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Apolipoprotein-Containing Lipoprotein Subclasses and Subclinical Atherosclerosis in Systemic Lupus Erythematosus

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Abstract

Objective—Traditional classification of hyperlipidemia using high-density lipoprotein, low-density lipoprotein (LDL), and very low-density lipoprotein does not provide information on lipoprotein function. Apolipoproteins (Apos), which are protein components of plasma lipoproteins (including A, B, C, D, E) with their different composition, metabolic, and atherogenic properties, provide insight on lipoprotein functioning. In particular, the Apo B/A-I ratio is associated with atherogenic LDL and development of cardiovascular disease. We explored the baseline association between these nontraditional risk factors with subclinical measures of atherosclerosis (coronary artery calcification [CAC] and carotid intima-media thickness [IMT]) in systemic lupus erythematosus (SLE).

Methods—A total of 58 SLE patients (97% women, 58% white, 40% African American, and 2% other, mean $\pm$ SD age 44 ± 11 years) had measurement of Apo and lipoproteins by immunoturbidimetric procedures, electroimmunoassays, and immunoprecipitation. CAC was measured by helical computed tomography and carotid IMT by carotid duplex. This study was based on the baseline assessment of subclinical atherosclerosis in the Lupus Atherosclerosis Prevention Study. The measurement of the lipoproteins was made on sera collected at the same time.

Results—There was no association between cardioprotective Apos (Apo A-I, LpA-I, LpA-I:A-II) and CAC ($P < 0.15$, $P < 0.41$, and $P < 0.39$, respectively) or carotid IMT ($P < 0.97$, $P < 0.53$, and $P < 0.76$, respectively). CAC and carotid IMT did not associate with atherogenic Apos either,

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AUTHOR CONTRIBUTIONS

All authors were involved in drafting the article or revising it critically for important intellectual content, and all authors approved the final version to be submitted for publication. Dr. Kiani had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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including LpB:E+LpB:C:E, Apo B, LpB, LpB:C, Apo C-III, Apo C-III-HS, Apo C-III-HP, Apo C-III-R, LpA-II:B:C:D:E, and Apo B/Apo A-I. Measures of disease activity, including physician's global assessment and Systemic Lupus Erythematosus Disease Activity Index, were not associated with CAC or carotid IMT.

Conclusion—Neither cardioprotective nor atherogenic lipoproteins were associated with measures of subclinical atherosclerosis in this series of SLE patients. Further studies with a larger sample size are warranted to confirm our findings.

Introduction

Coronary artery disease remains a major cause of mortality in systemic lupus erythematosus (SLE). Traditional cardiovascular risk factors, including hypertension, hyperlipidemia, obesity, and homocysteine (1), are associated with coronary artery disease in SLE but do not account for the entire increased risk of atherosclerosis (2). Traditional classification of hyperlipidemia using high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very low-density lipoprotein does not provide information on lipoprotein function. Apolipoproteins (Apos), which are protein components of plasma lipoproteins (including A, B, C, D, E), have different composition, metabolic, and atherogenic properties that might yield more insight on lipoprotein functioning in SLE.

Dyslipidemias are due to an imbalance between atheroprotective Apo A and atherogenic Apo B-containing lipoprotein subclasses, leading to lipid transport derangement and eventually development of atherosclerotic disease. Even within atherogenic Apo B-containing lipoprotein subclasses, LpB:E+LpB:C:E, LpA-II:B:C:D:E, LpB, and LpB:C have different affinities for lipoprotein lipase as well as for LDL receptors (3). Size, density of lipoprotein particles, and cholesterol richness all contribute to atherogenic potential of a subclass (4).

In particular the Apo B/A-I ratio is associated with atherogenic LDL and development of cardiovascular disease. In a study of 247 patients with type 2 diabetes mellitus, Apo B/A-I ratio was significantly associated with carotid intima-media thickness (IMT) (5). Another Apo, Apo C-III, induces expression of vascular cell adhesion molecule 1 in vascular endothelial cells and increases adhesion of monocytic cells, leading to atherogenesis in humans (6). Apo C-III, another atherogenic lipoprotein subclass constituent, is seen in triglyceride-rich Apo B-containing lipoproteins. In the general population Apo C-III has been linked to atherosclerotic progression as well as cardiovascular events (7). Apo C-III was a risk factor for coronary artery progression in rheumatoid arthritis patients (8), but its association with progression of subclinical measures of atherosclerosis in SLE has not been examined.

LpA-I or LpA-I:A-II are 2 major high-density cardioprotective lipoproteins containing Apo A-I. LpA-I accepts peripheral cholesterol (9), whereas LpA-I:A-II binds Apo C peptides and Apo E released during the lipolytic degradation of triglyceride-rich lipoproteins (10). Patients in the general population with coronary artery disease have been shown to have lower levels of LpA-I or LpA-I:A-II (11). We studied these new categorizations of Apos in a

series of SLE patients who had subclinical measures of atherosclerosis, including coronary artery calcification (CAC) and carotid IMT.

Patients and Methods

Study participants

Study participants were members of the Hopkins Lupus Cohort who had undergone measurements of carotid IMT and CAC by helical computed tomography (CT) as part of a trial of atorvastatin versus placebo (12). Patients with a history of an atherosclerotic event (such as angina or myocardial infarction) were excluded. All patients gave informed consent, and the study was approved by the Johns Hopkins University School of Medicine Institutional Review Board. The trial included baseline and 2-year followup measures of subclinical atherosclerosis. This report is based on the measures and sera collected at the baseline visit prior to the start of the atorvastatin or placebo medications. This is a crosssectional association study of baseline lipid parameters of subclinical atherosclerosis.

Fifty-eight SLE patients (97% women, 58% white, 40% African American, and 2% other, mean $\pm$ SD age 44 ± 11 years) had measurement of Apo and lipoproteins by immunoturbidimetric procedures, electroimmunoassays, and immunoprecipitation. These patients were selected because they were the only patients who had sufficient baseline serum left to measure Apos. Cutoffs were used as previously described (13). Cumulative American College of Rheumatology classification criteria included 62% malar rash, 26% discoid rash, 57% photosensitivity, 59% oral ulcers, 76% arthritis, 50% serositis, 41% proteinuria, 3% neurologic disorders, 76% hematologic disorders, 90% immunologic disorders, and 98% positive antinuclear antibody (14). Additional Systemic Lupus International Collaborating Clinics criteria included 55% low complement and 18% direct Coombs's test (15).

Measurement of CAC

Scores for CAC were calculated using Agatston scoring. CAC was assessed by multidetector CT with a Siemens Volume Zoom Scanner (Siemens Medical Solutions), using a 2.5 mm collimation and a slice width of 3 mm, with both scans done on the same machine. CAC was quantified using a standard scoring system, available as part of the scanner software package (16).

Measurement of carotid IMT

Carotid IMT was measured using high resolution B-mode ultrasound at baseline to image the right and left common carotid arteries using a single ultrasound machine, the Sonos 5500 (Philips Medical Systems), with a linear array 8-MHz scan head with standardized image settings, including resolution mode, depth of field, gain, and transmit focus. Carotid IMT was measured between the lumen intima and media-adventitia interfaces of the far wall of the common carotid artery (the 1-cm segment proximal to the bifurcation) by a single reader using an automated edge detection system. The mean IMT of this 1-cm segment was measured on 2 separate images of the left and the right common carotid artery at the peak of the R wave on a simultaneous electrocardiogram tracing. The mean of these 4 measurements

was used as the IMT. We chose this location because of its demonstrated reproducibility compared with measurements of carotid IMT at other sites (17).

This study was based on the baseline assessment of subclinical atherosclerosis in the Lupus Atherosclerosis Prevention Study. The measurement of the lipoproteins was made on sera collected at the same time. None of the patients were using statins.

Statistical analysis

Statistical analyses were carried out using SAS software, version 9.2. We transformed CAC by taking the $\log_e(\text{CAC} + 1)$ prior to calculating means and performing inference. This transformation was done because distribution of CAC was skewed. Patients were divided into subgroups, based on different cutoffs for each lipid variable and then were compared with respect to carotid IMT and CAC. Multivariable regression models were used to calculate *P* values, controlling age, gender, and ethnicity. *P* values less than or equal to 0.05 were considered statistically significant.

Results

Table 1 shows the association between traditional cardiovascular risk factors, cardioprotective and atherogenic lipoprotein subclasses, and CAC and carotid IMT. Of the traditional cardiovascular risk factors, including total cholesterol, triglycerides, low-density lipoprotein, very low-density lipoprotein, and high-density lipoprotein cholesterol, none were associated with either CAC or carotid IMT. None of the cardioprotective lipoprotein subclasses Apo A-I, LpA-I, and LpA-LA-II were associated with any subclinical measure of atherosclerosis. There was also no association between any atherogenic lipoprotein subclasses, including Apo B, LpB:E+LpB:C:E, LpA-II:B:C:D:E, LpB, LpB:C, Apo C-III, Apo C-III-HP, Apo C-III-HS, Apo C-III-R, LpA-II:B:C:D:E, Apo B/Apo A-I, and CAC or carotid IMT.

Measures of disease activity, including the physician's global assessment and Systemic Lupus Erythematosus Disease Activity Index, were not associated with CAC or carotid IMT (Table 2). Given the relatively small sample, confidence intervals for mean differences were somewhat wide. For example, the point estimate for the difference in mean IMT comparing those with and without high levels of Apo A-I was 0, but the confidence interval ranges from -0.5 to positive 0.5 mm. We then looked at the association between carotid plaque and cardioprotective and atherogenic lipoprotein subclasses. None of the lipids or lipid ratios studied were associated with statistically significant differences in carotid artery plaque (data not shown).

Discussion

In this study we found no association between traditional cardiovascular risk factors, cardioprotective lipoproteins, or atherogenic lipoproteins with subclinical measures of atherosclerosis. Although we observed no evidence of associations, given the small sample size, we cannot completely rule the possibility out. For example, although the mean IMT was the same among those with and without high levels of Apo A-I, based on the confidence

interval, the data are consistent with the possibility that the mean IMT is 0.5 mm greater or lesser among those with high Apo A-I. In addition, patients with known atherosclerotic disease or with high lipid levels for which statins are considered standard of care were excluded from the study due to ethical concerns. We did not look at these associations at the end of 2-year samples due to nonavailability of sera from that end point. We do, however, acknowledge that those data might have been more informative.

We have previously shown, in a much larger sample size, that there was no association between CAC and traditional cardiovascular risk factors, including low-density lipoprotein, high-density lipoprotein, triglycerides, and total cholesterol (18). However, total cholesterol did predict progression of CAC over a 2-year period (19).

Cholesterol or triglycerides are not specific markers for discrete lipoprotein families. Considerable evidence has shown that Apo A and Apo B-containing lipoprotein families differ in their antiatherogenic and atherogenic potential, respectively (20). Therefore, only a direct quantitative measurement of these lipoproteins can truly reflect the difference between normal and dyslipoproteinemic states.

In this study, however, there was no association between levels of cardioprotective Apo A-I, nor levels of LpA-I or LpA-I:A-II with CAC and carotid IMT. In a previous study of 152 rheumatoid arthritis patients who underwent helical CT at baseline and 3 years, there was no difference in baseline levels of these Apos and 2 groups of progressors of CAC as well as nonprogressors (8). Apo A-containing lipoproteins are considered atheroprotective. However, significantly high levels of anti-Apo A-I antibodies have been shown in patients with SLE (21). We did not measure these antibodies in our study.

The Apo B/Apo A-I ratio may add to the estimate of coronary artery disease risk (22). In a study of 391 men ages >58 years who underwent carotid ultrasound to measure carotid IMT at baseline, the Apo B/Apo A-I ratio was an important risk factor for carotid IMT progression (23). In our study, however, there was no association between the Apo B/Apo A-I ratio with carotid IMT or CAC.

Our group has, for the first time, looked at these novel lipid measures and their association with subclinical measures of atherosclerosis in SLE. The results are disappointing. The work of McMahon et al, on proinflammatory HDL in SLE, is the only proven association of a novel lipid measure and the risk of cardiovascular disease in SLE (24).

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References

1. Petri M, Roubenoff R, Dallal GE, Nadeau MR, Selhub J, Rosenberg IH. Plasma homocysteine as a risk factor for atherothrombotic events in systemic lupus erythematosus. *Lancet*. 1996; 348:1120–4. [PubMed: 8888164]

2. Esdaile JM, Abrahamowicz M, Grodzicky T, Li Y, Panaritis C, du Berger R, et al. Traditional Framingham risk factors fail to fully account for accelerated atherosclerosis in systemic lupus erythematosus. *Arthritis Rheum.* 2001; 44:2331–7. [PubMed: 11665973]
3. Alaupovic P, Knight-Gibson C, Wang CS, Downs D, Koren E, Brewer HB Jr, et al. Isolation and characterization of an apoA-II-containing lipoprotein (LP-A-II:B complex) from plasma very low density lipoproteins of patients with Tangier disease and type V hyperlipoproteinemia. *J Lipid Res.* 1991; 32:9–19. [PubMed: 1901345]
4. Nordestgaard BG, Tybaerg-Hansen A. IDL, VLDL, chylomicrons and atherosclerosis. *Eur J Epidemiol.* 1992; 8:92–8. [PubMed: 1505659]
5. Dahlen EM, Lanne T, Engvall J, Lindstrom T, Grodzinsky E, Nystrom FH, et al. Carotid intima-media thickness and apolipoprotein B/apolipoprotein A-I ratio in middle-aged patients with type 2 diabetes. *Diabet Med.* 2009; 26:384–90. [PubMed: 19388968]
6. Kawakami A, Aikawa M, Alcaide P, Luscinskas FE, Libby P, Sacks FM. Apolipoprotein CIII induces expression of vascular cell adhesion molecule-1 in vascular endothelial cells and increases adhesion of monocytic cells. *Circulation.* 2006; 114:681–7. [PubMed: 16894036]
7. Alaupovic P, Mack WJ, Knight-Gibson C, Hodis HN. The role of triglyceride-rich lipoprotein families in the progression of atherosclerotic lesions as determined by sequential coronary angiography from a controlled clinical trial. *Arterioscler Thromb Vasc Biol.* 1997; 17:715–22. [PubMed: 9108785]
8. Knowlton N, Wages JA, Centola MB, Giles J, Bathon J, Quiroga C, et al. Apolipoprotein B-containing lipoprotein subclasses as risk factors for cardiovascular disease in patients with rheumatoid arthritis. *Arthritis Care Res (Hoboken).* 2012; 64:993–1000. [PubMed: 22337612]
9. Barkia A, Puchois P, Ghalim N, Torpier G, Barbaras R, Ailhaud G, et al. Differential role of apolipoprotein A1-containing particles in cholesterol efflux from adipose cells. *Atherosclerosis.* 1991; 87:135–46. [PubMed: 1906713]
10. Alaupovic P. David Rubenstein Memorial Lecture: the biochemical and clinical significance of the interrelationship between very low density and high density lipoproteins. *Can J Biochem.* 1981; 59:565–79. [PubMed: 7028220]
11. Puchois P, Kandoussi A, Fievet P, Fourrier JL, Bertrand M, Koren E, et al. Apolipoprotein A-1 containing lipoproteins in coronary artery disease. *Atherosclerosis.* 1987; 68:35–40. [PubMed: 3120739]
12. Petri M, Kiani AN, Post W, Christopher-Stine L, Magder LS. Lupus Atherosclerosis Prevention Study (LAPS). *Ann Rheum Dis.* 2011; 70:760–5. [PubMed: 21177297]
13. Alaupovic P. Use of apolipoprotein parameters and endpoints in drug development and approval processes. *Am J Cardiol.* 1998; 81:40–7.
14. Hochberg MC, for the Diagnostic and Therapeutic Criteria Committee of the American College of Rheumatology. Updating the American College of Rheumatology revised criteria for the classification of systemic lupus erythematosus [letter]. *Arthritis Rheum.* 1997; 40:1725.
15. Petri M, Orbai AM, Alarcon GS, Gordon C, Merrill JT, Fortin PR, et al. Derivation and validation of the Systemic Lupus International Collaborating Clinics classification criteria for systemic lupus erythematosus. *Arthritis Rheum.* 2012; 64:2677–86. [PubMed: 22553077]
16. Budoff MJ, Georgiou D, Brody A, Agatston AS, Kennedy J, Wolfkiel C. Ultrafast computed tomography as a diagnostic modality in the detection of coronary artery disease: a multicenter disease, a multicenter study. *Circulation.* 1996; 93:898–904. [PubMed: 8598080]
17. Smilde TJ, Wollersheim H, Van Langen H. Reproducibility of ultrasonographic measurements of different carotid and femoral artery segments in healthy subjects and in patients with increased intima-media thickness. *Clin Sci (Lond).* 1997; 93:317–24. [PubMed: 9404223]
18. Kiani AN, Magder L, Petri M. Coronary calcium in SLE is associated with traditional cardiovascular risk factors, but not with disease activity. *J Rheumatol.* 2008; 35:1300–6. [PubMed: 18484694]
19. Kiani AN, Post W, Magder LS, Petri M. Predictors of progression in subclinical atherosclerosis over two years in systemic lupus erythematosus (SLE). *Rheumatology (Oxford).* 2011; 50:2071–9. [PubMed: 21875880]

20. Alaupovic P. The concept of apolipoprotein-defined lipoprotein families and its clinical significance. *Curr Atheroscler Rep.* 2003; 5:459–67. [PubMed: 14525679]
21. O'Neill SG, Giles I, Lambrianides A, Manson J, D'Cruz D, Schrieber L, et al. Antibodies to apolipoprotein A-I, high-density lipoprotein, and C-reactive protein are associated with disease activity in patients with systemic lupus erythematosus. *Arthritis Rheum.* 2010; 62:845–54. [PubMed: 20131231]
22. Walldius G, Jungner I. The apoB/apoA-I ratio: a strong, new risk factor for cardiovascular disease and a target for lipidlowering therapy. A review of evidence. *J Intern Med.* 2006; 259:493–519. [PubMed: 16629855]
23. Schmidt C, Wikstrand J. High apoB/apoA-I ratio is associated with increased progression rate of carotid artery intima-media thickness in clinically healthy 58-year old men: experiences from very long-term follow-up in the AIR study. *Atherosclerosis.* 2009; 205:284–9. [PubMed: 19124125]
24. McMahon M, Grossman J, Skaggs B, FitzGerald J, Sahakian L, Ragavendra N, et al. Dysfunctional proinflammatory high-density lipoproteins confer increased risk of atherosclerosis in women with systemic lupus erythematosus. *Arthritis Rheum.* 2009; 60:2428–37. [PubMed: 19644959]

Significance & Innovations

- Coronary artery disease remains a major cause of mortality in systemic lupus erythematosus (SLE). Traditional classification of hyperlipidemia using high-density lipoprotein, low-density lipoprotein and very low-density lipoprotein does not provide information on lipoprotein function. Apolipoproteins, protein components of plasma lipoproteins (including A, B, C, D, E) have different composition, metabolic, and atherogenic properties that might yield more insight on lipoprotein functioning in SLE.
- In our small study, we have shown neither cardioprotective nor atherogenic lipoproteins were associated with measures of subclinical atherosclerosis in SLE.

Carotid IMT, CAC, traditional cardiovascular risk factors, and cardioprotective and atherogenic lipoprotein subclasses*

Table 1

	N	Carotid IMT, mean $\pm$ SD	P†	Log _e (CAC score + 1), mean $\pm$ SD	P†
Traditional cardiovascular risk factors					
Total cholesterol (mg/dl)					
200	16	0.58 $\pm$ 0.05	0.68	2.20 $\pm$ 2.46	0.092
<200	42	0.57 $\pm$ 0.12		0.97 $\pm$ 1.64	
Triglycerides (mg/dl)					
150	10	0.60 $\pm$ 0.09	0.71	2.15 $\pm$ 3.23	0.20
<150	48	0.56 $\pm$ 0.11		1.14 $\pm$ 1.57	
VLDL cholesterol (mg/dl)					
25	17	0.59 $\pm$ 0.08	0.93	1.52 $\pm$ 2.65	0.89
<25	41	0.56 $\pm$ 0.12		1.23 $\pm$ 1.61	
LDL cholesterol (mg/dl)					
110	14	0.58 $\pm$ 0.05	0.40	2.41 $\pm$ 2.56	0.083
<110	40	0.56 $\pm$ 0.13		1.02 $\pm$ 1.66	
HDL cholesterol (mg/dl)					
45	10	0.61 $\pm$ 0.18	0.58	1.17 $\pm$ 1.30	0.39
>45	44	0.56 $\pm$ 0.09		1.43 $\pm$ 2.14	
Cardioprotective					
Apo A-I					
130	21	0.57 $\pm$ 0.13	0.97	0.80 $\pm$ 1.26	0.15
>130	37	0.57 $\pm$ 0.10		1.60 $\pm$ 2.22	
LpA-I					
33	38	0.57 $\pm$ 0.11	0.53	1.18 $\pm$ 1.95	0.41
>33	20	0.58 $\pm$ 0.11		1.57 $\pm$ 1.99	
LpA-I:A-II					
95	15	0.59 $\pm$ 0.15	0.76	1.08 $\pm$ 1.40	0.39
>95	43	0.56 $\pm$ 0.09		1.39 $\pm$ 2.12	
Atherogenic					
Apo B					

	N	Carotid IMT, mean $\pm$ SD	$P^{\dagger}$	Log _e (CAC score + 1), mean $\pm$ SD	$P^{\dagger}$
95	12	0.58 $\pm$ 0.05	0.53	1.28 $\pm$ 2.49	0.60
<95	46	0.57 $\pm$ 0.12		1.32 $\pm$ 1.82	
LpA-II:B:C:D:E					
13	22	0.60 $\pm$ 0.12	0.88	1.44 $\pm$ 2.25	0.63
<13	36	0.55 $\pm$ 0.10		1.23 $\pm$ 1.78	
LpB:E+LpB:C:E					
14	19	0.59 $\pm$ 0.14	0.31	1.81 $\pm$ 2.25	0.24
<14	39	0.56 $\pm$ 0.09		1.07 $\pm$ 1.78	
LpB					
62	3	0.56 $\pm$ 0.04	0.62	0.80 $\pm$ 1.10	0.47
<62	55	0.57 $\pm$ 0.11		1.34 $\pm$ 1.99	
LpB:C					
11.1	3	0.60 $\pm$ 0.02	0.47	0.00 $\pm$ 0.00	0.24
<11.1	55	0.57 $\pm$ 0.11		1.38 $\pm$ 1.98	
Apo C-III					
12	7	0.58 $\pm$ 0.04	0.47	2.08 $\pm$ 2.75	0.60
<12	46	0.57 $\pm$ 0.12		1.30 $\pm$ 1.90	
Apo C-III-HS					
6.0	23	0.55 $\pm$ 0.13	0.44	0.96 $\pm$ 1.55	0.17
>6.0	30	0.58 $\pm$ 0.10		1.75 $\pm$ 2.28	
Apo C-III-HP					
>3.0	11	0.56 $\pm$ 0.06	0.53	1.8 $\pm$ 2.70	0.43
<3.0	42	0.57 $\pm$ 0.12		1.30 $\pm$ 1.82	
Apo C-III-HP					
4.0	3	0.56 $\pm$ 0.03	0.85	1.1 $\pm$ 1.9	0.76
<4.0	50	0.57 $\pm$ 0.12		1.4 $\pm$ 2.0	
Apo C-III-R					
<1.5	0	–	–	–	–
>1.5	53	0.57 $\pm$ 0.11		1.41 $\pm$ 2.02	
Apo B/Apo A-I					
0.8 (0.7 for women)	14	0.58 $\pm$ 0.05	0.51	1.14 $\pm$ 2.12	0.97

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	N	Carotid IMT, mean $\pm$ SD	$P^{\dagger}$	$\text{Log}_e(\text{CAC score} + 1)$, mean $\pm$ SD	$P^{\dagger}$
<0.8 (0.7 for women)	44	0.57 $\pm$ 0.12		1.36 $\pm$ 1.92	

* IMT = intima-media thickness; CAC = carotid artery calcification; VLDL = very low-density lipoprotein; LDL = low-density lipoprotein; HDL = high-density lipoprotein Apo = apolipoprotein.

[†] Controlled for age, sex, and ethnicity.

Table 2

Carotid IMT, CAC, and disease activity indices *

Measures	N	Carotid IMT, mean $\pm$ SD	$P^{\dagger}$	Log _e (CAC score + 1), mean $\pm$ SD	$P^{\dagger}$
Physician's global assessment					
1	7	0.52 $\pm$ 0.07	0.73	1.76 $\pm$ 2.04	0.093
<1	51	0.58 $\pm$ 0.11		1.25 $\pm$ 1.96	
>0	46	0.57 $\pm$ 0.12	0.38	1.52 $\pm$ 2.10	0.23
0	12	0.57 $\pm$ 0.08		0.52 $\pm$ 0.96	
SLEDAI					
2	15	0.55 $\pm$ 0.08	0.69	1.38 $\pm$ 2.24	0.58
<2	43	0.58 $\pm$ 0.12		1.29 $\pm$ 1.87	
4	5	0.54 $\pm$ 0.10	0.35	1.41 $\pm$ 1.93	0.93
<4	53	0.57 $\pm$ 0.11		1.30 $\pm$ 1.97	

* IMT = intima-media thickness; CAC = carotid artery calcification; SLEDAI = Systemic Lupus Erythematosus Disease Activity Index

 † Controlled for age, sex, and ethnicity.