Abstract for NISBRE

Presentation format: Oral and Poster

Corresponding author: isilvestre@medicine.nevada.edu

Title: Fc gamma receptors (FcγRs) IIIA and IIC polymorphisms in myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS)

Authors: Stephen Anderson, Ph.D.[1], Daniel Peterson, M.D.[2,3], Lucinda Bateman, M.D.[4], Suzanne Vernon, Ph.D.[4], Dorothy Hudig, Ph.D.[5], Laura Meadows, B.S.[5], Wei Yang, M.D., Ph.D.[6], and Isabel Barao, Ph.D.[5]

Institutional Affiliations: [1]Leidos Biomedical Research Inc., [2]Simmaron Research, Inc., [3]Sierra Internal Medicine, [4]Bateman Horne Center for ME/CFS and FM, [5] University of Nevada, Reno, School of Medicine, [6]University of Nevada, Reno, School of Community Health Sciences

Key Words: Myalgic encephalomyelitis/chronic fatigue syndrome, Fcγ receptor**,** polymorphisms

BACKGROUND and OBJECTIVE:
Myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS) is a complex, multisymptom illness involving persistent fatigue and immune dysfunction. Fc gamma receptors (FcγRs) are key immune receptors required for an effective immune response. They function by binding the Fc region of immunoglobulin G (IgG) and transmitting stimulatory or inhibitory signal to immune cells. Polymorphisms of FcγRs have been identified as genetic factors influencing susceptibility to disease. In this study, we investigate a possible association between FcγRs polymorphisms and ME/CFS.

METHODS:
Polymorphisms of FcγRIIIA and FcγRIIC were determined in 39 patients with ME/CFS by polymerase chain reaction (PCR)-based allotyping methods with allele-specific primers and DNA sequencing.

RESULTS: In the ME/CFS patients, the frequency of the FcγRIIIA-158-F allele was 0.75, versus 0.57 in the healthy population, and the frequency of the FcγRIIIA-158-V allele was 0.24, versus 0.43 in the healthy population (P=0.0057). The genotype distribution of the FcγRIIIA-158 F/F and FcγRIIIA-158 V/V was also significantly different from that of the healthy population (P=0.0175). In the case of FcγRIIC, the Stop allele and Stop/Stop genotype were overrepresented in the ME/CFS patients ($P < 0.05$).

DISCUSSION and CONCLUSIONS:
The findings of this study suggest an association of the FcγRIIIA-158-F and FcγRIIC-Stop alleles with ME/CFS immune dysfunction. Validation of these results in additional patient cohorts is needed to support this finding and to understand the association of these polymorphisms as risk factors in ME/CFS.

Grant Support: This research has been supported by CTR-IN 1U54GM10444-2, R21AI117491, Simmaron Research, Inc., Solve ME/CFS Initiative, and private donors