Author manuscript

Ethn Dis. Author manuscript; available in PMC 2016 April 19.

Published in final edited form as:

Ethn Dis. 2012 ; 22(4): 445–450.

Leukocyte Count and Cardiometabolic Risk Among Healthy Participants with Parental Type 2 Diabetes: The Pathobiology of Prediabetes in a Biracial Cohort Study

Alexander A. Boucher, BA, **Chimaroke Edeoga, MBBS, MPH**, **Sotonte Ebenibo, MBBS, MPH**, **Jim Wan, PhD**, and **Samuel Dagogo-Jack, MD**

Division of Endocrinology, Diabetes and Metabolism and Clinical Research Center, University of Tennessee Health Science Center, Memphis, Tennessee

Abstract

Objective—White blood cell (WBC) count has been associated with cardiometabolic risk, but the data for African Americans are conflicting. We determined whether WBC count predicts subclinical inflammation and cardiometabolic risk in African Americans, despite their known lower WBC count, compared to Caucasians.

Research design and methods—The study cohort consisted of 334 normoglycemic subjects (153 Caucasian, 181 African American) with parental type 2 diabetes (T2DM), mean (± SD) age 43.90 ± 10.25 y and BMI 30.1 ± 6.84 kg/m^2. Each subject underwent clinical examination and a standard oral glucose tolerance test (OGTT) to document glycemic status. Blood specimens were obtained for determination of WBC counts, lipid profile and C-reactive protein (CRP) levels. Metabolic syndrome components were identified, using the NCEP cut-offs for waist circumference, blood pressure, HDL cholesterol and triglyceride levels.

Results—Leukocyte counts were lower by ~400/cm^3 (P=.04) in African Americans than Caucasians, and were significantly correlated with waist circumference, HDL cholesterol, triglycerides and 2-h OGTT plasma glucose (P=.024–.0009), but not blood pressure in both races. Leukocyte counts significantly predicted the presence of three or more components of the metabolic syndrome similarly in African Americans (P=.0076) and Caucasians (P=.0078), as did CRP levels. Leukocyte counts correlated significantly with CRP levels in African Americans (r=.30, P<.0001) and Caucasians (r=.29, P=.0003).

Address correspondence to Samuel Dagogo-Jack, MD; University of Tennessee Health Science Center; 920 Madison Avenue, Suite 300A; Memphis, TN 38163; 901.448.5318; 901.448.5332 (fax); sdj@uthsc.edu.

Author Contributions

Design and concept of study: Dagogo-Jack
Acquisition of data: Edeoga, Ebenibo, Dagogo-Jack
Data analysis and interpretation: Boucher, Edeoga, Ebenibo, Wan, Dagogo-Jack
Manuscript draft: Boucher, Wan, Dagogo-Jack
Statistical expertise: Wan
Acquisition of funding: Dagogo-Jack
Administrative: Boucher, Edeoga, Ebenibo, Dagogo-Jack
Supervision: Dagogo-Jack

Conclusions—Our data indicate that WBC count, despite being lower in African Americans than Caucasians, predicts low-grade inflammation and cardiometabolic risk with similar magnitude in normoglycemic African Americans and Caucasians with parental T2DM.

Keywords

Ethnicity; Metabolic Syndrome; Inflammatory Markers; C-Reactive Protein; Offspring; Leukopenia; Waist Circumference

Introduction

Epidemiological and cross-sectional studies have indicated that subclinical inflammation may be associated with increased risks for type 2 diabetes (T2DM) and cardiometabolic disorders.[1,2] The pro-inflammatory state precedes the development of clinical events, and numerous acute-phase reactants (particularly C-reactive protein, CRP) have been utilized to identify at-risk individuals.[3] Several studies have also shown that variations in white blood cell (WBC) count within the normal range are associated with subclinical inflammation and cardiometabolic risk.[4–7] However, there are conflicting data regarding the association of WBC count with cardiometabolic risk in African Americans compared with Caucasians. The Atherosclerosis Risk in Communities (ARIC) study reported strong and similar associations between WBC count and CVD in African Americans and Caucasians,[5,6] whereas a report from the Bogalusa Heart Study[7] showed a much weaker relationship between WBC count and cardio-metabolic risk in African Americans than Caucasians. In the multiethnic Diabetes Prevention Program, CRP levels (marker of subclinical inflammation) were higher in African Americans than Caucasians, but WBC data were not reported.[8]

African Americans have a higher risk of T2DM, hypertension, and mortality from cardiovascular disease (CVD)[9,10] but have lower WBC count,[11–13] compared with Caucasians. The lower WBC count in African Americans is due to a genetic variant at the Duffy Antigen Receptor for Chemokines (DARC) locus on chromosome 1.[14,15] Besides the systematic 10%–20% Black-White difference in WBC count,[11–13] other factors that alter circulating WBC numbers and function include family history of T2DM[16] and hyperglycemia.[17,18] To avoid such confounding factors, we studied WBC in relation to subclinical inflammation and cardiometabolic risk factors in normoglycemic African Americans and Caucasians with similar parental history of T2DM.

Research Design and Methodology

Study Participants

The study participants are enrolled in our ongoing Pathobiology of Prediabetes in a Biracial Cohort (POP-ABC) study, the design of which has been reported elsewhere.[19] In brief, POP-ABC is a long-term follow-up study of incident dysglycemia among initially normoglycemic African Americans and Caucasians who are offspring of parents with T2DM. The study participants were Memphis-area residents, recruited through advertising and community screening events. Eligible participants were adults aged 18–65 years, who are the biological children of at least one parent with T2DM, and had normal fasting plasma glucose (FPG)

levels and/ or normal glucose tolerance at enrollment.[20] In addition, subjects were in good general health, and were not taking medications known to alter blood glucose, insulin sensitivity, insulin secretion or body weight. Race/ ethnicity was assessed by self-report of non-Hispanic White or non-Hispanic Black status. The study protocol was approved by the institution review board of the University of Tennessee Health Science Center. All participants gave written informed consent prior to the initiation of the study, which was conducted at the University of Tennessee Clinical Research Center, in accordance with the principles of the Declaration of Helsinki. Data collected at baseline from 334 (181 African American, 153 Caucasian) participants were analyzed for this report. Among Whites, 132 (86.3%) had a single parent with diabetes and 21 (13.7%) had both parents with diabetes. Among Blacks, 157 (86.7%) had a single parent with diabetes and 24 (13.3%) had both parents with diabetes.

Measurements

Each participant underwent standard anthropometric measurements (weight, height, waist circumference). Waist circumference was determined to the nearest .1 cm at the midpoint between the highest point of the iliac crest and the lowest costal margin in the mid-axillary line, using a Gulick II tape measure. Blood pressure was recorded in the seated position, using an automated sphygmomanometer; the average of two readings was used for calculations. Each participant underwent a standard 75-g oral glucose tolerance test (OGTT) after fasting overnight, to establish normoglycemic status, using the revised American Diabetes Association criteria.[20] Plasma glucose was measured by the glucose oxidase method (Beckman Instruments, Fullerton, CA). High sensitivity CRP levels in fasting plasma specimens were measured with a chemiluminescent assay, using commercial kits (Immulite, Siemens Ltd., Llanberis, Gwynedd, UK). The limit of detection of the CRP assay was .1 mg/L and the within-run and between-run coefficients of variation were <5%. Fasting plasma lipid profiles (including HDL cholesterol and triglycerides) and complete blood cell counts (CBC) were measured using standard techniques in a commercial clinical laboratory. Venous blood specimens for CBC count were collected into tubes containing ethylenediaminetetraacetic acid (EDTA); the total WBC count and differentials were performed using automated hematology cell counters.

Waist circumference, blood pressure, HDL cholesterol and triglyceride levels were the four components of the metabolic syndrome assessed in our study, using cut-offs established by the National Cholesterol Education Program Adult Treatment Panel III (NCEP).[21] Fasting plasma glucose was excluded in the definition because all subjects had normal FPG and/or NGT in order to participate in the POP-ABC study.[19]

Statistical Analysis

Data are reported as means ±standard deviation unless standard error is specified. Continuous variables were analyzed using ANOVA. Linear regression analyses, Spearman rank correlations and chi square test were used to assess and compare the relationships among WBC, CRP, and metabolic syndrome components in African Americans and Caucasians. Significance level was set at $P<.05$. The analyses were performed using

StatView for Windows® Version 5.0 (SAS Institute, Cary, NC, USA) and SAS® statistical software, version 9.2 (SAS Institute, Cary, NC, USA).

Results

The demographic and baseline clinical and laboratory characteristics of the study subjects are shown in Table 1. Caucasians had higher FPG (*P*=.0064) and triglyceride (*P*=.0001) levels compared to African Americans. The mean BMI (*P*=.0043) and systolic blood pressure (*P*=.0151) were higher in African Americans than Caucasians. Waist circumference tended to be larger in African Americans than Caucasians, significantly so in women (94.42 ± 15.00 vs. 90.10 ± 15.75, *P*=.031) but not men (99.73 ± 17.43 vs 98.30± 11.7, *P*=.65). Diastolic blood pressure and HDL cholesterol levels did not differ by race (Table 1). In general, women had a better cardiometabolic profile than men: higher HDL cholesterol levels (*P*<.0001), lower waist circumference (*P*<.0056), lower triglycerides (*P*=.0033), FPG (*P*=.0046), SBP (*P*=.0285) and DBP (*P*=.0038). The sex pattern was consistent across ethnicity in all measures except for FPG and DBP, where male-female differences did not reach significance among African Americans. By contrast, CRP levels were significantly higher (*P*=.0056) in women than men, and also higher (*P*=.0034) in African Americans than Caucasians (Table 1). There was a significant correlation between CRP levels and BMI (*r*=.33, *P*<.0001) and waist circumference (*r*=.43, *P*<.0001). The mean WBC count (× 1000/cm^3) was 5.79 ± 1.60 in Caucasians and 5.40 ± 1.70 in African Americans (*P*=.04). The metabolic syndrome components in each individual component were added up to derive a metabolic syndrome score. Fifty one subjects (15.3%) met NCEP criteria[21] for diagnosis of the metabolic syndrome by harboring ≥3 components. African American subjects were less likely to have the metabolic syndrome than Caucasians (11.6% vs. 19.6%; chi square = 11.5, *P*=.0093).

Fig. 1 shows the relationships among WBC count, metabolic syndrome score, and CRP in African Americans and Caucasians. A significant correlation was observed between metabolic syndrome score and WBC (*r*=.19, *P*=.0004). The correlation was consistent among Caucasians (r=.19, *P*=.017) and African Americans (*r*=.20, *P*=.0068), as well as in women (*r*=.18, *P*=.0042) and men (*r*=.22, *P*=.03). Similarly, CRP levels correlated significantly with metabolic syndrome score in the entire group (*r*=.41, *P*<.0001), in African Americans (*r*=.44, P<.0001), Caucasians (*r*=.36, *P*<.0001), women (*r*=.42, *P*<.0001) and men (*r*=.33, *P*<.0001) (Table 2). With regard to individual components of the metabolic syndrome, leukocyte counts were significantly correlated with waist circumference (*r*=.23, *P*=.0043), HDL cholesterol (*r*=.18, *P*=.0009), and triglycerides (*r*=.22, *P*=.0007) (Fig. 1), but not blood pressure or fasting plasma glucose levels in African Americans and Caucasians. As already noted, normal fasting glucose and normal glucose tolerance were key inclusion criteria for the POP-ABC Study.[18] Nonetheless, we found that WBC correlated with 2-h OGTT plasma glucose (2-hPG) levels (*r*=.19, *P*=.002). In a multivariate ANOVA model, waist circumference (*P*=0.0029), triglycerides (*P*=.024), HDL cholesterol (*P*=.0009) and 2-hrPG (*P*=.0015) remained significant predictors of WBC, whereas FPG, systolic and diastolic blood pressures did not predict WBC. Leukocyte counts significantly predicted the presence of three or more components of the metabolic syndrome similarly among African

Americans (P=.0076) and Caucasians (P=.0078), and also correlated significantly with CRP levels in African Americans (r=.30, P<.0001) and Caucasians (r=.29, P=.0003).

Discussion

African Americans have a higher risk of T2DM, hypertension, and CVD mortality but lower WBC count compared with Caucasians.[9–13] In our study, we compared the association of WBC and subclinical inflammation and cardiometabolic risk markers in African Americans and Caucasians. Our findings indicate that the lower WBC count in African Americans did not obscure the expected relationship between WBC and subclinical inflammation (as indicated by CRP levels). Also, WBC count predicted the accumulation of metabolic syndrome components with comparable rigor in African Americans and Caucasians.

We used four out of the five NCEP criteria for the metabolic syndrome, omitting fasting glucose, because having a normal FPG (<100 mg/dL) was a requirement for inclusion in the POP-ABC study. We did not observe any correlation between WBC count and FPG levels within the normal range. However, WBC count correlated significantly with 2-hPG, as it did with waist circumference, HDL cholesterol, and triglycerides. Thus, WBC count held up as a predictor of the metabolic syndrome even in the absence of dysglycemia. The pattern of interaction between WBC count and metabolic syndrome components was qualitatively similar to that between CRP levels and the same components. Our data confirm the previously reported ethnic and sex differences in CRP levels[22] and demonstrate a strong interaction between WBC count and CRP levels in African Americans (as in Caucasians), indicating that WBC count is a valid marker of subclinical inflammation in our study population.

Our findings are consistent with the data derived from the ARIC study,[5] but in discord with the report from the Bogalusa Heart Study.[7] We took particular care to restrict our study to normoglycemic subjects, in order to avoid the potential confounding effect of dysglycemia on leukocyte migration and function.[17,18] An additional strength of our study is the similarity in the presumed genetic risk for T2DM among the African Americans and Caucasians in our study cohort, all of whom were offspring of parents with T2DM. The latter feature served to dampen the effects of family history of T2DM on WBC count.[16] Nonetheless, our study has some limitations. First, the cross-sectional design does not enable inferences regarding the temporal relationship between changes in WBC and the degree of accumulation of cardiometabolic risk markers. Secondly, the correlation we observed between WBC and diverse cardiometabolic risk markers does not provide information regarding causality.

The lower WBC count in African Americans, which is well-known and sometimes referred to as ‘‘benign ethnic neutropenia,’’[13] has recently been shown to be due to a common African-derived inactivating polymorphism of the Duffy Antigen Receptor for DARC, which protects against malaria.[14,15] Decreased expression of DARC has been associated with exaggerated responses to endotoxin and altered systemic and local tissue expression of chemokines in animal models.[23,24] Leukocytes play critical and complex roles in innate and adaptive immunity, and have also been implicated in the pathogenesis of atherosclerosis and

other disorders. However, it is presently unclear whether the lower WBC count in African Americans compared to Caucasians is adaptive or maladaptive with regard to cardiometabolic risk. Prospective results from ARIC showed that African Americans in the highest quartile of WBC count (≥7,000 cells/mm^3) had ~2-fold increased risk of incident coronary heart disease, ischemic stroke, and CVD mortality compared to persons in the lowest quartile of WBC count (<4,800 cells/mm^3).[6]

In conclusion, our present findings provide reassurance that WBC count is a valid marker of subclinical inflammation and cardiometabolic risk in normoglycemic African Americans. Well-designed, hypothesis-driven studies are needed to demonstrate whether WBC count is sensitive to lifestyle and pharmacological interventions that reduce cardiometabolic risk burden, as has been shown for CRP.[8] As more evidence emerges from clinical studies, the incorporation of serial changes in WBC count could be an inexpensive adjunct to the overall cardiometabolic risk surveillance strategy. Our data provide reassurance that such an approach would be informative in African Americans.

Acknowledgments

The POP-ABC study is supported by grants from the National Institutes of Health (R01 DK067269 and MO1 RR00211) and the American Diabetes Association. This study was funded by the National Institutes of Health. The funding source had no role in the study design, execution, interpretation of results, or in the decision to submit the manuscript for publication. POP-ABC Research Group: Current: Samuel Dagogo-Jack, MD (Principal Investigator and Guarantor), Ann Ammons, MT, John Crisler, Chimaroke Edeoga, MBBS, MPH, Sotonte Ebenibo, MBBS, MPH, Ebenezer Nyenwe, MBBS, Jim Wan, PhD. Past members: Ruben Cuervo, MD (2006–2007), Nonso Egbuonu, MBBS (2008–2010), Nicoleta Ionica, MD (2007–2008), Dorota Malinowski, MD (2007–2008), Emmanuel Chapp-Jumbo, MBBS (2009–2011). Consultant: Steven Haffner, MD, University of Texas Health Science Center, San Antonio, TX; Data and Safety Officer: Murray Heimberg, MD, PhD, University of Tennessee Health Science Center, Memphis, TN.

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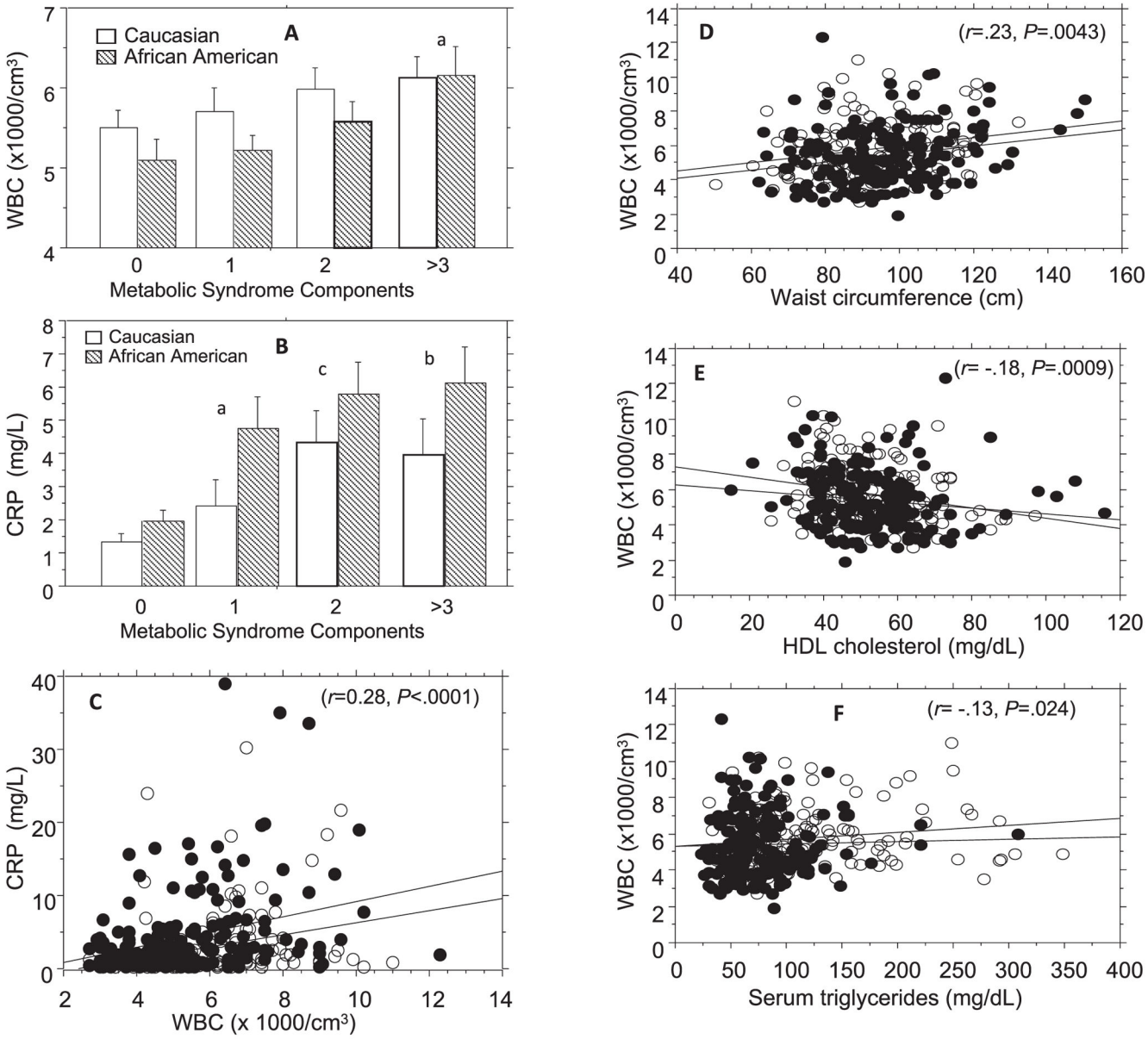

Fig 1.

Metabolic syndrome score in relation to mean white blood count (A) and C-reactive protein (B), and linear regression analyses of C-reactive protein vs. leukocyte count (C), and of leukocyte count vs. waist circumference (D), serum HDL cholesterol (E), and triglyceride (F) levels in African Americans (closed circles) and Caucasians (open circles). Metabolic syndrome score indicates the sum of components of the NCEP metabolic syndrome markers. WBC, white blood count; CRP, C-reactive protein; HDL, high density lipoprotein. To convert HDL cholesterol from mg/dL to mmol/L multiply by .02586. To convert triglycerides from mg/dL to mmol/L multiply by .0113

[a] P=0.004, [b] P=0.0003, [c] P<.0001 compared to participants with no metabolic syndrome component

The data in Fig. 1A and 1B are means ± SE

Table 1

Clinical and laboratory characteristics of study participants by sex and ethnicity

	Female		Male		
	Caucasian	**African American**	**Caucasian**	**African American**	***P***
n	*107*	*133*	*46*	*48*	
Age, years	47.7 ± 10.2	42.8 ± 10.3	45.9 ± 10.5	43.5 ± 9.14	.0002
BMI, kg/m^2	29.13 ± 7.24	31.26 ± 6.94	28.07 ± 6.17	30.6 ± 8.20	.0043
TG, mg/dL	109.1 ± 61.9	74.17 ± 30.92	127.30 ± 68	90.4 ± 51.5	<.0001
HDL, mg/dL	55.0 ± 13.3	55.32 ± 13.61	44.21 ± 7.88	47.5 ± 14.7	NS
Waist Circ, cm	90.1 ± 15.8	94.42 ± 15.00	98.30 ± 11.7	99.7 ± 17.4	.053
SBP, mm Hg	116 ± 13.7	120 ± 15.4	120 ± 11.8	124 ± 14.0	.015
DBP, mm Hg	71 ± 7.8	72.4 ± 8.41	74.7 ± 7.27	74.8 ± 9.76	NS
FPG, mg/dL	92.2 ± 6.56	90.6 ± 6.51	95.0 ± 5.19	92.2 ± 7.95	.0064
WBCx1000/cm^3	5.75 ± 1.53	5.40 ± 1.80	5.85 ± 1.76	5.41 ± 1.62	.044
CRP, mg/L	3.55 ± 5.28	5.09 ± 6.97	1.48 ± 3.53	3.44 ± 5.31	.0033

Data are means ± SD.

P are for ethnic comparisons.

BMI, body mass index; TG, Triglycerides; HDL, High-density lipoprotein cholesterol; Waist Circ, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; FPG, fasting plasma glucose; WBC; white blood cell count; CRP, C-reactive Protein; NS, not significant.

Table 2

Spearman correlation coefficients of metabolic syndrome score with white blood cell count and C-reactive protein by race and sex

	Overall		Caucasian		African American		Female		Male	
	r	***P***	***r***	***P***	***r***	***P***	***r***	***P***	***r***	***P***
WBC	.19	.0004	.19	.0168	.20	.0068	.18	.0042	.22	.0304
CRP	.41	<.0001	.36	<.0001	.44	<.0001	.42	<.0001	.33	.0011

Ethn Dis. Author manuscript; available in PMC 2016 April 19.