Abstract of Dissertation (continued)

librium. I have also used the data generated here to perform several tests of selective neutrality and have tested whether the alleles at each locus are correlated with malaria endemicity. The results of my analyses show that variants at these loci are indeed correlated with malaria endemicity and there are some suggestive signatures natural selection at both *ICAM-1* and *CD36*. The work accomplished here contributes to our knowledge of the scope of genetic diversity at *ICAM-1* and *CD36* and help to elucidate whether there are variants at these loci that are functionally adaptive in malaria-endemic environments.

The George Washington University
Hominid Paleobiology Program
and Department of Anthropology
are pleased to announce

Felicia Gomez

in defense of her dissertation

Genetic Variation at *ICAM-1* and *CD36*: A Study of Malaria Resistance Candidate Loci in Diverse Global Human Populations

Tuesday, June 26, 2012, 1:30 p.m.
CASHP Seminar Room
Building BB 308
2114 G St., NW
Washington, DC

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Abstract of Dissertation

Infectious disease has played an important role in driving human evolution and human population genetic variation. Malaria, caused by *Plasmodium falciparum*, is a particularly important infectious disease and is arguably one of the strongest selective forces in recent human evolution. Signatures of natural selection within the human genome can be important indicators of functional and/or adaptive variation. These data have the potential to identify loci that affect disease susceptibility that may be missed by conventional genotype/phenotype association studies or modern genome-wide association studies.

ICAM-1 and *CD36* are two loci that code for receptors on the surface of endothelial cells. During a *P. falciparum* infection, parasite proteins that are exported to the red blood cell surface adhere to ICAM-1 and CD36. This process of cytoadherence is the primary occurrence that leads to the development of fatal syndromes called severe and cerebral malarial. Because *ICAM-1* and *CD36* are integrally involved in the pathogenesis of these syndromes I have hypothesized that variants that are functionally adaptive in malaria endemic environments will be targets of natural selection. I have also hypothesized that identifying patterns of genetic variation that are consistent with natural selection will help to recognize functionally adaptive variants.

In the current study I have re-sequenced *ICAM-1* and *CD36* in several diverse African and non-African populations. Using these data I have identified single nucleotide polymorphisms and have described nucleotide diversity at each locus. I have also inferred haplotype phase, characterized patterns of haplotype diversity at each locus, and examined patterns of linkage disequi-

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