

Patient's Ethnicity Does Not Influence Utilization of Effective Therapies in Rheumatoid Arthritis

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ABSTRACT. *Objective.* Biological agents have revolutionized the treatment of rheumatoid arthritis (RA). Given the previously documented ethnic disparity in the health service literature, we sought to determine if ethnic difference exists in the lag time between the diagnosis of RA and use of first biological agent.

Methods. RADIUS 1 and 2 are observational studies designed to document how rheumatologists treat RA across the United States. The sample analyzed here included early patients with RA who entered RADIUS with the initiation of the first biological agent. Ethnic status was categorized as White (W), African American (AA), and Hispanic (H). Lag time (months from RA diagnosis to initiation of the first biological agent) was the principal outcome variable.

Results. Compared to W (n = 1616), AA (n = 147) and H (n = 116) were more likely to be female, younger, and have less than a high school education. Despite similar swollen and tender joint counts, AA and H had more active disease on the basis of Health Assessment Questionnaire and patient global assessments. Almost 97% of patients had some type of insurance coverage. On multivariable analysis, ethnic affiliation was not associated with lag time (14.5 months W vs 14.9 AA vs 14.3 H; p = NS). Similarly, there were also no significant ethnic differences in time to first DMARD (e.g., methotrexate) initiation.

Conclusion. In a national sample of patients with RA, most of whom were insured, the length of time from diagnosis of RA to initiation of the first biological agent was not significantly different among Whites, African Americans, and Hispanics. (J Rheumatol 2006;33:870–8)

Key Indexing Terms:

DISEASE MODIFYING ANTIRHEUMATIC DRUGS
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BIOLOGICAL AGENTS
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The last 20 years have seen a major shift in the management of rheumatoid arthritis (RA). Previously, disease modifying antirheumatic drugs (DMARD) were withheld until significant joint damage justified the potential additional toxicity. The weight of evidence suggests that delayed use of DMARD therapy will lead to poorer physical function and more severe radiographic joint damage¹⁻⁵. Thus, the early use of DMARD is now the accepted standard.

Most experts recognize a window of opportunity for therapeutic intervention in RA, i.e., the earlier the disease is treated, the better the outcome^{6,7}. Early intervention in RA is important because joint damage begins early, disease progression is the norm, and spontaneous remission is rare⁸⁻¹³. The literature overwhelmingly supports early use of DMARD in improving outcome for patients in terms of disability and radiographic progression, without unacceptable side effects^{4,14-17}.

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The emergence of biological agents in the recent past has revolutionized the treatment and care of patients with RA. Short and longterm controlled trials have consistently shown the superiority of these newer DMARD (e.g., etanercept and infliximab) against traditional DMARD (e.g., methotrexate) for various outcome measures such as pain, physical function, and radiographic damage¹⁸⁻²⁰. Observational studies also confirm the efficacy and relative safety of these newer agents²¹⁻²⁴. Appropriate and early use of biological agents not only alleviates symptoms more rapidly, but also improves longterm prognosis^{21,23,25}. Despite the high cost [i.e., \$16,000 to 20,000 US per patient-year (2001 values)]²⁶ of the biological agents, preliminary economic studies render evidence that the cost-effectiveness of this medication is comparable to other accepted therapies in internal medicine²⁷⁻³².

With the advent of powerful biological agents that can significantly influence the course of early RA, patients from different ethnic backgrounds may not equally benefit from early use of biologics or other types of DMARD. A summary of health services literature published over the past 15 years indicates that ethnic minorities (i.e., African Americans and Hispanics) are less likely than Whites to receive a range of costly procedures, including cardiac revascularization³³, cerebrovascular surgery^{34,35}, total hip and knee replacement³⁶⁻³⁸, and renal transplantation^{39,40}. Ethnic differences also exist in other areas of medicine such as the management of diabetes⁴¹,

use of newer effective antipsychotic medications⁴², and receipt of various nonsurgical treatments including antiretroviral therapy⁴³, preventive services (i.e., childhood immunization, colonoscopy, flu vaccine)⁴⁴⁻⁴⁶, and cancer-directed therapy⁴⁷. Racial disparities persist even in medical systems where the confounding effects of access to medical care are minimized^{48,49}.

Our research goal was to determine if ethnic differences exist in the lag time, specifically, in the number of months from RA diagnosis to the initiation of the first biologic agent (or the first traditional DMARD), in a clinic sample of patients across the United States. Although there are published data^{50,51}, albeit cross-sectional, that suggest RA is clinically and radiographically similar among the various ethnic groups, we hypothesized that African Americans (AA) and Hispanics (H) have longer lag times (i.e., delayed use of the first biological agent or first traditional DMARD) as compared to Whites (W), even after controlling for disease-specific covariates.

MATERIALS AND METHODS

The Rheumatoid Arthritis DMARD Intervention and Utilization Study (RADIUS) is a 2-phase "real-world" observational study that focuses on how DMARD influence RA progression. RADIUS was specifically designed to collect information about how rheumatologists treat RA across the US.

The first phase of RADIUS (R1), a prospective, multicenter, observational registry, enrolled patients with RA who required DMARD therapy. Enrollment began in October 2001 and was completed in December 2002. Inclusion criteria were intentionally broad so the study population would reflect general clinical practice. Patients were required to be at least 18 years of age, meet the 1987 American Rheumatism Association (ARA) criteria for RA⁵², and currently warrant a new DMARD (starting, adding, or switching). Dose augmentation alone did not qualify a patient for enrollment. Excluded were patients who were enrolled in a clinical trial, those with active infection, and women who were breastfeeding or pregnant.

The second phase of RADIUS (R2), which was devised to enrich the etanercept population and capture adverse events, was identical to R1 except it was limited to patients who were given etanercept alone or in addition to their current therapy. Enrollment in R2 began in October 2002 and was completed in June 2003. In addition to the exclusion criteria in R1, R2 also excluded current or previous enrollment in R1 and known allergy to etanercept or any of its components. Patient participation was voluntary for both R1 and R2, and all patients were required to read and sign approved informed consent forms. The institutional review board (IRB) of the investigator's institution and/or Western IRB approved the study. The same data, except for events of interest in R1 initially, were collected on patients at baseline and will be collected for up to 5 years, with visit frequencies determined by the evaluating rheumatologist.

We used the cross-sectional data of R1 and R2 at study entry or at baseline visit. Patient-reported baseline demographic data included date of RA diagnosis, highest education achieved, employment status, and insurance coverage index. The insurance coverage index was the mean number of insurance coverages by type (i.e., health maintenance organization, patient preferred organization, Medicare, Medicaid, Veterans Administration, and private insurance). Physician-reported baseline data included the number of prior DMARD and comorbidity index, a count data of predetermined comorbid conditions of interest (i.e., asthma, chronic liver disease, congestive heart failure, ischemic disease, chronic obstructive pulmonary disease, cutaneous ulcers, leukemia/lymphoma, solid tumor, prior hospitalization for infection, renal disease, and other non-RA connective tissue disease).

The following RA clinical disease activity variables were also collected at baseline: tender and swollen joint counts, Stanford Health Assessment

Questionnaire (HAQ) score⁵³⁻⁵⁷, physician and patient global assessments, and laboratory values (erythrocyte sedimentation rate and C-reactive protein) if evaluated⁵⁸. In both the baseline and followup visit forms, the antirheumatic drug history required completion of the start and the stop dates.

Definitions

Study populations. We defined 2 different study populations. The first population, referred to as the "first biologic" sample, comprised R1 and R2 participants who met the following criteria: (1) RA disease duration ≤ 3 years, (2) entered RADIUS in order to start first biological agent, and (3) identified themselves as W or AA or H. There were 1953 participants who met all the inclusion criteria. After excluding 74 (3.8%) patients who had incomplete or missing diagnosis date, the final sample size was 1879.

The second study population (referred to as "first DMARD" sample) was similar to the first, except that the reason for RADIUS inclusion was the initiation of the first DMARD. Seventy-four patients in R2 and one patient in R1 were excluded because of incomplete or missing diagnosis date (total missing data = 5%). The final sample size was 1405. The 2 study populations were not mutually exclusive. From the first DMARD sample, 294 had a biological agent as their first DMARD.

Investigators. Of the 432 investigators who enlisted in R1 and R2, 230 participated in both. Most were in private practice, while 17 were listed at full-time academic sites. These investigators were distributed throughout the US, with a slight preponderance in the East.

Biological agents. Etanercept (Enbrel), infliximab (Remicade), adalimumab (Humira), and anakinra (Kineret) were considered biological agents.

DMARD. In addition to the biological agents, the following were also counted as DMARD: methotrexate, sulfasalazine, leflunomide, gold, cyclosporine, azathioprine, and minocycline.

Lag time 1 and 2. We have 2 different outcome measures: lag time 1 and lag time 2. Lag time 1 was the number of months from the diagnosis of RA to the initiation of the first biological agent. Lag time 2 was the number of months from RA diagnosis to the initiation of the first DMARD. Both measures ranged from 0 to 36 months. Lag time 1 and 2 were used as the dependent variables for the first biologic and first DMARD samples, respectively.

Statistical analysis. We considered a period of 6 months (SD 12 mo) as the clinically meaningful difference in lag time (1 and 2) between any 2 ethnic groups^{3,4,6}. With 90% power and an alpha level of 0.05 (2 tailed test), 84 patients per ethnic group were required to achieve statistical significance.

Before statistical modeling was performed, the 3 ethnic groups were compared with respect to key clinical or demographic variables. An analysis of variance was used to compare continuous variables and Cochran-Mantel-Haenszel chi-square to compare categorical variables.

An analysis of covariance was used to determine the effect of ethnicity (as a 3-category variable) on lag time 1 (or lag time 2) after controlling for disease activity, socioeconomic factors, and geographic location. R1 and R2 datasets were analyzed separately and combined.

RESULTS

Sample Characteristics

First biologic sample. Table 1 shows the demographic and clinical characteristics of 1879 participants from the R1 and R2 datasets. There were 1616 (86%) W, 147 (7.5%) AA, and 116 (6%) H. As compared to W, AA were more likely to be female (71% W vs 88% AA; $p < 0.05$) and from the Southern US (34% W vs 51% AA; $p < 0.05$). There was no significant difference between W and AA who attained \leq high school education (33% W vs 36% AA; $p =$ nonsignificant). On the other hand, H were more likely to be younger (51 W vs 44 H; $p < 0.05$), female (71% W vs 83% H; $p < 0.05$), and have \leq high school education (33% W vs 49% H; $p < 0.05$), when

Table 1. Characteristics of the “first biologic” sample at study entry (n = 1879). Values are mean ± SD or percentages.

| | White, n = 1616 | African American, n = 147 | Hispanic, n = 116 | p |
|-----------------------------|--------------------|------------------------------|----------------------|----------|
| Age, yrs | 51.5 ± 0.3 | 49.5 ± 1.0 | 44.2 ± 1.2* | < 0.0001 |
| Gender | | | | < 0.0001 |
| Male | 28.8 | 11.6 | 17.2 | |
| Female | 71.2 | 88.4* | 82.8** | |
| Education level | | | | 0.002 |
| ≤ High school education | 33.4 | 36.1 | 49.1** | |
| > High school education | 66.7 | 63.9 | 50.9 | |
| Employment status | | | | 0.2 |
| Currently working | 61.8 | 54.6 | 64.2 | |
| Not currently working | 38.2 | 45.4 | 35.8 | |
| Insurance index | 1.13 ± 0.01 | 1.07 ± 0.03 | 1.08 ± 0.03 | 0.15 |
| Geographic location | | | | < 0.0001 |
| Midwest | 23.4 | 18.4 | 8.6 | |
| Northeast | 25.6 | 23.8 | 22.4 | |
| South | 34.1 | 51.0 | 28.5 | |
| West | 16.9 | 6.8 | 40.5 | |
| Comorbidity index | 0.32 ± 0.02 | 0.24 ± 0.05 | 0.11 ± 0.03** | 0.001 |
| Patient global assessment | 5.9 ± 0.1 | 6.5 ± 0.2* | 6.5 ± 0.2** | 0.001 |
| Physician global assessment | 5.9 ± 0.1 | 6.1 ± 0.2 | 6.3 ± 0.2 | 0.057 |
| HAQ score | 1.2 ± 0.0 | 1.5 ± 0.1* | 1.4 ± 0.1** | < 0.0001 |
| No. of tender joints | 13.0 ± 0.2 | 13.9 ± 0.8 | 14.6 ± 0.8 | 0.07 |
| No. of swollen joints | 10.9 ± 0.2 | 11.1 ± 0.7 | 11.0 ± 0.7 | 0.9 |
| No. of prior DMARD | 1.1 ± 0.0 | 1.1 ± 0.1 | 1.2 ± 0.1 | 0.3 |
| Time to first biologic, mo | 14.5 ± 0.3 | 14.9 ± 0.8 | 14.3 ± 0.9 | 0.8 |

* Statistically significant difference (p < 0.05) between Whites and African Americans. ** Statistically significant difference (p < 0.05) between Whites and Hispanics. Insurance index: the mean number of insurance policies. HAQ: Health Assessment Questionnaire.

compared to W. Geographically, more H were from the Western region of the country, and more W were from the South. As for insurance coverage, the 3 ethnic groups have the same average number of insurers (by type) (1.1 W vs 1.0 AA vs 1.0 H; p = 0.15). Only 3.3% of the sample had no insurance coverage.

Based on HAQ and patient global assessments, AA and H had higher RA disease activity when compared to W (p < 0.05). Despite such findings, the 3 groups had comparable scores on physician global assessment, number of prior DMARD (1.1 W vs 1.0 AA vs 1.1 H; p = 0.34), and number of swollen (11 W vs 11.1 AA vs 11 H; p = 0.9) and tender joint counts (13 W vs 14 AA vs 15 H; p = 0.07). There was no significant difference in lag time 1 (i.e., number of months from the diagnosis of RA to first biological agent) (14.5 months W vs 14.9 AA vs 14.2 H; p = 0.8) among the 3 ethnic groups. In Figure 1, for every 5-month interval in the time to the first biologic, the proportion of W, AA, and H within a particular time period was fairly equivalent.

First DMARD sample. Table 2 shows the demographic and clinical characteristics of 1405 participants from both R1 and 2 datasets. The first DMARD sample was 82% W (n = 1157), 12% AA (n = 161), and 6% H (n = 87). The average ages were 54 (W), 53 (AA), and 45 (H) years. H were significantly younger than both AA and W (p < 0.05). A majority of W were

from the Midwest or the South, while about one-half of AA and H came from the South and West, respectively. As compared to W, a greater percentage of AA were female (71% W vs 82% AA; p < 0.05). In contrast, W and H have comparable gender distribution. Although more H than W had ≤ high school education (44% W vs 61% H; p < 0.05), there was no significant difference between W and AA. Similar to the first biologic sample, there was no difference in terms of insurance coverage among the 3 groups. As a whole, participants in the first DMARD sample were predominantly insured, with only 4.4% having no insurance.

When compared to W, AA scored worse on 2 disease activity variables (i.e., patient global assessment and HAQ score; p < 0.05), despite similar counts of tender and swollen joints and physician global assessment scores. Interestingly, W and H have about the same degree of RA disease activity. When compared to W, H scored worse on the patient global assessment (p < 0.05). W have a higher comorbidity index than H (p < 0.05). As seen in Table 2, the time to first DMARD was about the same in all the 3 groups (5.8 months W vs 5.9 AA vs 6.4 H; p = 0.7]. In Figure 2, for every 5-month interval in time to the first DMARD, the proportion of W, AA, and H within a particular time period was about the same.

Multivariable analyses. After controlling for important

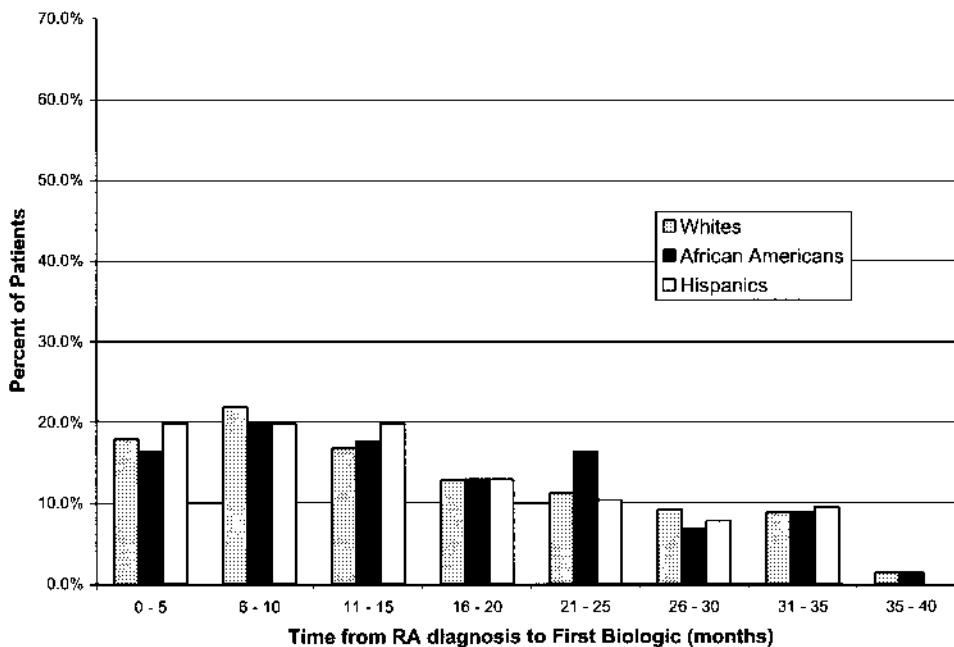


Figure 1. Frequency distribution of lag time 1 (time to first biologic) comparing African Americans, Whites, and Hispanics.

Table 2. Characteristics of the “first DMARD” sample at study entry (n = 1405).

| | White, n = 1157 | African American, n = 161 | Hispanic, n = 87 | p |
|-----------------------------|--------------------|------------------------------|---------------------|----------|
| Age, yrs | 54.6 ± 0.4 | 53.7 ± 1.1 | 45.5 ± 1.5** | < 0.0001 |
| Gender | | | | 0.01 |
| Male | 28.3 | 18.0 | 22.9 | |
| Female | 71.7 | 81.9* | 77.0 | |
| Education level | | | | 0.005 |
| ≤ High school education | 43.8 | 50.6 | 60.5** | |
| > High school education | 56.2 | 49.4 | 39.5 | |
| Employment status | | | | 0.45 |
| Currently working | 57.9 | 53.3 | 60.9 | |
| Not currently working | 42.0 | 46.7 | 39.0 | |
| Insurance index | 1.2 ± 0.02 | 1.1 ± 0.04 | 1.1 ± 0.04** | 0.051 |
| Geographic location | | | | < 0.0001 |
| Midwest | 31.1 | 16.2 | 8.1 | |
| Northeast | 21.7 | 22.4 | 27.6 | |
| South | 31.1 | 50.9 | 17.2 | |
| West | 15.9 | 10.5 | 47.1 | |
| Comorbidity index | 0.3 ± 0.02 | 0.3 ± 0.05 | 0.1 ± 0.04** | 0.01 |
| Patient global assessment | 5.6 ± 0.1 | 6.1 ± 0.2* | 6.2 ± 0.3** | 0.02 |
| Physician global assessment | 5.7 ± 0.1 | 6.0 ± 0.2 | 5.9 ± 0.2 | 0.06 |
| HAQ score | 1.2 ± 0.0 | 1.5 ± 0.1* | 1.3 ± 0.1 | < 0.0001 |
| No. of tender joints | 13.0 ± 0.3 | 13.1 ± 0.7 | 13.1 ± 0.9 | 0.9 |
| No. of swollen joints | 10.9 ± 0.2 | 10.3 ± 0.6 | 10.7 ± 0.9 | 0.6 |
| Time to first DMARD, mo | 5.8 ± 0.2 | 5.9 ± 0.7 | 6.4 ± 0.9 | 0.7 |

* Statistically significant difference (p < 0.05) between Whites and African Americans. ** Statistically significant difference (p < 0.05) between Whites and Hispanics. Insurance index: the mean number of insurance policies. HAQ: Health Assessment Questionnaire.

covariates (i.e., demographic data, including age, geographic location, RA-specific disease activity, and comorbid medical diseases), ethnic status was not associated with lag time 1

(first biologic sample; p = 0.35) or lag time 2 (first DMARD sample; p = 0.51). Ethnic status remained nonsignificant even when R1 and R2 datasets were analyzed separately.

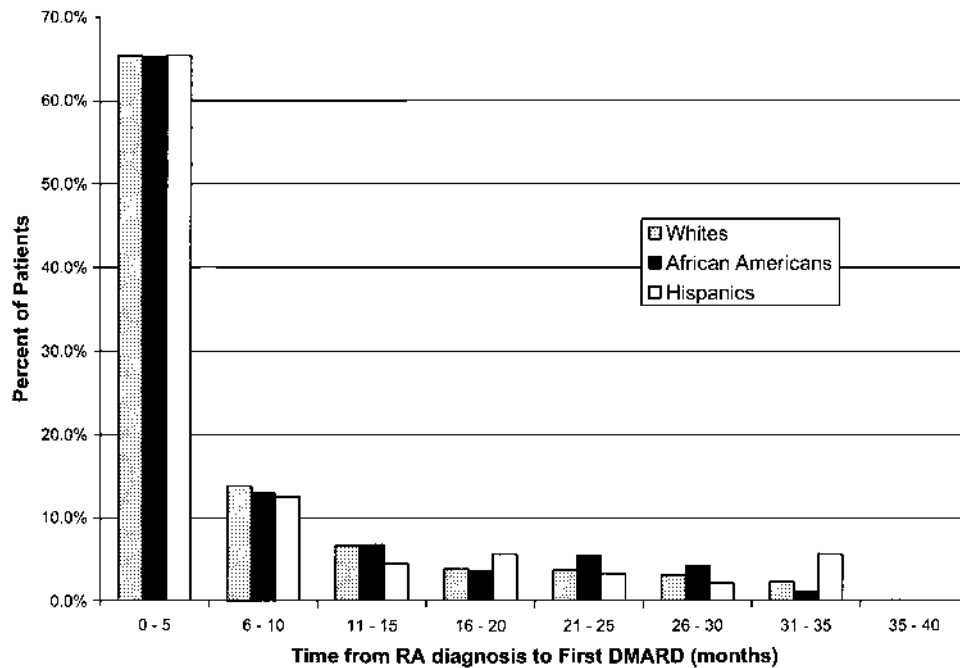


Figure 2. Frequency distribution of lag time 2 (time to first DMARD) comparing African Americans, Whites, and Hispanics.

Interestingly, gender was associated with both lag time 1 and lag time 2. The time to first biologic use was 1.1 months (34 days) longer for females compared to males ($p = 0.04$). Similarly, the time to first DMARD use was 1.4 months (43 days) longer for women compared to men ($p = 0.009$).

In addition, both HAQ ($p = 0.01$) and physician global assessment ($p = 0.01$) correlated (i.e., worse scores were associated with shorter lag time) with lag time 1, but not lag time 2.

DISCUSSION

The data from this large “real-world” observational study of predominantly insured patients with RA demonstrate that the length of time from the diagnosis of RA to the initiation of the first biologic agent or the first DMARD was similar among W, AA, and H. In both the bivariate and multivariable analyses, ethnic status was not associated with either lag time 1 or lag time 2. In contrast, we did find a delay in the use of the first biologic by about 1.1 months (34 days) and the first DMARD by about 1.4 months (42 days) among females as compared to males.

Potential explanations for racial disparities in healthcare use abound, including racial differences in disease severity, patient preferences, provider bias, geographic proximity to care, health beliefs, cultural compatibility with physicians, and access to care (i.e., insurance coverage)⁵⁹⁻⁶⁶. While many studies suggest that ethnic disparities persist even in systems where the confounding effects of access to healthcare are minimized, other studies report that equal access to care occurs in a universally accessible system such as the Veterans Health Administration (VHA). Taylor, *et al* studied 1441 veterans

seeking care for acute myocardial infarction in the VHA system, and found no racial differences in the rate of cardiac catheterization or revascularization after controlling for important covariates⁶⁷. In a large nationwide longitudinal study of chronically ill patients, differences between ethnic groups in use of cardiovascular procedures narrowed markedly or were eliminated after the acquisition of health insurance through Medicare⁶⁸. Powe, *et al* found no ethnic disparity in receipt of life-saving chronic kidney disease treatment in a healthcare system where health insurance was not an issue⁶⁹. Further, after controlling for stage at diagnosis, there were no statistically significant differences between AA and W with regard to prostate cancer treatment received by patients in the Department of Defense Tumor Registry, an equal-access treatment facility⁷⁰. A recent review article concluded that a sizable share of the differences in whether a person has a regular source of care could be reduced if ethnic minorities were insured at levels comparable to those of W⁷¹.

Gender differences in the use of medical services are also well documented⁷²⁻⁷⁶. For example, women are less likely to receive highly active antiretroviral therapy⁷⁷. Giacomini reported that the odds of males receiving most procedures exceeded those of females by 115% for coronary artery bypass grafting, 86% for heart transplantation, 38% for defibrillator implants, 34% for angioplasty, 28% for pacemaker implants, and 24% for hip replacement⁷⁸. While the main focus of our study was ethnic differences in the use of health services, it was interesting to find that the lag to initial DMARD therapy was 42 days longer in women compared to men. We hypothesized that the female concern about the

influence of DMARD and biologic therapies on childbearing potential may have played a role in the observed gender differences in lag times. However, although female sex was significantly associated with longer lag times in the analyses, the magnitude (i.e., 1.4 mo) of the difference may not be clinically meaningful. A 3 to 12-month delay in therapy is believed by several authors to be clinically meaningful^{3-5,79,80}.

Although our study was meant to determine if ethnic disparities exist in lag times from RA diagnosis to effective therapies, it was important to note that the mean overall lag time from RA diagnosis to the first traditional DMARD use was 6 months and to first biological agent use was 14 months. Many clinical trials have clearly documented that even minor delays in initiation of DMARD can have disastrous downstream effects on radiographic outcomes^{3-5,79,80}. Causes for delay in initiation of DMARD are likely multifactorial (e.g., patient reluctance to start DMARD, delay in referral to a rheumatologist, a dearth of specific markers that predict persistent disease, etc.) and deserve further scrutiny.

Our study has several limitations. Because the accuracy of the date of RA diagnosis was critical, we were concerned about potential recall bias. Memory for date is sometimes unreliable. Ethnic minorities may have underestimated the real date of RA diagnosis. For instance, AA may have reported their diagnosis date as November 2000, when in fact the diagnosis was made 6 months earlier. Based on our review of the literature, however, we could not find any evidence to suggest that race or ethnicity is associated with more data recall inaccuracies. In an observational study of patients with RA⁸¹, current disease activity (and not demographic characteristics) was found to be associated with the accuracy of recall of RA symptom-onset date. Specifically, RA patients with higher disease activity tended to be more precise in recalling their symptom-onset date. Because AA and H appeared to have higher disease activity, at least on certain variables (i.e., HAQ and patient global assessments), we may assume that AA and H were at least as accurate, if not more accurate, than W in correctly identifying their diagnosis date. In addition, using total disease duration of ≤ 3 years (as opposed to longer disease duration) in the analyses may also have reduced the chance of inaccuracies in date recall.

In our study, we defined lag times as the time interval from the date of diagnosis to the initiation of the first biologic (or first DMARD) therapy. Alternatively, one can define lag time as the number of months from symptom onset to first DMARD therapy. This latter definition encompasses the interval from symptom onset to the initial physician encounter, and from the initial physician encounter to RA diagnosis. Although we found no ethnic differences using our predefined lag times, it is possible that ethnic minorities may have longer lag times when one considers the duration from symptom onset to RA diagnosis. Ethnic minorities may tend to postpone or delay their first visit to a healthcare provider until they are more disabled, which then delays their diagnosis. Delay in

seeking care by ethnic minorities was found to play a major role in their having a more advanced stage of cancer at presentation⁸²⁻⁹⁰. Given that both AA and H had worse disease activity on HAQ and patient global assessment on initiation of first biologic (or first DMARD), AA and H in our study may have waited much longer than W before their first visit to their physician; or there may have been a delay in presentation of symptoms and diagnosis by the physician. While the current study cannot address this issue, it is a tenable hypothesis that warrants future investigation. Lastly, since our study population primarily comprised patients starting their first biologic agent (or first DMARD), ethnic disparity may still be present if RA patients who are appropriate candidates for biologics have not been considered to receive such an aggressive therapy. A large inception cohort of early RA patients would be required to verify whether such disparity exists.

Our report is the first to examine ethnic and gender differences in the use of the first biologic agent or first DMARD for RA. The use of a large registry such as RADIUS allowed us to study 3 ethnic groups, as opposed to the usual 2-group categorization from prior disparity publications. The participation of clinically diverse RA patients cared for by practicing rheumatologists across the US adds further credence to our findings. Importantly, our study also contributes to the emerging literature suggesting that the provision of insurance among chronically ill patients can provide equalized access of care across ethnic and racial lines. Our data raise the promise that the characteristics of a healthcare system can mitigate ethnic bias in medicine, even in situations where the medical product offered is beyond the reach of most healthcare consumers.

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