Abstract 901 Table 2 Median CVH scores by subgroup

		CVH Summary (CVH-S)		CVH Behavior (CVH-B)		CVH Factors (CVH-F)	
Participant Subgroups	Group	median (IQR)	p-value	median (IQR)	p-value	median (IQR)	p-value
Race/Ethnicity	Non-Hispanic White	74.6 (64, 81.5)	0.146	55 (33.1, 65)	0.01	86.9 (68.8, 92.5)	0.894
	Any US Minority Racial/Ethnic Background	67.1 (60, 77.5)		42.5 (25, 53.1)		82.5 (72.5, 95)	
Age	< 16 yo	71.7 (61.7, 79.6)	0.265	52.5 (41.2, 65)	<0.001	81.2 (67.5, 90.6)	0.105
	> or = 16 yo	66.7 (59.6, 77.1)		32.5 (21.2, 53.8)		85 (74.4, 97.5)	
Diagnosis	JSLE	63.3 (57.5, 75.4)	0.003	37.5 (25, 52.5)	0.016	77.5 (68.4, 95)	0.115
	JDM	73.8 (67.9, 81.5)		55 (35, 65)		90 (78.8, 93.1)	
Gender	Female	68.3 (60.8, 77.5)	0.311	45 (25, 55)	0.536	80.6 (70.6, 93.4)	0.359
	Male	76.7 (60, 81.7)		52.5 (30, 65)		90 (75, 93.8)	
On Corticosteroids	Yes	67.5 (60, 77.1)	0.316	45 (32.5, 62.5)	0.248	77.5 (69.4, 90)	0.025
	No	71.7 (61.2, 80.8)		42.5 (23.8, 55)		90 (76.2, 95)	

Abstract 901 Table 3 Median CVH indicator scores by subgroup

		CVH Behavior Indicators						CVH Factors Indicators					
Participant Characteristics	Group	Diet Quality	p-value	Physical Activity	p-value	Body Mass Index	p-value	Blood Pressure	p-value	Non-HDL Cholesterol	p-value	HbA1c	p-value
Race/Ethnicity	Non-Hispanic White	50 (25, 50)	0.15	60 (45, 80)	0.004	100 (70, 100)	0.882	77.5 (60, 100)	0.667	60 (40, 100)	0.369	100 (100, 100)	0.413
	Any US Minority Racial/Ethnic Background	25 (25, 50)		40 (20, 60)		100 (70, 100)		100 (55, 100)		100 (60, 100)		100 (100, 100)	
Age	< 16 yo	50 (25, 50)	0.045	60 (40, 80)	<0.001	100 (70, 100)	0.632	100 (55, 100)	0.748	60 (40, 100)	0.041	100 (100, 100)	0.904
	> or = 16 yo	25 (25, 50)		40 (20, 60)		100 (70, 100)		80 (55, 100)		100 (60, 100)		100 (100, 100)	
Diagnosis	JSLE	25 (25, 50)	0.395	40 (20, 60)	0.012	100 (70, 100)	0.533	80 (53.8, 100)	0.249	60 (40, 100)	0.32	100 (100, 100)	0.132
	JDM	50 (25, 50)		60 (40, 80)		100 (70, 100)		100 (75, 100)		100 (60, 100)		100 (100, 100)	
Gender	Female	50 (25, 50)	0.556	40 (40, 80)	0.545	100 (70, 100)	0.601	80 (55, 100)	0.595	70 (40, 100)	0.618	100 (100, 100)	0.384
	Male	25 (25, 50)		60 (40, 80)		100 (70, 100)		100 (75, 100)		100 (60, 100)		100 (100, 100)	
On Corticosteroids	Yes	50 (25, 50)	0.317	60 (40, 80)	0.406	85 (70, 100)	0.424	100 (55, 100)	0.753	60 (40, 100)	0.002	100 (100, 100)	0.261
	No	25 (25, 50)		40 (40, 60)		100 (70, 100)		80 (55, 100)		100 (60, 100)		100 (100, 100)	

Clinical: 'Systemic Lupus Erythematosus – Treatment'

902 EFFECT OF CUMULATIVE HYDROXYCHLOROQUINE DOSE ON PREVENTION OF DAMAGE PROGRESSION AND MAJOR ADVERSE CARDIOVASCULAR EVENTS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Background/Purpose Hydroxychloroquine (HCQ) has demonstrated benefit in multiple outcomes in systemic lupus erythematosus (SLE), including disease activity, flare rates, survival, and thrombotic events. Previous studies, however, assessed HCQ as a dichotomous variable and may have been confounded by discontinuation of HCQ in more severely ill patients. A better understanding of the impact of sustained HCQ use on key disease outcomes will allow clinicians to more accurately appraise the benefits and risks of continued use. We examined cumulative HCQ doses in a cohort of SLE patients to better assess the relationship with damage accrual. A lupus severity index (LSI) was employed to control for potential confounding from disease severity.

Methods We studied a prospective cohort of 286 adult SLE patients, followed for a minimum of 10 years. The cumulative HCQ dose from time of diagnosis to last known follow-up was calculated via retrospective chart review. This value was then divided by SLE disease duration to give an average HCQ dose/year disease duration (HCQ/year). Disease damage was assessed using SLICC/ACR Damage Index (SDI) scores at baseline, 3, and 5 years after enrollment. The incidence of major adverse cardiovascular events (MACE) was defined as coronary artery disease, myocardial infarction, ischemic stroke, transient ischemic attack (TIA), or peripheral artery disease. A validated lupus severity index (LSI) was calculated. Student's t-test and

Spearman's test were used for bivariate analysis. Logistic regression was used for multivariate analysis.

Results The cohort was composed of 99% females (mean age 40.7 years). There was a statistically significant negative correlation between HCQ/yr and SDI at baseline, 3, and 5 years (table 1). At all specified time points, patients with an SDI of zero had a higher mean HCQ/yr compared to patients with SDI $\geq$1 ($p<0.001$) (table 2). 68/286 (23.8%) of patients had at least one MACE during follow-up. Patients with a MACE had lower mean HCQ/yr compared to patients without MACE ($p=0.03$). The same pattern was seen when looking at cardiac events ($p=0.04$) and ischemic stroke ($p=0.02$) (table 3). The LSI mean was significantly higher in patients with SDI >1 at baseline, 3, and 5 years ($p < 0.009$) and in patients with MACE ($p=0.05$). In a logistic regression model that included LSI and HCQ/yr, the odds of any MACE significantly decreased with higher mean HCQ/yr (OR 0.994, $p=0.027$).

Conclusion We found an inverse association between average yearly HCQ dose and damage in our cohort of SLE patients. This was seen with overall damage accrual and more specifically with cardiovascular damage. When the LSI was applied to our cohort of patients, it proved to be a useful predictor of long-term damage accrual; however, when the LSI was applied as a potential confounder to the inverse association between higher mean HCQ/yr and MACE, the association was still significant. These findings reaffirm existing data on the benefits of HCQ use in SLE and provide additional support for the continuous use of HCQ at appropriate dosages over the course of disease unless clearly contraindicated.

Abstract 902 Table 1 Correlation between lifetime yearly HCQ dose and SDI

SDI	Correlation	p-value
Baseline	-0.21	p<0.001
3 Years	-0.20	p=0.001
5 Years	-0.21	p<0.001

Abstract 902 Table 2 Relationship between lifetime HCQ dose per year disease duration and disease damage

	Lifetime Yearly Average HCQ Dose (grams, mean ± SD)		p-value
	SDI = 0	SDI ≥ 1	
Baseline	97.2 ± 49.2 (n=97)	74.2 ± 54.3 (n=189)	p<0.001
3 years	99.2 ± 47.0 (n=75)	76.4 ± 54.6 (n=204)	p<0.001
5 years	103.4 ± 45.0 (n=65)	77.2 ± 53.9 (n=200)	p<0.001

HCQ = hydroxychloroquine; SD = standard deviation; SDI = SLICC/ACR damage index.
Note: HCQ 200 mg/day is equivalent to 73 grams/year, HCQ 300 mg/day is equivalent to 110 grams/year, HCQ 400 mg/day is equivalent to 146 grams/year.

Abstract 902 Table 3 Relationship between lifetime yearly HCQ dose and major adverse cardiovascular events

	Lifetime Yearly Average HCQ Dose per year disease duration(grams, mean ± SD)		p-value
	Had Event	No Event	
Any MACE	71.2 ± 58.0 (n=68)	85.4 ± 51.9 (n=218)	p=0.03
Cardiac Event	56.1 ± 58.1 (n=17)	83.7 ± 53.1 (n=269)	p=0.04
Stroke/TIA	62.2 ± 52.7 (n=39)	85.2 ± 53.2 (n=247)	P=0.02

HCQ = hydroxychloroquine; SD = standard deviation; CVE = cardiovascular event; TIA = transient ischemic attack.

Lupus & Cardiovascular Disease

903 CARDIOVASCULAR RISK MANAGEMENT IN CUTANEOUS AND CUTANEOUS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Objective Patients with lupus erythematosus (LE) are at heightened risk for clinical events, chiefly heart attacks and strokes, from atherosclerotic cardiovascular disease (ASCVD). We recently proposed new guidelines to assess and manage ASCVD event risk specifically in LE. Here, we examined current cardiovascular management in light of these new recommendations.

Methods We studied our entire UPenn Longitudinal Lupus Cohort of patients with cutaneous LE, without (CLE-only) or with (CLE+SLE) concurrent systemic LE, for whom we had full access to medical records (n=370, LE-ASCVD Study Cohort).

Results Of our LE-ASCVD Study Cohort, 336/370 (90.8%) had a designated primary-care physician. By the new guidelines, the most recent LDL levels were above-goal for 249/370 (67.3%) **(figure 1)**. Two-hundred sixty-six (71.9%) had hypertension, which was under- or un- treated in 198/266 (74.4%) **(figure 2)**. Of current smokers, 51/63 (81.0%) had no documented smoking cessation counseling or referrals. Diabetes and triglyceridemia were generally well-managed. Of the Cohort, 278 qualified for two widely used online estimators of ASCVD event risk in primary prevention: the ACC-ASCVD Risk Estimator Plus and QRisk3. We also stratified these 278 patients into our recently defined categories of ASCVD event risk in LE. These three methods for estimating ASCVD event risk showed clinically meaningful discordance for 169/278 (60.8%) **(figure 3)**. The documented rate of ASCVD events in the first 10 years after enrollment was 13.5% (95% CI 8.9%, 17.9%), similar between CLE-only and CLE+SLE, indicating an at-risk population despite the preponderance of women and a median age at enrollment of only 47 years **(figure 4)**.