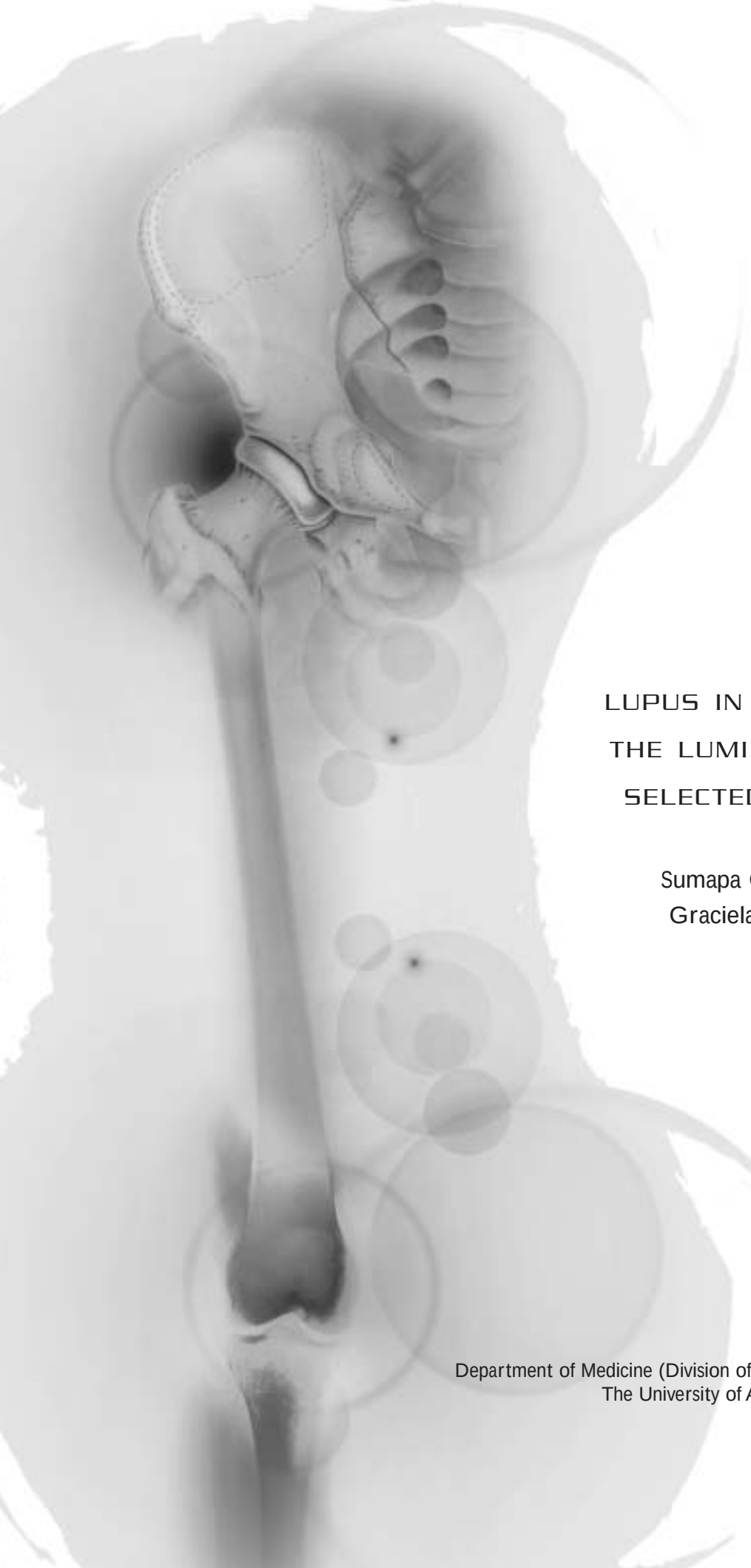

LUPUS IN MINORITIES, THE LUMINA COHORT: SELECTED FINDINGS

Sumapa Chaiamnuay
Graciela S. Alarcón

Department of Medicine (Division of Clinical Immunology and Rheumatology)
The University of Alabama at Birmingham

ABSTRACT

Clinical manifestations and outcome in systemic lupus erythrematosus (SLE) vary in patients from different ethnic groups; genetic, socioeconomic and psychosocial-behavioral factors may explain these differences. The LUMINA (*LU*pus in *MI*nority populations: *NA*ture vs Nurture) cohort was established in 1994, in order to further delineate the influence of these factors. LUMINA is a longitudinal, multi-center, multiethnic (Hispanics, African Americans, and Caucasians) cohort of SLE patients (less than 5 years of disease duration). In this review we are presenting selected data pertinent to cohort characteristics, disease activity, damage accrual, and vascular arterial events in our cohort.

Keywords: LUMINA; Ethnicity; Systemic Lupus Erythematosus

RESUMO

As manifestações clínicas e o prognóstico no Lúpus Eritematoso Sistémico (LES) variam entre doentes de grupos étnicos diferentes; factores genéticos, socioeconómicos e comportamentais poderão explicar estas diferenças. A coorte LUMINA (*LU*pus in *MI*nority populations: *NA*ture vs Nurture) foi iniciada em 1994, de forma a compreender a influência destes factores. Esta coorte de doentes com menos de 5 anos de duração de LES é longitudinal, multicêntrica e multiétnica (hispânicos, afro-americanos e caucasianos). Nesta revisão apresentamos dados seleccionados sobre as características da coorte, actividade da doença, lesão de órgão e eventos vasculares.

Palavras-Chave: LUMINA; Etnias; Lúpus Eritematoso Sistémico.

LUPUS IN MINORITIES, THE LUMINA COHORT: SELECTED FINDINGS

Sumapa Chaiamnuay M.D. and Graciela S. Alarcón, M.D., M.P.H.

Introduction

Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease of unclear etiology; genetic predisposition underlies its occurrence and probably its course. In the US, SLE occurs more frequently in minority population groups such as African Americans, Hispanics, Asians and Native-Americans. The disease appears to be also more severe in these population groups, as evidenced by higher mortality rates and greater damage accrual in different organ systems[1-3]. Similar findings have been observed in minority population groups in the United Kingdom[4].

The LUMINA cohort, an acronym derived from *LU*pus in *MI*norities: *NA*ture vs Nurture, was constituted in 1994 as a longitudinal study of outcome. The primary objective of LUMINA is to clarify the role that genetic and non-genetic factors have in the expression, course and outcome of SLE in patients from different ethnic groups. It is a collaborative effort between the University of Alabama at Birmingham, the University of Texas Health Science Center at Houston and the University of Puerto Rico Medical Sciences Campus. Patients who meet four of the ACR criteria for the classification of SLE are recruited into LUMINA if their disease duration is less than 5 years (from the date of diagnosis). These patients are also of self-defined (Hispanic, African-American or Caucasian) ethnicity. Visits are conducted at entry into the cohort, at 6 months and yearly thereafter.

From the Department of Medicine (Division of Clinical Immunology and Rheumatology), The University of Alabama at Birmingham, Birmingham Alabama, USA.
Grant support: National Institute of Arthritis and Musculoskeletal and Skin Diseases #R01-AR42503 and General Clinical Research Centers #M01-RR02558 (University of Texas Health Science Center at Houston) and M01-RR00032 (University of Alabama at Birmingham) and the National Center for Research Resources (NCRR/NIH) RCMI Clinical Research Infrastructure Initiative (RCRII) award 1P20RR11126 (University of Puerto Rico).

It is impossible to summarize all the data generated from the LUMINA cohort in this brief article. We thus have chosen to address in some detail the following: cohort characteristics, disease activity, damage accrual, and vascular arterial events. We will then describe how we expect to continue our research efforts as our cohort enters its second decade of existence.

Cohort Characteristics

As of June 2005, the LUMINA cohort consists of 612 patients, 117 (19%) Hispanics from Texas, 102 (17%) Hispanics from Puerto Rico, 222 (36%) African Americans and 171 (28%) Caucasians. As shown in Table 1, the majority of the patients are women (91%). Caucasians and Hispanics from Puerto Rico are older than the African Americans and Hispanics from Texas at entry to the cohort.

Hispanic patients from Texas and from the Island of Puerto Rico are quite different with the former having comparable features similar to the African Americans and the later having comparable features to the Caucasians. In general, the Hispanics from the Island of Puerto Rico (as well as the Caucasians), have overall more benign disease (longer time to the accrual of four ACR criteria, more cutaneous manifestation and less serositis, renal involvement and thrombocytopenia), and better socioeconomic status (education, marital status, health insurance, income and home ownership) than the Hispanics from Texas and the African Americans. These data are depicted in Table 1.

The underlying basis for the differences observed among these two Hispanic subgroups is probably multifactorial, as we will now briefly address: (1). Genetic factors: Both Hispanic subgroups are populations of mixed ethnicity (Caucasian/European, African American, and Ameridian/Native of the America continent). The predominant ancestry for the Hispanics from Texas (of Mexican or Central American in origin) is Amerindian, whereas those from the Island of Puerto Rico have a much

Table 1. Baseline Socioeconomic-Demographic and Selected Clinical Features of LUMINA Patients as a Function of Ethnic Groups

	Ethnic Group				
	Hispanic				
Variables (means or %)	Texas n=117	Puerto Rico n=102	African American n=222	Caucasian n=171	p value
Gender, women	93	95	90	85	0.026
Age, years	33	37	35	41	<0.001
Education, years	11	15	13	14	<0.001
Married/living together	54	60	32	73	<0.001
Health insurance	50	99	81	87	<0.001
Poverty, below line	42	30	46	14	<0.001
Home ownership	53	78	50	72	<0.001
Acute onset	52	9	42	28	<0.001
Cutaneous manifestations					
Malar rash	44	64	39	57	<0.001
Discoid rash	3	12	23	5	<0.001
Photosensitivity	40	75	32	67	<0.001
Serositis	59	12	55	31	<0.001
Renal involvement	42	21	41	16	<0.001
Thrombocytopenia	23	4	18	12	<0.001

Table 2. Disease Activity in LUMINA Patients as a Function of Ethnic Groups

	Ethnic Group				
	Hispanic				
Variables (means or %)	Texas n=117	Puerto Rico n=102	African American n=222	Caucasian n=173	p value*
SLAM† score at baseline visit	11.1 (6.5)	6.8 (4.0)	11.1 (6.4)	7.9 (4.1)	<0.001
SLAM score at last visit	9.7 (6.0)	5.8 (3.3)	9.0 (5.9)	5.9 (4.5)	<0.001
Physician global assessment at baseline visit ‡	2.7 (2.4)	1.2 (1.2)	3.1 (2.4)	2.2 (1.6)	<0.001
Patient global assessment at baseline visit ‡	3.4 (3.0)	3.6 (2.8)	3.9 (3.0)	3.9 (2.6)	NS
Discrepancy at baseline visit §	0.7 (2.8)	2.4 (2.6)	0.8 (3.0)	1.7 (2.8)	<0.001

*p values ≤ 0.05 are noted; † Systemic Lupus Activity Measure; ‡ using a 10 cm visual analogue scale or VAS; § between Patient and Physician Global Assessment by VAS.

smaller Ameridian ancestry but larger African and Caucasian/European ancestries than those from Texas[5]. Not surprisingly, therefore, these two subgroups have distinct distributions of HLA-DR and HLA-DQ alleles; for example, *HLA-DRB1*08*, which is associated with presence of renal involvement, is more frequent in the Hispanics from Texas than those from the Island of Puerto Rico[6]. (2). Environmental factors: particularly sun exposure. According to the US 2001 National Weather Service, the reported UV index in San Juan, Puerto Rico is higher than in Houston, Texas. Given that UV light can aggravate and also induce cutaneous lupus[7,8], the presence of more cutaneous manifestations in the Hispanics from the Island of Puerto Rico than in those from Texas seems logical. (3). Socioeconomic factors: Although the differences in the various socioeconomic parameters between these two subgroups are greatly evident, when these variables were entered into the regression analyses

Table 3. Multivariable Model for Persistently High Disease Activity in LUMINA Patients

Variable	Parameter Estimate	p value*
SLAM† at baseline visit	0.290	<0.001
African American ethnicity	0.982	0.040
Gender (men)	1.783	0.007
Social support	-0.447	0.003

*p ≤0.05 are shown (14); † Systemic Lupus Activity Measure.

for disease activity and damage accrual, none of them was significant; instead ethnicity was retained as a variable. These findings probably relate to the tight association between non-Caucasian ethnicity and poor socioeconomic status; ethnicity may override the socioeconomic factors and thus represent a composite of both ethnicity and socioeconomic factors.

Disease Activity

Disease activity , as measured with the Systemic Lupus Activity Measure (SLAM)[9,10], has been found to be higher in the Hispanics from Texas and the African Americans than in the Caucasians and the Hispanics from Puerto Rico regardless of the time at which it has been assessed[11,12]. Similar findings apply to the physician's global assessments of disease activity (measured on a 10-cm visual analogue scale) which has been higher in the African Americans and Hispanics from Texas than in the other two ethnic groups[11,12]. In contrast, patients in all four ethnic groups tend to rate their disease activity at comparable levels; therefore, there are discrepancies between the patients' and physicians' assessment of disease activity which may result in health-seeking behaviors or in non-compliance[13]. These data are shown in Table 2.

We have also examined the variables associated with persistently high disease activity, defined as a SLAM >10 in more than 50% of visits and an average SLAM score >10; persistently high disease activity was found to be independently associated with disease activity at the baseline visit, African American ethnicity, male gender and low levels of social support[14]. Of interest, none of the immunogenetics variables examined was retained in the regression model, even after baseline disease activity was removed from the model (given that earlier cross-sectional analyses have shown that disease activity was associated with the absence of *HLA-DRB1*0301*[12]). These data are greatly relevant as active disease is an important predictor of damage accrual and damage, in turn, is a predictor of mortality[14,15]. These data are shown in Table 3.

Damage

Damage accrual is the irreversible consequence of either the disease itself or the treatments used for it. Damage is being measured with the Systemic Lupus International Collaborating Clinics (SLICC) ACR Damage Index (SDI)[16]. The variables predictive of damage accrual had been examined earlier[17]. Half of the patients have already accrued so-

Table 4. Poisson Multivariable Analyses of Damage Accrual in LUMINA patients

Feature	Parameter Estimate	Chi-square	p value*
Age, years	0.0214	53.48	<0.0001
Ethnicity†			
Hispanic from Texas	0.5847	24.28	<0.0001
Hispanic from Puerto Rico	-0.9989	26.96	<0.0001
African American	0.4582	21.41	<0.0001
Total disease duration§	0.0061	49.04	<0.0001
ACR¶ criteria number at baseline visit	0.1162	20.42	<0.0001
SLAM** score at diagnosis	0.0316	34.24	<0.0001
Glucocorticoid use (mean dose)	0.0240	42.89	<0.0001

*p ≤0.05 are shown; †Caucasian ethnicity is the reference group; §from diagnosis to last visit; ¶American College of Rheumatology; **Systemic Lupus Activity Measure [Modified from Vitae Academia Biomedica Digital.2004;20: http://caibco.ucv.ve/caibco/CAIBCOVitae/VitaeVeinte/Portada/homevitae.php (19)].

Table 5. Multivariable Analyses of Predictors of Time to the Occurrence of Initial Damage in LUMINA patients

Variable	Hazard Ratio*	95% Confidence Interval
Hispanic (Texas) ethnicity (reference category: All other patient groups)	2.11	1.15 – 3.88
SLAM† score	1.09	1.04 – 1.15
Thrombotic events‡	7.66	2.13 – 27.51
Maximum prednisone dose (reference category: prednisone dose > 30mg/day)		
10-30 mg/day	0.46	0.22 – 0.96
< 10 mg/day	2.53	1.15 – 5.55

*A Hazard Ratio (HR) ≥1 indicates a shorter time and a HR <1 a longer time; †Systemic Lupus Activity Measure; ‡central and/or peripheral, venous and/or arterial [Modified from Toloza SM et al. Arthritis Rheum. 2004;50: 3177-86 (23)].

me damage at the baseline visit; the organ systems more frequently involved were neuropsychiatric (20%), renal (16%) and ocular (15%)[17]. African Americans showed higher SDI scores at the baseline visit; however, the Hispanics (from Texas) tend to accrue damage more rapidly[18]. We have now reexamined these data. Factors found to independently predict damage accrual were age, Hispanic (from Texas) and African American ethnicities, ACR criteria number, acute disease onset, disease activity at baseline and the use of glucocorticoids[19]. These data are comparable to the data we had previously reported[17] and are now depicted in Table 4.

In other analyses, we have found : (1). that the presence of damage is a strong predictor of the accrual of further damage[20]; (2). that hydroxychloroquine usage may also prevent the accrual of damage overall (in particular renal and cardiovascular damage) in patients who have not yet accrued any damage[21]; and (3). that among patients that have not yet accrued any damage at entry into the cohort (n=158), Hispanics (from Texas) accrue damage more rapidly than patients in the other three ethnic groups. Independent predictors of more rapid damage accrual among these patients were baseline disease activity, the occurrence of thrombotic events and prednisone dosage < 10 mg/day. Interestingly a prednisone dose between 10 and 30 mg per day was found to be protective of the accrual of initial damage in these analyses[22]. These data are depicted in Table 5.

Vascular and Thrombotic Events

SLE patients now live longer; cardiovascular disease has thus become the major cause of death in these patients[23]. Lupus patients are at risk of both myocardial infarction and strokes even at a relatively young age[24]. Traditional risk factors have failed to fully explain their occurrence[25]. Identification of other potential risk factors is important in order to prevent these vascular arterial events. We thus decided to study what other risk factors

Table 6. Factors Associated with Vascular Events (VE) in LUMINA Patients

	Univariable analyses			Multivariable Analyses		
Variable % or mean (SD)	Without VE (n=512)	With one or more VE (n=34)	p value	Odds ratio	95% CI*	p value
Age, years	36 (12)	46 (14)	<0.001	1.075	1.037-1.114	<0.001
Smoking	12	32	0.001	3.731	1.391-10.000	0.001
Disease duration†	36 (34)	69 (29)	<0.001	1.452	1.223-1.725	<0.001
hs-CRP, mg/L‡	16	38	<0.001	3.356	1.264-8.929	0.015
Any antiphospholipid antibody§	12	29	0.004	4.717	1.675-13.158	0.004

*Confidence interval; †from the baseline to the last visit; ‡Highest quintile of the distribution of values for high sensitivity C-Reactive protein; §IgM and/or IgG and/or the Lupus Anticoagulant [Modified from Toloza SM et al. Arthritis Rheum. 2004;50:3947-57(27)].

might be implicated in the occurrence of vascular arterial events in our cohort. One or more vascular arterial events (myocardial infarction, angina, stroke, intermittent claudication and gangrene) occurred in 34 out of 546 LUMINA patients (6.2%) at the time these analyses were performed. There were 13 cardiovascular, 18 cerebrovascular and 5 peripheral vascular events. By logistic regression, not only traditional risk factors (older age and smoking), but also disease-related factors such as disease duration, damage accrued prior to the vascular event, higher high sensitivity C-reactive protein (hs-CRP) levels and the presence of antiphospholipid antibodies were found to independently predict the occurrence of vascular arterial events[26]. These data are shown in Table 6.

Given the fact that CRP was found to be an independent risk factor for these vascular events and that plasma CRP levels have been reported to be higher in individuals carrying GT^{20} alleles (a polymorphic dinucleotide GT^{n} repeat in the intron of the C-reactive protein gene)[27], we performed a nested case-control study including 25 patients from those 34 patients who developed vascular arterial events after enrollment and 32 ethnically matched LUMINA patients with no previous vascular arterial events. SLE patients carrying the GT^{20} repeat were more likely to develop vascular arterial events (26.0% vs 15.6%), but the difference was not significant. A higher frequency of the GT^{20} repeat in patients with vascular arterial events, as compared with patients without vascular arterial events, was observed in the African Americans (29% vs 21%) and the Hispanics (33% vs 0%) but not in the Caucasians (14% vs 20%). Having the GT^{20} repeat seems to increase the risk of vascular arterial events as evidenced by the fact that all African Americans homozygous for the GT^{20} repeat, and all four Hispanics heterozygous for the GT^{20} repeat developed vascular arterial events. There was no homozygous for the GT^{20} repeat in the Hispanics and the Caucacians. This pattern was not observed among the GT^{20} repeat Caucasian heterozygous. These data are shown in Figure 1[28].

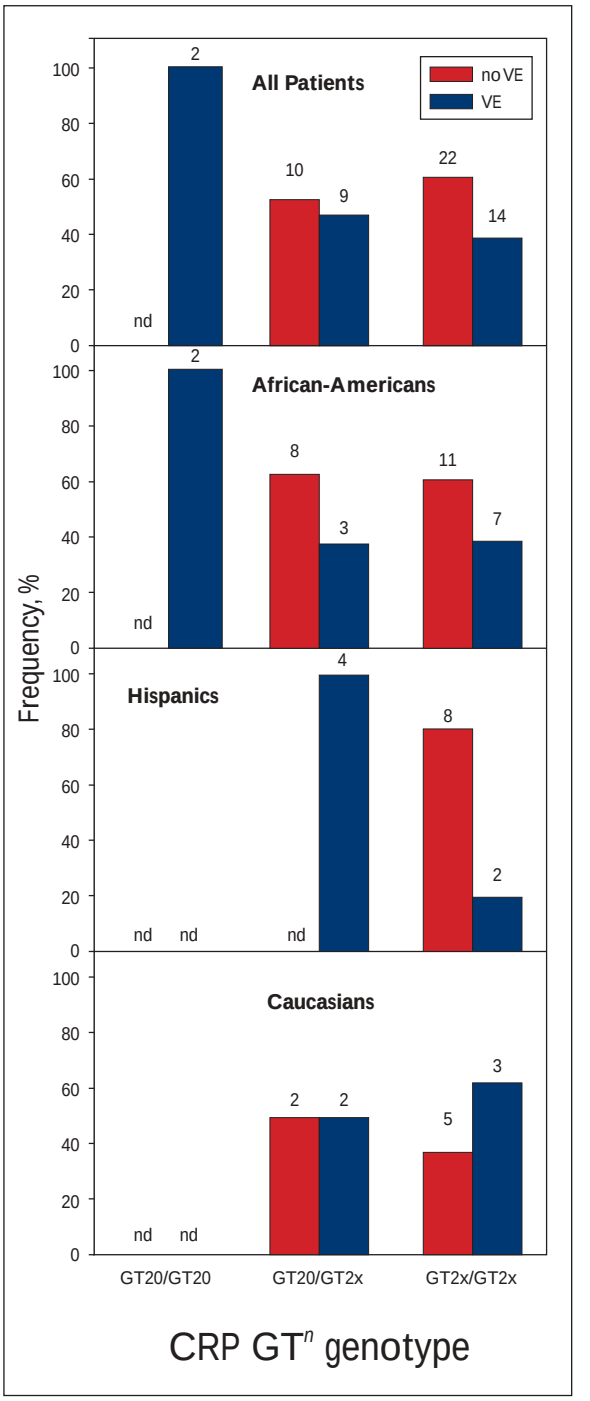

Figure 1. Influence of CRP genotype on risk of vascular events (VE) in SLE patients. For each of the three genotypes, the proportion of patients with and without VE (bars) and the number of patients (numbers) is indicated. nd, not detected [from Szalai AJ et al. Rheumatol. 2005;Epub online March, with permission (25)].

Future Research Agenda

The LUMINA cohort represents a well-developed, high quality, longitudinal database with a serum bank and a DNA repository. The cohort has grown and matured; therefore, we have the ability to ge-

nerate and test diverse hypotheses in order to understand the factors that might impact the clinical course and outcome of SLE, particularly what accounts for the different outcomes observed among minorities. Since a longitudinal study depends, to a large extent, in the ability to retain all patients, we are currently examining the factors associated with "loss to follow-up", for example. We are also examining a variety of other aspects including pregnancy outcomes, the impact of menopause, the impact of hormone replacement therapy, late-onset lupus, hypothyroidism, vasculitis, worsening renal disease and osteonecrosis, among others.

We also hope to be able to examine novel markers of subclinical artherosclerosis while at the same time we follow all patients for the occurrence of clinical and subclinical vascular events. Moreover, we are interested in continuing to examine the disparate outcomes between patients from the different ethnic groups in terms of disease activity, damage accrual and mortality. We hope to be able to contribute to elucidate the basis for these disparities and hopefully set the stage to develop preventive and treatment strategies, aiming at ameliorating the impact of this disease.

References:

1. Reveille JD, Bartolucci A, Alarcón GS. Prognosis in systemic lupus erythematosus. Negative impact of increasing age at onset, black race, and thrombocytopenia, as well as causes of death. Arthritis Rheum. 1990;33:37-48.
2. Serdula MK, Rhoads GG. Frequency of systemic lupus erythematosus in different ethnic groups in Hawaii. Arthritis Rheum. 1979;22:328-33.
3. Walsh SJ, Algert C, Gregorio DI, Reisine ST, Rothfield NF. Divergent racial trends in mortality from systemic lupus erythematosus. J Rheumatol. 1995;22: 1663-68.
4. Hopkinson ND, Jenkinson C, Muir KR, Doherty M, Powell RJ. Racial group, socioeconomic status, and the development of persistent proteinuria in systemic lupus erythematosus. Ann Rheum Dis. 2000;59:116-19.
5. Alarcón GS, Beasley M, Roseman JM, McGwin GJr, Fessler BJ, Bastian H. Ethnic disparities in health and disease: The need to account for ancestral admixture when estimating the genetic contribution to both. Lupus. 2005;14: In Press.
6. Vilá LM, Alarcón GS, McGwin GJr, Baethge BA, Reveille JD. HLA-Class II alleles among two distinct U.S. Hispanic populations with systemic lupus erythematosus. Arthritis Rheum. 2003;48:S574.
7. Hasan T, Nyberg F, Stephansson e, Puska P, Hakkinen M, Sarna S et al. Photosensitivity in lupus erythematosus, UV photoprovocation results compared with history of photosensitivity and clinical findings. Br J Dermatol. 1997;136:699-705.
8. Vilá LM, Mayor AM, Valentin AH, Rodriguez SI, Reyes ML, Acosta E et al. Association of sunlight exposure and photoprotection measures with clinical outcomein systemic lupus erythematosus. Puerto Rico Health Sci J. 1999;18:89-94.
9. Liang MH, Socher SA, Larson MG, Schur PH. Reliability and validity of six systems for the clinical assessment of disease activity in systemic lupus erythematosus. Arthritis Rheum. 1989;32:1107-18.
10. Gladman DD, Goldsmith CH, Urowitz MB, Bacon P, Bombardier C, Isenberg D et al. Sensitivity to change of 3 systemic lupus erythematosus disease activity indices: International validation. J Rheumatol. 1994;21:1468-71.
11. Alarcón GS, Friedman AW, Straaton KV, Moulds JM, Lisse J, Bastian HM et al. Systemic lupus erythematosus in three ethnic groups: III. A comparison of characteristics early in the natural history of the LUMINA cohort. LUpus in MInority populations: NAture vs. Nurture. Lupus. 1999;8:197-209.
12. Alarcón GS, Roseman J, Bartolucci AA, Friedman AW, Moulds JM, Goel N et al. Systemic lupus erythematosus in three ethnic groups: II. Features predictive of disease activity early in its course. Arthritis Rheum. 1998;41:1173-80.
13. Alarcón GS, McGwin G, Jr., Brooks K, Roseman JM, Fessler BJ, Sanchez ML et al. Systemic lupus erythematosus in three ethnic groups: XI. Sources of discrepancy in perception of disease activity: A comparison of physician and patient visual analog scale scores. Arthritis Rheum (Arthritis Care Res). 2002;47:408-13.
14. Calvo-Alén J, Uribe A, McGwin G, Reveille J, Alarcón GS. Persistently active disease early in the course in systemic lupus erythematosus is associated with sociodemographic factors and portends a poor prognosis (damage accrual). 7th International Lupus Conference, New York. 2004;Abstract.
15. Alarcón GS, McGwin G, Jr., Bastian HM, Roseman JM, Lisse J, Fessler BJ et al. Systemic lupus erythematosus in three ethnic group VII: Predictors of early mortality in the LUMINA cohort. Arthritis Rheum (Arthritis Care Res). 2001;45:191-202.
16. Gladman DD, Urowitz MB, Goldsmith CH, Fortin P, Ginzler E, Gordon C et al. The reliability of the Systemic Lupus International Collaborating Clinics/ American College of Rheumatology Damage Index in patients with systemic lupus erythematosus. Arthritis Rheum. 1997;40:809-13.
17. Alarcón GS, McGwin G, Jr., Bartolucci AA, Roseman J, Lisse J, Fessler BJ et al. Systemic lupus erythematosus in three ethnic groups. IX. Differences in damage accrual. Arthritis Rheum. 2001;44:2797-806.
18. Alarcón GS, McGwin G, Jr., Roseman JM, Uribe A, Fessler B, Bastian HM et al. Systemic lupus erythematosus in three ethnic groups. XIX: Natural history of the accrual of the American College of Rheumato-

logy Criteria prior to the occurrence of criteria diagnosis. Arthritis Rheum (Arthritis Care Res). 2003;51:609-15.
19. Fernández M, Calvo-Alén J, Li Q, McGwin GJr, Vilá LM, Reveille JD et al. LUMINA, una Cohorte de pacientes con lupus en minorías étnicas de Estados Unidos. ¿Qué hemos aprendido? (LUMINA, a multiethnic U.S. lupus cohort. What have we learned?). Vitae Academia Biomedica Digital. 2004;20:http://caibco.ucv.ve/caibco/CAIBCOVitae/VitaeVeinte/Portada/homevitae.php .
20. Alarcón GS, Roseman JM, McGwin G, Jr., Uribe A, Bastian HM, Fessler BJ et al. Systemic lupus erythematosus in three ethnic groups: XX. Damage as predictor of further damage. Rheumatology. 2004;43: 349-52.
21. Fessler BJ, Alarcón GS, McGwin GJr, Roseman JM, Bastian HM, Friedman AW et al. Systemic lupus erythematosus in a multiethnic group: XVI. Hydroxychloroquine usage is associated with a lower risk of damage accrual. Arthritis Rheum. 2005;52:1473-80.
22. Toloza SMA, Roseman JM, Alarcón GS, McGwin G, Jr., Uribe AG, Fessler BJ et al. Systemic lupus erythematosus in a multiethnic US cohort (LUMINA) XXII: Predictors of time to the occurrence of initial damage. Arthritis Rheum. 2004;50:3177-86.
23. Urowitz MB, Bookman AA, Koehler BE, Gordon DA, Smythe HA, Ogryzlo MA. The bimodal mortality pattern of systemic lupus erythematosus. Am J Med. 1976;60:221-25.
24. Manzi S, Meilahn EN, Rairie JE, Conte CG, Medsger TA, Jr., Jansen-McWilliams L et al. Age-specific incidence rates of myocardial infarction and angina in women with systemic lupus erythematosus: comparison with the Framingham study. Am J Epidemiol. 1997;145:408-15.
25. 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;44:2331-37.
26. Toloza SMA, Uribe AG, McGwin G, Jr., Alarcón GS, Fessler BJ, Bastian HM et al. Systemic lupus erythematosus in a multiethnic US cohort (LUMINA) XXIII: Baseline predictors of vascular events. Arthritis Rheum. 2004;50:3947-57.
27. Szalai AJ, McCrory MA, Cooper GS, Wu J, Kimberly RP. Association between baseline levels of C-reactive protein (CRP) and a dinucleotide repeat polymorphism in the intron of the CRP gene. Genes Immun. 2002;3:14-19.
28. Szalai AJ, Alarcón GS, Calvo-Alén J, Toloza SMA, McCrory MA, Edberg JC et al. Systemic lupus erythematosus in a multiethnic US cohort (LUMINA). XXX: Association between C-reactive protein (CRP) gene polymorphisms and vascular events. Rheumatol. 2005;44: Epub online March.

Address for correspondence and reprint requests:
Sumapa Chaiamnuay, MD.
Street address: 850 FOT, The University of Alabama at Birmingham
Birmingham, Alabama 35294
Telephone: 205-996-2508
Fax: 205-934-4198
E-mail: sumapa.chaiamnuay@ccc.uab.edu

2006 EULAR Congress

Holanda, Amesterdão
21-24 de Junho de 2006

Limite de Envio de Resumos:
31 de Janeiro de 2006