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Racial and Ethnic Disparities in Disease Activity and Function among Persons with Rheumatoid Arthritis from University-Affiliated Clinics

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Abstract

Background—Health outcomes in rheumatoid arthritis (RA) have improved significantly over the past two decades. However, research suggests that disparities exist by race/ethnicity and socioeconomic status, with certain vulnerable populations remaining understudied. Our objective was to assess disparities in disease activity and function by race/ethnicity and explore the impact of language and immigrant status at clinics serving diverse populations.

Methods—A cross-sectional study of 498 adults with confirmed RA at two rheumatology clinics, a university hospital clinic and a public county hospital clinic. Outcomes included the Disease Activity Score 28 (DAS-28), its components, and a measure of function, the Health Assessment Questionnaire (HAQ). We estimated multivariable linear regression models including interaction terms for race/ethnicity and clinic site.

Results—After adjusting for age, gender, education, disease duration, rheumatoid factor and medication use, clinically meaningful and statistically significant differences in DAS-28 and HAQ were seen by race/ethnicity, language, and immigrant status. Lower disease activity and better function was observed among Whites compared to non-Whites at the university hospital. This same pattern was observed for disease activity by language (English compared to non-English) and immigrant status (U.S.-born compared to immigrant) at the university clinic. No significant differences in outcomes were found at the county clinic.

Conclusion—The relationship between social determinants and RA disease activity varied significantly across clinic setting with pronounced variation at the university, but not at the county clinic. These disparities may be a result of events that preceded access to subspecialty care, poor adherence, or healthcare delivery system differences.

INTRODUCTION

Over the past two decades, health outcomes for persons with rheumatoid arthritis (RA) have improved significantly(1) due to the introduction of biologic therapies(2) and aggressive treatment with combination therapy(3). Biologic therapies (e.g., tumor necrosis factor inhibitors) reduce clinical symptoms, radiographic erosions, and improve quality of life and function(3-9). Despite this progress, worse outcomes have been observed among certain groups, such as African Americans and Hispanics relative to their White counterparts, and among those of lower socioeconomic status (SES)(10-13).

Studies from a variety of populations have shown that RA is more prevalent, the burden of disease greater, and risk of mortality greater among individuals of low SES(10-14). Time to initiate disease-modifying anti-rheumatic drugs (DMARDs) has been shown to be prolonged for minority populations, as well as for patients in public hospital clinics as compared to private settings(15). Observed variation in practice settings may conflate differences by race/ethnicity which may be due to socioeconomic factors, language or immigrant status.

Significant gaps in our knowledge of disparities in this chronic, disabling condition remain. Specifically, variation in disease activity and function among expanding segments of the U.S. population, such as Asians/Pacific Islanders, Hispanics, immigrants and those with limited English language proficiency (LEP), is largely unknown. In the present study, we investigated associations of race/ethnicity with disease activity and function in a diverse RA cohort, including a significant proportion of Asians/Pacific Islanders, immigrants, and those with LEP. The diversity of our sample and inclusion of two clinic sites allowed us to take into account a greater range of vulnerable populations, differences in clinical sites (university vs. public hospital clinics), and treatment differences that could confound or mediate the relationship between race/ethnicity and health outcomes in RA.

METHODS

Data source—The data source was the University of California, San Francisco (UCSF) RA Cohort, a dual-site observational cohort. Beginning in October 2006(16, 17), existing patients were consecutively enrolled from two clinics staffed by UCSF faculty and fellows: the RA Clinic at a county hospital, and a university-based arthritis clinic. At time of enrollment, patients must have been ≥ 18 years of age and met the 1987 ACR criteria for RA. Enrollment in the cohort is ongoing; as of June 2010, there were 498 patients in the cohort. The research protocol was approved by the UCSF Committee on Human Research.

Data for the cohort were obtained from patients and physicians at the time of each regular clinical visit and integrated with laboratory and radiology test results.

Primary outcome

The primary outcome was the Disease Activity Score 28 (DAS-28), an extensively validated, composite measure of disease activity in RA(18, 19). The DAS-28 is a continuous scale from 0 to 9.4 with established cut-offs: low (≤ 3.2), moderate ($>3.2 - 5.1$), or high (>5.1). An absolute level of disease activity of ≤ 3.2 is considered a clinically meaningful goal for therapeutic intervention(20). The European League Against Rheumatism response criteria define an improvement in DAS-28 of > 1.2 as “good,” and > 0.6 and ≤ 1.2 as “moderate”(21).

The four components of the DAS-28 are tender joint count (0-28), swollen joint count (0-28), patient global assessment of disease activity (0-100 mm visual analogue scale), and an erythrocyte sedimentation rate (ESR). Physicians recorded joint counts at each visit. The patient completed the visual analogue scale for disease activity (0 = no activity and 100 = maximal activity) before each visit in her or his preferred language (English, Spanish, or Chinese). An ESR, measured according to standard Westergren techniques, was drawn at the end of the clinical encounter or within 14 days of the visit. Due to the laboratory component of the score, it was not possible to calculate a DAS-28 for each visit. Accordingly, we selected the first visit per patient with a complete DAS-28 score. The interval between enrollment into the cohort and first DAS-28 score averaged 5 months and over 60% of the scores were obtained within one month of enrollment.

Secondary outcome

At clinic visits, patients completed a self-report measure of function administered by a bilingual research assistant, the Health Assessment Questionnaire (HAQ)(22). HAQ scores range from 0 (no disability) to 3 (severe disability); a minimum clinically important difference is defined as 0.22(22, 23). The HAQ was obtained approximately every six months. Because HAQ scores are relatively stable over time(24), we took the score closest to, but within one year of the first DAS-28 score. All but 26 patients had a HAQ score within that time frame.

Primary predictor

Disparities were first assessed based on self-reported race/ethnicity (Hispanic, Asian/Pacific Islander, African-American, and non-Hispanic White). Because of the possible effects of nativity and language on variation in outcomes, we also explored disparities by immigrant status (non-U.S. born vs. U.S.-born) and preferred language (English vs. other) in separate models.

Other covariates—Patient age, gender, disease duration, rheumatoid factor (RF) status, and clinic site were recorded at enrollment. Education level (less than high school, high school graduate, and any college education) was included as a measure of SES. Medication use as reported by the patient was recorded by the physician in the chart at each visit. Disease-modifying medications were dichotomized into two groups, non-biologic DMARDs (methotrexate, hydroxychloroquine, leflunomide, minocycline, and sulfasalazine) and biologic DMARDs (etanercept, infliximab, adalimumab, rituximab, and abatacept). Corticosteroids and NSAIDs were not categorized as DMARDs(25). Corticosteroids were recorded separately, and categorized as none, low dose (<7.5 mg of prednisone or equivalent) or high dose (≥ 7.5 mg)(26). Medication use was always obtained from the visit with the first DAS-28.

Handling missing data

Except for education, sociodemographic measures were available for all patients. Educational attainment was collected for 364 (73%) of the patients. The only other variables with any missing data were HAQ (5%) and disease duration (9%). To reduce possible bias and loss of power from using only a subset of the data, we performed multiple imputation (MI) to estimate non-reported values and their variability. Using the method of Rubin(27) and Schafer(28), each missing value was estimated 20 times from a Bayesian Markov Chain Monte Carlo (MCMC) model; all analyses were then conducted separately on the resulting datasets and combined using the formulae of Rubin(27) to yield the results presented here. The MI model included all variables associated with either the study outcomes or with having missing values; it also included interaction terms for site with race/ethnicity, language, and immigrant status. Sensitivity analyses including the 330 patients with complete case data showed no substantial differences from the imputed results presented here.

Statistical Analysis

We first examined sample characteristics of the cohort by clinic site. We tested the differences by site, using t-tests for continuous variables and chi square tests for categorical variables. Additionally, we explored the relationships of race/ethnicity and language with site and medication use, calculating chi-square tests for the difference in non-biologic and biologic DMARD use by groups defined by site and either race/ethnicity or language.

To examine differences in DAS-28, HAQ, and the four components of the DAS-28 (tender and swollen joint counts, ESR, and patient global status), we estimated bivariable linear regression models including race/ethnicity. Covariates for multivariable regressions included age, gender, disease duration, education level, RF status, and medication use (including biologic and non-biologic DMARDs and corticosteroids). Because enrollment into the cohort may have coincided with heightened disease activity, the models for DAS-28 and its components also included a variable for the number of months between enrollment and the date of the DAS-28 score. For each outcome measure we also estimated bivariable and multivariable regression models in which we replaced race/ethnicity as the primary predictor first with language and, in a separate model, immigrant status. Regression models were examined using standard collinearity and influence diagnostics; no problems were detected. Squared multiple correlation coefficients (R^2) ranged from 0.20 to 0.35 for these models. All statistical analyses were conducted using SAS v9.2 (Cary, NC).

Exploratory analyses—We ran two additional multivariable models for DAS-28 and HAQ: one adding language and another immigrant status to the models with race/ethnicity. The purpose of these exploratory analyses was to examine whether language or immigrant status could explain any variation in disease activity or function by race/ethnicity.

Addressing distributional differences in clinic sites—Although physicians from the university staff both clinics, the two clinic sites differed in populations served, both in terms of sociodemographics and disease status. Additionally, there were significant interactions of clinic site with the sociodemographic effects for the various outcomes under study. Therefore, we included interaction terms for site and the sociodemographic effects for all models and present results for the sociodemographic variables separately for each clinic site.

RESULTS

Most of the 498 patients included in this study were female (84%), with a mean (standard deviation, SD) age of 54 (14) years and a disease duration of 10 (11) years (Table 1). Patients were almost evenly divided between the two clinic sites. Approximately one-third was non-Hispanic White, 34% Hispanic, 23% Asian/Pacific Islander, 10% African American, and 1% American Indian. A majority (56%) were immigrants. Seventy-six percent were RF positive with a mean (SD) DAS-28 of 4.0 (1.5) and a mean HAQ (SD) score of 1.2 (0.8), signifying moderate disease activity and disability. Sixty-nine percent were on at least one non-biologic DMARD and 31% were on a biologic DMARD; 22% were on no DMARD.

Significant differences were observed between the two clinic sites by sociodemographic characteristics, although the university clinic patients were also racially/ethnically diverse (43% non-White). There were no significant differences by site in non-biologic DMARDs ($p=0.05$) or corticosteroid use ($p=0.40$). In contrast, we observed differences in the use of biologic agents by site (19% at the county clinic compared to 42% at the university clinic, $p<0.001$).

To further explore the relationships of sociodemographics, clinic site, and medication use, we compared non-biologic and biologic DMARD use in groups of patients defined by both the sociodemographic characteristics and site (Table 2). Use of non-biologic DMARDs did not vary by race/ethnicity or immigrant status within clinic sites, nor were there any substantial differences between the two sites when we controlled for either race/ethnicity ($p=0.08$) or immigrant status ($p=0.13$). Non-English speakers at the university hospital clinic were more likely to receive non-biologic DMARDs ($p=0.04$), a difference that was not apparent at the county hospital clinic ($p=0.96$). By contrast, there were pronounced

differences in the use of biologic DMARDs at the two clinic sites across all sociodemographic groups. Regardless of race/ethnicity, language or immigrant status, patients at the county hospital were less likely to receive biologic therapies ($p<0.001$).

Sociodemographic and clinic site differences in disease activity

Observed DAS-28 scores were higher (worse) for patients at the county clinic than at the university hospital clinic (mean 4.4 vs. 3.5, respectively, $p<0.001$). Likewise, all non-White racial/ethnic groups had significantly higher mean DAS-28 scores than Whites. However, in a model of DAS-28 as a function of clinic site and race/ethnicity, there was significant interaction between these two variables (p value for interaction term = 0.04). Results for bivariable and multivariable models were similar; p -values in the text that follows derive from the multivariable models. Significant and clinically meaningful differences in disease activity were seen by race/ethnicity in both bivariable and multivariable analyses at the university hospital clinic (Table 3). A higher mean DAS-28 was observed in all non-White ethnic groups compared to Whites ($p<0.001$). African Americans had an adjusted mean DAS-28 of 4.4 (95% CI, 3.8-4.9); Hispanics, 4.0 (3.6 – 4.4); and Asians/Pacific Islanders 4.2 (3.7 - 4.7), compared to 3.3 (3.0 – 3.6) for Whites at the university hospital clinic. Statistically significant differences were also observed in separate models for nativity ($p=0.01$) and language ($p=0.01$) as reflected by higher mean DAS-28 for immigrant and non-English language groups in both unadjusted and adjusted analyses at the university hospital clinic. While observed disease activity, on average, was worse in the county than the university hospital clinic there were no significant differences in DAS-28 within the county clinic for any of these models ($p = 0.18$ for race/ethnicity, $p = 0.95$ for language, $p = 0.59$ for nativity).

In the multivariable model of DAS-28, not graduating from high school and high-dose corticosteroid use were both associated with higher disease activity, while both biologic and synthetic DMARD use was associated with lower disease activity. None of the other variables, including age, gender, disease duration, RF status, and time from enrollment to first DAS-28 measurement, were associated with disease activity. The adjusted R-square for this model was 0.25.

Function

At the university clinic, African Americans and Hispanics had clinically and statistically significant poorer function (0.5 point higher mean HAQ scores) when compared to non-Hispanic Whites ($p<0.001$; Table 3). A similar, but non-significant pattern was seen for the county hospital clinic ($p=0.49$). Among university clinic patients, immigrants had significantly poorer function, 1.4 (1.2 - 1.6) than U.S.-born subjects, 1.1 (0.9 – 1.2), but this difference was attenuated and no longer significant in the adjusted model ($p= 0.81$).

With respect to the individual DAS-28 components (Table 4), the patient global assessment varied significantly by race/ethnicity with higher adjusted mean scores (worse disease activity) for African Americans compared to non-Hispanic Whites at the university clinic ($p=0.001$). We observed significant differences in adjusted analyses for ESR with mean ESR between 30 and 41 mm/hr for non-White ethnic groups compared to 23 mm/hr for non-Hispanic Whites at the university clinic ($p=0.001$); a similar, but less pronounced pattern ($p=0.049$) was seen at the county clinic. No significant differences were seen in patient global assessment or ESR for nativity or language. No significant differences were observed for joint counts at either clinic site ($p=0.58$ for the university clinic, $p=0.09$ for the county).

Exploratory analyses

To explore whether language or immigrant status explained any of the race/ethnic disparities in disease activity or function, we fit separate models which added each of these variables to the multivariable models already containing race/ethnicity. For both DAS-28 and HAQ, there was no independent significant effect of either language or immigrant status, nor did the addition of either variable modify (i.e., confound) the associations of race/ethnicity with the outcomes. Thus, neither language nor nativity was a mediator of the relationship between race/ethnicity and the DAS-28 or HAQ.

DISCUSSION

In this study of an ethnically diverse population of 498 adults with RA, we found significant variation by race/ethnicity in disease activity and function. Most striking, the relationship between race/ethnicity and RA outcomes differed between two clinic settings, with significant racial and ethnic disparities in disease activity and function observed *only* at the university hospital clinic. Whites were observed to have less disease activity and better function than non-Whites, and English speakers compared to non-English speakers as well as U.S.-born compared to immigrants also had less disease activity at the university hospital clinic; these differences were not observed at the county clinic.

This is the first U.S. study to examine whether variation in disease activity among diverse racial/ethnic groups with RA is moderated by clinic setting. Our study population included a significant number of subjects of Asian/Pacific Islander ethnicity, and with non-English proficiency and immigrant status. The fact that clinically and statistically significant disparities in outcomes persisted even after adjusting for medication use indicates that variation in current treatment – an important indicator of quality of care--did not explain these differences.

Possible mechanisms for the clinic-level differences in outcomes observed in our sample include differences in patient characteristics and preceding events (e.g., residual effects of low socioeconomic status, communication barriers such as health literacy and LEP, or genetic/biologic differences), patient behavior (e.g., adherence or preferences for medication), or delivery system structure (e.g., variation in time to initial rheumatology care and access to treatment). Research in other chronic diseases indicates that limited English language proficiency can contribute to health disparities(29, 30), and ours is the first study to demonstrate this in RA. Of note, the addition of language to the race/ethnicity models in this study had no effect on the race/ethnicity-outcomes associations, suggesting that language does not substantially explain the racial and ethnic disparities observed in our study. While there is significant language diversity in our cohort, the county hospital clinic is staffed with full-time in-person interpreters which may substantially mitigate the contribution of language to disparities at that clinic site. Given the small numbers of non-Hispanic Whites as well as small numbers of English-speaking Hispanics or Asian/Pacific Islanders, our analysis may have been underpowered to distinguish between the effects of language and race/ethnicity at the county hospital.

Significant progress in the field of genetic epidemiology has revealed ethnic differences in the genetic predisposition to RA between persons of European and Asian ancestries(31). While genetics may be one mechanism by which variation in outcomes occur, it is unlikely to be the sole factor(32). Racial/ethnic differences in disease activity within the university but not in the public hospital setting does not support a genetic or biologic basis for observed racial/ethnic differences.

Patient preference is another possible mechanism by which disparities may occur(33, 34). In the 2003 report, *Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care*, the Institute of Medicine notes that patient preferences based on inaccurate understanding may be a source of racial disparity in care(35). Constantinescu, et al. found that African Americans with RA were more risk-averse than Whites(34). Difference in rates of biologic use across settings may also be a reflection of insurance coverage for biologics, a patient's ability to pay, or of sociodemographic differences within all racial/ethnic groups between patients at the university and county hospital clinics, such that university hospital clinic patients have increased access to subspecialty care and fewer barriers to obtaining biologic agents (e.g., lower rates of latent tuberculosis). Despite the fact that we found large differences in rates of biologic agents across clinic sites (more than twice the rate of use for all subgroups at the university hospital compared to the county clinic), differences in outcomes persisted in multivariable models controlling for current treatment, indicating that variation in preferences or access cannot fully explain the differences in RA outcomes.

Our study parallels findings from the United Kingdom where patients from socially deprived areas and those with lower individual-level SES had higher disease activity and poorer function(11, 36, 37). In a U.S. study, Bruce et al. observed worse function, pain and global health among Hispanics and African Americans compared to non-Hispanic Whites but only found statistically significant differences in pain after adjusting for age, gender, education, disease duration, comorbidities, and DMARDs(13). In contrast to our study, that sample was predominantly White and patients were cared for in numerous community and university clinics. A study by Yazici et al examined racial and ethnic differences in baseline clinical status measures in 118 DMARD-naïve subjects with early RA (<3 years) at one site(14); Hispanics had statistically significantly higher (worse) HAQ scores, longer morning stiffness and higher psychological distress scores compared to African Americans and Whites adjusting for age and disease duration. While our study found racial/ethnic differences in patient-reported measures of function and patient global assessments similar to the study by Yazici, we also saw differences in ESR and the DAS-28. Therefore we conclude that racial/ethnic differences are observed in "objective" outcomes beyond those that are patient-reported. Prior examples of racial/ethnic differences in ESR have been reported. Del Rincon et al. found higher ESR levels in Hispanic and African American RA patients compared to non-Hispanic Whites from multiple clinical sites in Texas(32).

Our study has several limitations. The cross-sectional design does not allow inferences regarding causation. If minority RA patients with milder disease tend not to seek or receive subspecialty care, this could lead to an overestimation of disparities. By contrast, the public hospital clinic we studied is staffed with interpreters and routinely utilizes drug-assistance programs for biologic therapies, which may mitigate disparities that could occur in other settings. Our co-variables did not include potential confounders related to SES, including prior access to care, insurance status, and income. However, our main measure of SES was education level, previously shown to be a more reliable measure of long-term SES as it remains relatively constant throughout adult life(38). Given the main findings of our study, that Whites at the university hospital clinic had significantly lower disease activity and better function than non-Whites, this may be explained in part by higher educational attainment among Whites at the university. The pattern of educational attainment by race/ethnicity was the same at both clinic sites with Whites having higher educational levels than other race/ethnic groups. We therefore performed additional analyses which categorized education into four levels rather than three, and then further subdivided the top category of BA degree or higher into college graduate and post-graduate education. The results presented herein were not affected by either change. Due to the limited number of non-Hispanic white subjects at the county hospital (n=19), this study may have been underpowered to detect a true difference between the racial and ethnic groups at the county

hospital. It should be noted however, that the relationship between the other sociodemographic variables of language and immigrant status (which included greater numbers of subjects in each category) with disease activity and function at the county hospital were also not significant which is consistent with the race/ethnicity finding. Another potential limitation is that given the cross-sectional design we are unable to account for variation in disease activity among patients over time (early, potentially un- or undertreated vs. later disease, better controlled) and whether that may differ based on the length of time treated at either clinic. However, disease duration is included in the multivariable models, and descriptively, the majority of patients had longer disease duration with only 15% having less than 12 months and 50% with disease duration greater than seven years. Lastly, we cannot determine whether disparities resulted from problems with delays in diagnosis or initial treatment, access, or self-management (e.g., adherence).

By including a diverse sample of patients who received care from a uniform set of university-employed rheumatologists, this study confirms prior research by demonstrating that racial/ethnic disparities exist in patient-reported and “objective” outcomes (e.g., inflammatory markers). It also extends prior research by demonstrating that disparities exist for Asians/Pacific Islanders as well as for immigrants and non-English speakers, and that clinic setting/context can influence overall disease severity and modify the relationships between race/ethnicity and RA outcomes. The next steps will be to explore whether these across-clinic differences occur as a result of differences in recognition, access, and quality of care in early disease, from the stress associated with disadvantaged circumstances, from challenges in self-management or communication barriers once specialty care is underway, or from some combination of these factors. An additional challenge is to discover how characteristics of patients, providers, and local policies associated with different healthcare systems can affect outcomes and either contribute to or eliminate disparities.

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Significance and Innovation

- In multivariable analyses, lower disease activity and better function was observed among Whites with rheumatoid arthritis compared to non-Whites at the university hospital.
- This same pattern was observed for disease activity by language (English compared to non-English) and immigrant status (U.S.-born compared to immigrant) at the university clinic.
- No significant differences in outcomes were found by race/ethnicity, language or immigrant status at the county clinic.
- This is the first U.S. study to examine whether variation in disease activity among diverse racial/ethnic groups with RA is moderated by clinic setting.

Table 1

Sample description for 498 RA cohort members, by clinic site

Characteristic	All patients	County hospital clinic patients (n=254)	University hospital clinic patients (n=244)	
	mean $\pm$ sd (range) or n (%)			
Age, years	54 $\pm$ 14 (19-86)	52 $\pm$ 13 (19-82)	56 $\pm$ 14 (21-86)	*
Female	419 (84)	221 (87)	198 (81)	
Race/ethnicity				*
African American	48 (10)	26 (10)	22 (9)	
Hispanic	171 (34)	124 (49)	47 (19)	
Asian/Pacific Islander	117 (23)	84 (33)	33 (14)	
American Indian	4 (1)	1 (0)	3 (1)	
White, non-hispanic	158 (32)	19 (7)	139 (57)	
Education				*
<HS graduate	158 (32)	125 (49)	32 (13)	
HS graduate	103 (21)	65 (26)	37 (15)	
Any college education	237 (48)	63 (25)	175 (72)	
Immigrant	278 (56)	204 (80)	74 (30)	*
Language				*
English	287 (58)	75 (30)	212 (87)	
Spanish	127 (25)	107 (42)	19 (8)	
Chinese	70 (14)	61 (24)	9 (4)	
Other	15 (3)	11 (4)	4 (2)	
Disease duration, years	10 $\pm$ 11 (0-53)	6 $\pm$ 11 (0-42)	14 $\pm$ 18 (0-53)	*
Rheumatoid factor positive	380 (76)	209 (82)	171 (70)	*
Medication Use				
Synthetic DMARD	345 (69)	186 (73)	159 (65)	
Biologic agent	152 (31)	49 (19)	103 (42)	*
Both DMARD and biologic	107 (21)	40 (16)	67 (27)	*
Either DMARD or biologic	390 (78)	195 (77)	195 (80)	
Corticosteroids				
None	198 (40)	95 (37)	103 (42)	
Low dose (<7.5mg prednisone or equiv.)	192 (39)	98 (39)	94 (39)	
High dose (7.5+)	108 (22)	61 (24)	47 (19)	

*
p<0.05 for difference by site

Table 2

Treatments received, by sociodemographics and clinic site

Sociodemographic characteristics	County hospital	University hospital	County hospital	University hospital
	% treated with non-biologic DMARD		% treated with biologic DMARD	
Race/ethnicity				
African American	77 (60-94)	55 (32-77)	19 (3-35)	32 (11-53)
Hispanic	69 (60-77)	72 (59-86)	23 (15-30)	55 (41-70)
Asian/Pacific Islander	81 (72-90)	52 (34-70)	13 (6-20)	36 (19-54)
White, non-hispanic	63 (39-87)	67 (59-75)	21 (1-41)	41 (33-49)
p-value for within site differences*	0.23	0.16	0.38	0.19
p-value for between site differences†			0.08	<0.001
Nativity				
Immigrant	75 (68-81)	64 (52-75)	19 (14-25)	41 (29-52)
Native	68 (55-81)	66 (59-73)	20 (9-31)	43 (35-50)
p-value for within site differences*	0.36	0.72	0.73	0.90
p-value for between site differences†			0.13	<0.001
Language				
Other language	73 (67-80)	81 (67-96)	18 (13-24)	38 (20-55)
English	73 (63-84)	63 (56-69)	21 (12-31)	43 (36-50)
p-value for within site differences*	0.96	0.04	0.61	0.56
p-value for between site differences†				<0.001

* p-value from chi-square test for difference within site.

† p-value from chi-square test for difference by site, controlling for sociodemographic characteristic.

Table 3

Measures of disease activity and function, by sociodemographic characteristics and clinic, with and without adjustment for covariates

Characteristic	DAS-28			HAQ		
	Unadjusted	Adjusted ¹	mean (95% CI)	Unadjusted	Adjusted ¹	mean (95% CI)
All participants	4.0 (3.8-4.1)			1.2 (1.2-1.3)		
Model 1: Race/Ethnicity (n=494)²						
County hospital clinic						
African American	4.1 (3.6-4.7)	4.2 (3.6-4.7)		1.4 (1.1-1.7)		1.5 (1.2-1.8)
Hispanic	4.6 (4.4-4.9)	4.3 (4.1-4.6)		1.3 (1.2-1.5)		1.3 (1.1-1.5)
Asian/Pacific Islander	4.3 (4.0-4.7)	4.2 (3.9-4.5)		1.3 (1.1-1.4)		1.2 (1.1-1.4)
White, non-hispanic	4.1 (3.4-4.7)	4.1 (3.4-4.7)		1.0 (0.7-1.4)		1.1 (0.7-1.5)
p-value for within site differences		0.18	0.47		0.49	0.41
University hospital clinic						
African American	4.2 (3.6-4.8)	4.4 (3.8-4.9)		1.5 (1.1-1.8)		1.5 (1.2-1.8)
Hispanic	4.1 (3.7-4.5)	4.0 (3.6-4.4)		1.5 (1.3-1.8)		1.5 (1.2-1.7)
Asian/Pacific Islander	4.2 (3.7-4.6)	4.2 (3.7-4.7)		1.1 (0.9-1.4)		1.1 (0.8-1.4)
White, non-hispanic	3.0 (2.8-3.3)	3.3 (3.0-3.6)		1.0 (0.9-1.1)		1.0 (0.9-1.2)
p-value for within site differences		<0.01	<0.01		<0.01	<0.01
Interaction term p-value		0.04	0.06		0.07	0.22
Model 2: Nativity						
County hospital clinic						
Immigrant	4.5 (4.3-4.7)	4.2 (4.0-4.5)		1.3 (1.2-1.4)		1.2 (1.1-1.4)
Native	4.2 (3.8-4.6)	4.2 (3.9-4.6)		1.3 (1.1-1.5)		1.4 (1.2-1.6)
p-value for within site differences		0.15	0.59		0.94	0.25
University hospital clinic						
Immigrant	4.1 (3.8-4.4)	4.0 (3.7-4.4)		1.4 (1.2-1.6)		1.3 (1.1-1.4)
Native	3.2 (3.0-3.5)	3.6 (3.3-3.8)		1.1 (0.9-1.2)		1.2 (1.0-1.3)
p-value for within site differences		<0.01	0.01		<0.01	0.32

Characteristic	DAS-28			HAQ		
	Unadjusted	Adjusted ¹		Unadjusted	Adjusted ¹	
Interaction term p-value		0.06	0.07		0.03	0.07
Model 3: Language preference						
County hospital clinic						
Other language	4.5 (4.3-4.7)			1.3 (1.2-1.4)		1.2 (1.1-1.4)
English	4.2 (3.9-4.6)			1.3 (1.1-1.5)		1.4 (1.2-1.6)
p-value for within site differences		0.62	0.95		0.15	0.15
University hospital clinic						
Other language	4.5 (4.0-5.0)			1.5 (1.2-1.7)		1.2 (0.9-1.5)
English	3.3 (3.1-3.5)			1.1 (1.0-1.2)		1.2 (1.1-1.3)
p-value for within site differences		<0.01	0.01		0.81	0.81
Immigration term p-value		<0.01	<0.01		0.02	0.16

All results derived from models with an interaction term for site with the given sociodemographic characteristic.

¹ Adjusted for age, gender, education, disease duration, RF status, and medication use (including corticosteroids, DMARDs, biologic agents).

² 4 subjects of American Indian ethnicity dropped from these models.

Table 4Components of DAS-28, by sociodemographic characteristics and clinic, controlling for covariates¹

Characteristic	Patient global assessment (0-100)	Erythrocyte sedimentation rate	Tender Joint Count (0-28)	Swollen Joint Count (0-28)
	mean (95% confidence interval)			
All participants	45 (42-47)	30 (28-32)	4 (3-4)	4 (4-5)
Race/Ethnicity (n=494)²				
County hospital clinic				
African American	51 (42-61)	32 (23-41)	3 (1-5)	5 (3-7)
Hispanic	48 (43-53)	34 (29-39)	5 (4-6)	5 (4-6)
Asian/Pacific Islander	47 (41-52)	34 (29-40)	4 (3-5)	5 (4-6)
White, non-hispanic	45 (33-56)	18 (8-29)	5 (2-7)	5 (3-7)
p-value for within site differences	0.77	0.049	0.09	0.98
University hospital clinic				
African American	56 (46-67)	41 (31-50)	4 (2-6)	4 (2-6)
Hispanic	43 (36-51)	30 (23-37)	4 (3-6)	4 (3-5)
Asian/Pacific Islander	49 (40-58)	36 (28-44)	3 (2-5)	5 (3-6)
White, non-hispanic	36 (31-41)	23 (18-28)	3 (2-4)	4 (3-5)
p-value for within site differences	<0.01	<0.01	0.58	0.71
Interaction term p-value:	0.17	0.30	0.76	0.46
Nativity				
County hospital clinic				
Immigrant	47 (43-52)	33 (29-36)	4 (4-5)	5 (4-6)
Native	47 (40-55)	31 (24-38)	4 (2-5)	6 (4-7)
p-value for within site differences	0.60	0.46	0.18	0.87
University hospital clinic				
Immigrant	43 (37-49)	33 (27-38)	4 (2-5)	4 (3-5)
Native	41 (36-46)	26 (22-30)	3 (2-4)	4 (3-5)
p-value for within site differences	0.45	0.03	0.41	0.28
Interaction term p-value:	0.14	0.12	0.92	0.68
Language preference				
County hospital clinic				
Other language	45 (41-50)	33 (28-37)	4 (4-5)	5 (4-6)
English	51 (45-57)	30 (25-36)	4 (3-5)	5 (4-6)
p-value for within site differences	0.31	0.33	0.19	0.99
University hospital clinic				
Other language	42 (33-52)	28 (19-36)	5 (3-6)	5 (4-7)
English	42 (38-46)	28 (24-32)	3 (2-4)	4 (3-4)
p-value for within site differences	0.77	0.99	0.07	0.02
Interaction term p-value:	0.02	0.66	0.10	0.08

¹ Adjusted for age, gender, education, disease duration, RF status, and medication use (including corticosteroids, DMARDs, biologic agents).² 4 subjects of American Indian ethnicity dropped from these models.