**ANTIBODIES TO MALONDIALDEHYDE-ACETALDEHYDE (MAA) PROTEIN ADDUCT AS A BIOMARKER FOR CARDIOVASCULAR MANIFESTATIONS IN SYSTEMIC LUPUS ERYTHEMATOSUS**

**K. Su**1, Y. Yu1, T. Wang1, P.D. Lopez2, C. Tineo3, G. Paulino3, M. Hearth-Holmes1, E. Loyo3

1University of Nebraska Medical Center, Omaha, NE, USA

2New York Medical College, New York, NY, USA

3Hospital Regional Universitario José Ma Cabral Baez, Santiago, Dominican Republic

**Objective**: Systemic lupus erythematosus (SLE) is a complex autoimmune disease characterized by devastating end-organ manifestations. Cardiovascular disease (CVD) is a leading cause for pre-mature death in SLE patients across different ethnicities. While the traditional Framingham risk factors likely contribute to CVD in SLE, they cannot fully account for the highly increased risk of CVD in SLE. Malondialdehyde-acetaldehyde (MAA) protein adduct (a byproduct of oxidative stress) and antibodies to MAA have been suggested to mediate early inflammation in atherosclerotic disease. The purpose of this study is to determine whether SLE patients develop anti-MAA antibodies and whether anti-MAA antibodies identify SLE patients with cardiovascular manifestations.

**Methods:** 186 SLE patients who fulfilled the 1997 revised American College of Rheumatology criteria for the classification of SLE and 186 non-diseased healthy controls (matched in age and sex) were recruited from the Dominican Republic for the study. Enzyme-linked immunosorbent assay (ELISA) was performed to determine the serum levels of anti-MAA IgG antibodies. Data were analyzed using Mann Whitney test. A p value of <0.05 was considered significant.

**Results:** The mean anti-MAA IgG levels in SLE patients was 0.13 (in optical density units; SD 0.27, 95% CI 0.09-0.17), which is significantly higher than that in healthy controls (mean: 0.06; SD 0.07, 95% CI 0.05-0.07; p=0.043). SLE patients with vasculitis have significantly higher levels of anti-MAA than SLE patients without vasculitis (mean: 0.22 versus 0.07; p=0.013). SLE patients with pericarditis also have higher levels of anti-MAA than SLE patients without pericarditis (mean: 0.25 versus 0.07; p=0.019).

**Conclusion:** Anti-MAA IgG antibodies are significantly elevated in SLE patients compared to healthy controls. The pattern of anti-MAA IgG antibodies is able to distinguish SLE patients with cardiovascular manifestations (such as vasculitis and pericarditis) from those without. These results suggest that anti-MAA antibodies may be a biomarker for CVD in SLE. Future studies will determine if MAA and/or anti-MAA antibodies play a role in the pathogenesis of CVD in SLE.