Race, Ethnicity, and Disease Outcomes in Juvenile Idiopathic Arthritis: A Cross-sectional Analysis of the Childhood Arthritis and Rheumatology Research Alliance (CARRA) Registry

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ABSTRACT. Objective. To measure the associations between self-reported race and ethnicity and disease outcomes, including joint damage, pain, and functional ability, in children with juvenile idiopathic arthritis (JIA).

Methods. A cross-sectional analysis of children with JIA enrolled in the Childhood Arthritis and Rheumatology Research Alliance (CARRA) Registry between May 2010 and March 2012. Mann-Whitney U test and chi-square testing were used to compare patient characteristics between race (white, African American, or Asian) and ethnicity (Non-Hispanic and Non-Latino; Hispanic or Latino) categories. Logistic regression was used to measure the associations between each race or ethnicity category and the outcome of interest.

Results. Race category was available for 4292 of 4682 children (93% white, 5% African American, Asian 3%). Ethnicity data were available for 4644 (11% Hispanic or Latino). African American children with polyarticular-course JIA had an elevated OR for joint damage on radiographic imaging compared to white children (OR 1.9, 95% CI 1.0–3.1; p = 0.04). Hispanic/Latino children had increased odds of having disability scores > 75th percentile (OR 1.5, 95% CI 1.1–2.1; p < 0.01) compared to non-Hispanic/Latino children; however, these odds were no longer significant when the cohort was limited to children with polyarticular-course JIA. Asian children had decreased odds of higher pain and functional disability compared to white children (p < 0.05).

Conclusion. Race and ethnicity were variably associated with joint damage, pain, and functional ability. Understanding outcome variation between different race and ethnicity groups may help to optimize care for children with JIA. (First Release April 15 2013; J Rheumatol 2013;40:936–42; doi:10.3899/jrheum.121147)

Key Indexing Terms:

JUVENILE IDIOPATHIC ARTHRITIS  RACE  ETHNICITY  DISEASE OUTCOMES

Race and ethnicity are known to influence disease outcomes for a wide range of pediatric chronic diseases, including cancer, asthma, and type I diabetes. Differences in outcomes among race and ethnicity categories are hypothesized to result from a complex combination of biologic, genetic, sociocultural, and socioeconomic effects, although the models are complex and the interactions are not fully understood. While juvenile idiopathic arthritis (JIA) is the most common pediatric rheumatic disease and one of the leading causes of disability in the United States, the associations between race, ethnicity, and disease outcomes are not well described. Investigations of race and ethnicity have described the distribution of different JIA categories among different races and ethnicities, and have reported higher rates of oligoarticular JIA and uveitis among children of European descent and higher rates of rheumatoid factor (RF)-positive polyarticular JIA among children of non-European origin and African American race. However, the association between these findings and clinical or patient-reported outcomes has not been described, primarily because of lack of large databases containing sufficiently detailed patient data regarding disease activity, disease outcomes, race and ethnicity, and additional important covariates including insurance status and income.

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The Childhood Arthritis and Rheumatology Research Alliance (CARRA) is a North American organization of pediatric rheumatologists created to facilitate research in the pediatric rheumatic diseases. The CARRA Registry, which began enrolling patients in 2010, is currently one of the largest registries in the world of children with rheumatic diseases. In addition to data regarding the child’s diagnosis, treatment, and disease activity, the registry also includes family-reported data on race and ethnicity, providing a unique dataset with which to examine the associations between these outcomes.

The objective of our analysis was to describe the association between race and ethnicity and important outcomes in JIA, including joint damage, disability, and pain, to identify children at risk for poorer outcomes who may require more aggressive medical therapies or specialized treatment strategies.

MATERIALS AND METHODS

Children were included in the analysis if they were enrolled into the CARRA Registry between May 2010 and March 2012, and had a physician diagnosis of JIA. Patients were enrolled from 55 sites during that time period. Consent was obtained for all patients. Patients completed their study questionnaires in English. Children are approached for enrollment into the CARRA Registry at any time during their disease course. Patients enrolled in the registry may have data collected from a followup visit about 6 months after their enrollment visit. Because followup data are currently available for only a subset of patients, only data from each patient’s enrollment visit were used for this analysis. Data are collected from both the families and the physician and entered into the database through an online interface by trained clinical research assistants. Race and ethnicity data are provided by the family. Non-English-speaking families were excluded from the registry. Children with missing race or ethnicity data and who were identified as being of more than 1 race or ethnicity were excluded from the analyses of race or ethnicity (n = 176 for race; n = 33 for ethnicity), respectively. American Indian or Alaska native, and native Hawaiian or other Pacific Islander race categories were excluded because of small numbers of patients (n = 19 and n = 9, respectively). One hundred thirty-one children indicated their race as “other” and were also excluded.

Approval for this study was obtained from the Seattle Children’s Hospital institutional review board.

Primary outcomes. Joint damage on radiographic imaging was indicated as present, absent, not obtained, or missing by the treating physician. Patients who did not have radiographic imaging were excluded from the analyses of radiographic outcomes. Disability was assessed by the Child Health Assessment Questionnaire (CHAQ), which was completed by the child or their parent/proxy. The CHAQ measures the child’s ability to perform activities of daily living and is scored on a 0–3 scale, higher scores indicating increased disability. Because CHAQ scores were quite skewed for this cohort, with the majority of children reporting no disability (CHAQ = 0), the CHAQ was dichotomized as either 0 or > 0, based on the median score for the cohort, or as ≤ 0.5 or > 0.5 based on the 75th percentile scores for the cohort. Pain was measured on a 10-point numeric rating scale completed by the patient or parent/proxy, 0 indicating no pain and 10 indicating maximal pain. Pain scores were skewed toward less severe pain and were dichotomized as ≤ 2 or > 2, based on the median score for the cohort, and as ≤ 5 or > 5, based on the 75th percentile.

Covariates. The following covariates were included and were categorized as follows: (1) baseline demographic characteristics: age of disease onset, disease duration, and sex; (2) socioeconomic status: insurance status (dichotomized as yes/no) and annual income (dichotomized as < $50,000 or ≥ $50,000); (3) disease-related characteristics: physician-assigned JIA category based on the Edmonton International League of Associations for Rheumatism criteria (2nd revision), RF status, physician global assessment of disease activity (assessed on a 0–10 numeric rating scale, 0 indicating no active disease and 10 most severe disease), active joint count (dichotomized as < 1 or ≥ 1, because the distribution of this variable was extremely skewed, and both the mean and median active joint count of the cohort was 1); and (4) treatment: current or past treatment with a nonbiologic disease-modifying antirheumatic drug (DMARD), current or past treatment with a biologic DMARD.

Statistical analyses. Descriptive statistics, including proportions, means, and medians, were used to summarize patient characteristics. Variables were compared between race and ethnic categories using chi-square testing for categorical variables and the Mann-Whitney U test for continuous variables. Logistic regression with robust standard errors was used to measure the associations between race and ethnicity and each of the study outcomes.

Multivariable models were developed a priori using the sequential addition of groups of covariates, similar to the methods used by Bruce and colleagues and Iren and colleagues in their analyses of race and ethnicity in rheumatoid arthritis (RA). The blocks of covariates were added into the multivariable models in the following order: (1) patient demographics; (2) socioeconomic status; (3) disease-related characteristics; and (4) treatment. These models were also performed for children with polyarticular-course disease, excluding children with ≥ 4 joints involved during their disease course and children with systemic JIA, to determine whether the associations would be similar for children at higher risk for poorer outcomes.

Analyses were performed using Stata version 10.0 statistical software (Stata).

RESULTS

Race category was available for 4292 of 4682 children (93% white, 5% African American, 3% Asian). African American children were significantly older than white children at disease onset and had a shorter disease duration (Table 1). Asian children also had an older age at onset than white children. There was no significant difference between disease duration prior to symptom onset and first visit to a rheumatologist among race categories. A larger proportion of African Americans than white subjects reported an annual income < $50,000. African American children also tended to have increased disease activity relative to white children, with a larger proportion of children having an active joint count ≥ 1 (57% vs 48%; p < 0.01), and African American children having a higher mean physician global assessment. A larger proportion of African American children was currently receiving or had previously received a biologic DMARD (55% vs 44%; p < 0.01). Higher proportions of both African American and Asian children were RF-positive relative to white children (p < 0.05 for both).

Ethnicity data were available for 4644 of 4682 children (11% Hispanic/Latino). A higher proportion of Hispanic/Latino children reported income < $50,000 than non-Hispanic/Latino children; and there was a small but statistically significant difference between health insurance status between the 2 groups (99% of non-Hispanic/Latino children with insurance vs 97% of Hispanic/Latino children; p < 0.01; Table 2). There was no significant difference between disease duration prior to symptom onset and first
visit to a rheumatologist between ethnicity categories. A higher proportion of Hispanic/Latino children were RF-positive compared to non-Hispanic/Latino children (23% vs 9%; p < 0.01) and Hispanic/Latino children had a higher average physician global assessment of disease activity.
Joint damage. Eighty percent of the children included in the race analyses and 15% of those included in the ethnicity analyses had an imaging study. Asian children were more likely than white children to have had an imaging study (94% vs 85%; \( p = 0.03 \)). There were no other significant differences between groups. Twenty-five percent of children included in the analyses who had imaging performed had evidence of joint damage.

African American children had a higher OR of joint damage on radiographic imaging (unadjusted OR 1.8, 95% CI 1.3–2.4; Table 3). This association approached statistical significance after adjustment for each of the sets of covariates (adjusted OR 1.6, 95% CI 1.0–2.5). Hispanic/Latino children had increased odds of joint damage compared to non-Hispanic/Latino children, which was no longer significant after sequential adjustment for each of the sets of covariates (adjusted OR 1.2, 95% CI 0.9–1.7). Limiting the analysis to only children with polyarticular-course disease did not change the associations. However, limiting the analysis to children with polyarticular-course disease did result in significantly increased odds of joint damage among African American children (adjusted OR 1.9, 95% CI 1.0–3.1; \( p = 0.04 \)).

Because of the smaller number of children of Asian race and the relatively small number of these patients with joint damage on radiographic imaging \((n = 20)\), the association between Asian race and joint damage was explored in a limited model. There was no significant difference in the unadjusted odds of joint damage for children of Asian race compared to white subjects either for all JIA categories combined (OR 1.2, 95% CI 0.7–2.0) or among the subset of the cohort with polyarticular-course JIA (OR 1.3, 95% CI 0.6–2.6). In a limited model restricted to children with polyarticular-course JIA and adjusted for RF status, income, age at disease onset, sex, and duration of disease, the association remained nonsignificant (OR 1.2, 95% CI 0.5–2.9).

Pain. There was no significant association between African American race and odds of higher parent or proxy-reported pain (pain score \( \leq 2 \)) after sequential adjustment for each set of covariates (adjusted OR 1.1, 95% CI 0.7–1.7; Table 4). Parent/proxies of Asian children reported significantly decreased odds of increased pain compared to those of white children (adjusted OR 0.2, 95% CI 0.1–0.6). This association remained significant after limiting the analyses to children with polyarticular-course disease. The associations between race and pain were similar when pain scores were dichotomized as \( \leq 5 \) or \( > 5 \) (results not shown). Hispanic/Latino children had significantly increased odds of higher pain compared to non-Hispanic/Latino children in the unadjusted univariate model; however, this association was not significant after sequential adjustment for the sets of covariates (adjusted OR 1.1, 95% CI 0.9–1.5). The \( R^2 \) for both of the final models of race, ethnicity, and pain were not significant after sequential adjustment for the sets of covariates (adjusted OR 1.1, 95% CI 0.7–1.7; Table 4). Parent/proxies of Asian children reported significantly decreased odds of an elevated CHAQ at both cutoffs analyzed. These odds remained significantly decreased for Asian children with polyarticular-course disease when the median CHAQ score was used as a cutoff; however, it was not significant when the 75th percentile was used (OR 0.2, 95% CI 0.05–1.0; \( p = 0.06 \)).

There was no significant difference between CHAQ scores for Hispanic/Latino children compared to non-Hispanic/Latino children for the model in which CHAQ scores were dichotomized as 0 or > 0; however, Hispanic/Latino children had 1.5-fold increased odds of having CHAQ scores greater than the 75th percentile compared to non-Hispanic/Latino children (OR 1.5, 95% CI 1.1–2.1). When this analysis was limited to children with polyarticular-course JIA, Hispanic/Latino children no longer had evidence of joint damage.

Table 3. Unadjusted and adjusted (OR) for the presence of joint damage on radiographic imaging. Data in bold type indicate \( p < 0.05 \).

<table>
<thead>
<tr>
<th>Joint Damage on Radiographic Imaging</th>
<th>African American*</th>
<th>Hispanic or Latino**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unadjusted OR (95% CI)</td>
<td>1.8 (1.3–2.4)</td>
<td>1.4 (1.1–1.8)</td>
</tr>
<tr>
<td>Adjusted OR (95% CI)</td>
<td>1.7 (1.2–2.4)</td>
<td>1.5 (1.2–1.9)</td>
</tr>
<tr>
<td>Age at disease onset, disease duration, sex</td>
<td>1.6 (1.0–2.6)</td>
<td>1.3 (1.0–1.7)</td>
</tr>
<tr>
<td>+ Socioeconomic statusa</td>
<td>1.6 (1.0–2.5)</td>
<td>1.3 (0.9–1.8)</td>
</tr>
<tr>
<td>+ Disease characteristicsb</td>
<td></td>
<td></td>
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<tr>
<td>+ Treatmentc</td>
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</tbody>
</table>

* Whites as referent group; ** non-Hispanics and non-Latinos as referent group. Each group of variables was added to the logistic regression model in the sequence indicated above. Groups consisted of the following variables: \( a \) income; insurance status. \( b \) JIA category (oligoartthritis, polyarthritus, systemic), rheumatoid factor status; physician global assessment of disease activity; active joint count. \( c \) Current or past treatment with a non-biologic disease-modifying antirheumatic drug (DMARD); current or past treatment with a biologic DMARD.
increased odds of higher disability (OR 1.4, 95% CI 1.0–2.1; p = 0.06). The R² for these models ranged from 0.11 to 0.13, indicating that a small amount of variance (11%–13%) in the CHAQ was explained by the covariates.

DISCUSSION
This analysis describes the associations between race, ethnicity, and important disease outcomes in JIA, including joint damage, pain, and disability. In this cohort of patients enrolled in the CARRA Registry, both African American and Hispanic/Latino children tended to have increased disease activity relative to the referent groups. A higher proportion of African American children were receiving or had received a biologic agent, potentially reflecting their increased disease activity and higher prevalence of RF positivity, a known risk factor for aggressive disease. In the univariate analyses, both African American race and Hispanic/Latino ethnicity were significantly associated with joint damage on imaging. However, the OR for joint damage on radiographic imaging were no longer significant after adjustment for patient characteristics and socioeconomic status. Asian children reported less pain and disability relative to white children and these associations remained significant after controlling for patient, disease-related, and treatment variables including annual income, insurance status, JIA category, RF status, active joint count. Current or past treatment with a nonbiologic disease-modifying antirheumatic drug (DMARD); current or past treatment with a biologic DMARD. JIA: juvenile idiopathic arthritis.

Table 4. Unadjusted and adjusted OR for pain. * Pain was measured on a 0–10 numeric rating score. Pain scores were dichotomized as ≤ 2 or > 2, based on the median score for the cohort. Data in bold type indicate p < 0.05.

<table>
<thead>
<tr>
<th>Patient- or Proxy-reported Pain</th>
<th>African American*</th>
<th>Asian*</th>
<th>Hispanic or Latino**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unadjusted OR (95% CI)</td>
<td>1.6 (1.2–2.1)</td>
<td>0.4 (0.2–0.7)</td>
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</tr>
<tr>
<td>Adjusted OR (95% CI)</td>
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<td>0.3 (0.2–0.5)</td>
<td>1.3 (1.1–1.6)</td>
</tr>
<tr>
<td>Age at disease onset, disease duration, sex</td>
<td>1.2 (0.9–1.6)</td>
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</tr>
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<td>0.2 (0.1–0.5)</td>
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<tr>
<td>+ Disease characteristicsb</td>
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* Whites as referent group; ** non-Hispanics and non-Latinos as referent group. Each group of variables was added to the logistic regression model in the sequence indicated above. Groups consisted of the following variables: a Income; insurance status. b JIA category (oligoarthritis, polyarthritis, systemic); rheumatoid factor status; physician global assessment of disease activity; active joint count. c Current or past treatment with a nonbiologic disease-modifying antirheumatic drug (DMARD); current or past treatment with a biologic DMARD. JIA: juvenile idiopathic arthritis.

Table 5. Unadjusted and adjusted (OR) for functional ability. Functional ability was measured using the Childhood Health Assessment Questionnaire (CHAQ). CHAQ scores were dichotomized as either 0 or > 0 (median score for the cohort), or ≤ 5 or > 5 based on the 75th percentile scores for the cohort. Data in bold type indicate p < 0.05.

<table>
<thead>
<tr>
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<th>African American*</th>
<th>Asian*</th>
<th>Hispanic or Latino**</th>
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</thead>
<tbody>
<tr>
<td>Unadjusted OR (95% CI)</td>
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<td>0.5 (0.3–0.7)</td>
<td>1.4 (1.2–1.7)</td>
</tr>
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<tr>
<td>Age at disease onset, disease duration, sex</td>
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Although the association between race, ethnicity, and disease outcomes has not been described in JIA, these associations have been examined in more detail for patients with RA; and specific initiatives, such as the Consortium for Longitudinal Evaluation of African Americans with Early Rheumatoid Arthritis (CLEAR), are focusing specifically on this area. In RA, disparities have been noted for medical treatments, with non-white patients experiencing significantly increased disease duration prior to initiation of DMARD therapy after disease onset and also being significantly less likely to receive DMARD therapy overall. This finding is in contrast to our cohort, in which a larger proportion of African Americans received biologic DMARD therapy compared to whites, perhaps reflecting confounding by indication, with patients with more severe disease receiving more aggressive therapy. This difference may also reflect that the majority of US children without private insurance have coverage through Medicaid to assist with medication costs, which may not be the case for adults without private insurance. Additional sources of disparities between race and ethnicity categories that have been explored in RA include variation in patient preferences regarding treatment options, medication adherence, and the patient-provider relationship and communication.

While these data from adults with RA are not directly applicable to patients with JIA, particularly because of the higher prevalence of comorbidities in adults, they highlight important considerations for future analyses.

The analyses presented here should be interpreted in the context of several important limitations that may have influenced the results and their generalizability. Although the CARRA Registry includes children from centers across the United States, this cohort represents a convenience sample and selection bias may have influenced the cohort composition, with patients with more stable disease status and stronger relationships with their provider being more likely to be approached for inclusion. Children included in this cohort were also English-speaking, were treated by pediatric rheumatologists at academic centers, and a large majority had health insurance. The duration between symptom onset and first rheumatology visit, a potential surrogate for access to care, was similar across the race and ethnicity categories in this cohort, as well. More heterogeneous cohorts that include non-English speakers and children treated by adult rheumatologists or at centers not enrolling into the CARRA Registry may show different associations than those reported here, particularly in comparison to those that assessed joint damage and approached statistical significance. Additional variables that could have had potentially important effects on the models, including parent education, language(s) spoken in the home, private versus public insurance, and medication adherence are not currently collected in the registry and could not be evaluated here. Race and ethnicity data were based on self-report, which may have led to some increased heterogeneity within the groups and/or misclassification. Further, these are cross-sectional analyses, which limit our ability to determine how overall disease course may vary between children of difference race and/or ethnicity. Lastly, these analyses represent only one approach to modeling the complicated relationship between race, ethnicity, and outcomes. Different models assessing different combinations of covariates may generate different results.

These data suggest that race and ethnicity are likely variably associated with disease outcomes in children with JIA in the United States. Identification of patient characteristics that determine the direction of these associations and additional explorations of the relationship between race, ethnicity, and patient-reported outcomes may enable early identification of children at risk for poorer outcomes who may benefit from specifically tailored interventions.

APPENDIX 1
List of study collaborators. CARRA Registry Site Principal Investigators: James Birmingham, West Michigan Rheumatology, Michigan State University; Peter Blier, Baystate Medical Center; Norman Hlouwite, Children’s Hospital at Montefiore; Thomas Graham, Vanderbilt Children's Hospital; Fatma Dedegolu, Children’s Hospital Boston; Pamela Weiss, Children’s Hospital of Philadelphia; Kathryn Tokor, Children’s Hospital of Pittsburgh of UPMC; Rita Jerath, Children’s Medical Center, Medical College of Georgia Health System; Marisa Klein-Gitelman, Ann & Robert H. Lurie Children’s Hospital of Chicago; Andrew Lasky, Children’s Mercy Hospital; Lawrence Jung, Children’s National Medical Center; Steven Spalding, Cleveland Clinic Foundation; Lawrence Zemel, Connecticut Children’s Medical Center; Consuelo Rabinovich, Duke University Medical Center; Sampath Prahalad, Emory University School of Medicine; Marc Natter, Tufts Medical Center; Ali Yalcindag, Hasbro Children’s Hospital; Kathleen O’Neil, Indiana University School of Medicine; Jennifer Weiss, Hackensack University Medical Center; Daniel Kingsbury, Randall Children’s Hospital; Ann Reed, Mayo Clinic; Judd Olson, Children’s Hospital of Wisconsin; Natasha Ruth, Medical University of South Carolina; Lisa Imundo, Columbia University Medical Center; Gloria Higgins, Nationwide Children’s Hospital; Philip Kahn, New York University Langone Medical Center; Ilona Szer, Children’s Hospital of San Diego; Nora Singer, Metro Health System; Nandini Moorthy, Robert Wood Johnson University Hospital; Elizabeth Chalom, Saint Barnabas Medical Center; Reema Syed, Saint Louis University Hospital; Ana Quintero, San Jorge Children’s Hospital Santurce, Ponce School of Medicine; Beth Gottlieb, Cohen Children’s Medical Center of New York; Deborah Rothman, Shriners Hospitals for Children; Donald Goldberg, Drexel University College of Medicine; Christy Sandborg, Stanford University Medical Center; Barry Myones, Baylor College of Medicine; Andrew White, Saint Louis Children’s Hospital; Emily von Scheven, University of California at San Francisco Medical Center; Karen Onel, University of Chicago Medical Center; J. Roger Hollister, The Children’s Hospital Colorado; Polly Ferguson, University of Iowa Hospitals and Clinics; Carol Lindsley, University of Kansas Medical Center; Kenneth Schikler, University of Louisville School of Medicine; Richard Vehe, University of Minnesota; Andrea Sestak, University of Oklahoma Health Sciences Center; Marilynn Punaro, University of Texas Southwestern Medical Center Dallas; Christi Inman, University of Utah Hospitals and Clinics; Anna Huttonlocher, American Family Children’s Hospital; Leslie Abramson, The University of Vermont – Fletcher Allen Health Care; Angela Robinson, Rainbow Babies and Children’s Hospital; Deborah McCurdy, University of California, Los Angeles Medical Center; Thomas
Lehman, Hospital for Special Surgery; Amanda Brown, Louisiana State University Health Science Center; Thomas Griffin, Levine Children’s Hospital/Carolina’s Medical Center.

REFERENCES