Background: Complement activation is a hallmark of SLE pathology. We previously found that iC3b/C3 ratios associated with active disease and clinically meaningful changes in SLE disease activity. Since SLE is more severe in non-white populations, we hypothesized that iC3b/C3 ratios would be a more sensitive marker of disease activity in non-white populations.

Objectives: We examined the relationship of iC3b/C3 ratios between African-American (AA) and White subjects with classified SLE.

Methods: 159 adult SLE patients treated at the Washington University Lupus Center were enrolled in this observational study. 83 patients with 3-7 study visits were used for this longitudinal analysis. C3 and C4 were measured by nephelometry; iC3b by a lateral flow assay using an investigational medical device. SLE disease activity was measured using the SLEDAI 2K Responder Index=50 instrument. Statistical analyses were performed using SAS v9.4. Multilevel regression models examined associations for SLE disease activity. Ordinal logistic regression models with generalized estimating equation modeling (GEE) examined associations for SLE disease activity. Ordinal logistic regression models with generalized estimating equation modeling (GEE) examined associations for SLE disease activity. Ordinal logistic regression models with generalized estimating equation modeling (GEE) examined associations for SLE disease activity. Ordinal logistic regression models with generalized estimating equation modeling (GEE) examined associations for SLE disease activity.

Results: iC3b/C3 ratios correlated with active disease and clinically meaningful changes in SLE disease activity. Furthermore, in univariate regression analysis, only the iC3b/C3 ratio in AA with active disease, SLE subjects, with the association of the iC3b/C3 ratio in AA was stronger (Figure 1). In addition, AA with SLE associated C4, ESR, and dsDNA with active disease, SLE patients, with the association of the iC3b/C3 ratio in AA was stronger (Figure 1). In addition, AA with SLE associated C4, ESR, and dsDNA with active disease, SLE patients, with the association of the iC3b/C3 ratio in AA was stronger (Figure 1). In addition, AA with SLE associated C4, ESR, and dsDNA with active disease, SLE patients, with the association of the iC3b/C3 ratio in AA was stronger (Figure 1). In addition, AA with SLE associated C4, ESR, and dsDNA with active disease.

Conclusion: iC3b/C3 ratios better correlated with active disease in AA compared with Whites. Furthermore, iC3b/C3 ratios correlated with clinically meaningful changes in disease activity only in AA. These data suggest that complement activation in SLE is dependent on race.

REFERENCE: