

# 3<sup>rd</sup> International Conference and Exhibition on Clinical & Cellular Immunology

September 29-October 01, 2014 DoubleTree by Hilton Baltimore-BWI Airport, USA

## Differential expression of SOCS1/SOCS3 ratios in virus-infected macrophage cell lines

Nancy J Bigley

Wright State University, USA

Herpes simplex virus type 1 (HSV-1) infection was examined on murine J774A.1 and RAW 264.7 macrophage cell lines unpolarized (M0) or polarized by IFN- $\gamma$  and or cytokines to either an M1 (pro inflammatory) or M2 (immunomodulatory and tissue remodeling) phenotype. Dengue virus2 (DENV2) infection of RAW264.7 cells was also investigated. Morphology, cell viability, expression of cell surface markers, expression ratios of suppressor of cytokine signaling molecules SOCS1/ SOCS3, and capacity to replicate HSV-1 were determined. Morphological differences were abrogated by virus infection. Infection with either virus diminished expression of CD14 and CD86 in M1 populations, cells which exhibited significant decreases ( $p < 0.001$ ) in cell viability at 24 hours post infection. Western blotting suggested that SOCS1/SOCS3 expression ratios differed between uninfected and HSV-1-infected RAW264.7 M1 cells, while ratios in the uninfected cells and the DENV2-infected M1 cells remained similar. Flow cytometry demonstrated that this ratio between uninfected M1 cells and HSV-1-infected M1 phenotypes fell from 6:1 to 1:1 in M1 phenotypes of J774A.1 cells. SOCS3 expression increased 7-fold in the M1 infected J774A.1 cells compared to uninfected M1 cells. Three- to four-fold decreases in HSV-1 yield occurred by 24 hours after infection in the M1 subpopulations. This decrease may reflect the anti-inflammatory effect of the relative increases in SOCS3 expression in these cells and/or the decreased survival of M1 cells after virus infection. Increases in SOCS3 expression by the HSV-1-infected M1 populations suggest a shift in the cell's ability to respond to virus, a change not seen in DENV2 infection.

### Biography

Nancy J Bigley received the BS degree in bacteriology from Penn State University in 1953 and the PhD from The Ohio State University in 1957 and has taught immunology and microbial pathogenesis at The Ohio State University, Chicago Medical School and Wright State University. She has published more than fifty papers in the field and authored two editions of a textbook in immunology. Her areas of research involve viral and cellular immunity and more recently innate immunity at the cellular level.

[nancy.bigley@wright.edu](mailto:nancy.bigley@wright.edu)