Alfredo Garzino-Demo, J Antivir Antiretrovir 2011, 3:4
doi: http://dx.doi.org/10.4172/1948-5964.S1.4

OMICS Group Conferences
Accelerating Scientific Discovery

International Conference and Exhibition on
VIROLOGY
5-7 September 2011 Baltimore, USA

Defensins and Chemokine receptors: Protecting highly susceptible cells from HIV infection by inducing APOBEC3G expression

Alfredo Garzino-Demo[1,2], Mark K. Lafferty[1,2], Jennifer Bharucha[1,2], Lingling Sun[1,2], Walter Royal[3], Suzanne Gartner[1,4], Wuyuan Lu[1,5]

[1]Institute of Human Virology
[2]Department of Microbiology and Immunology
[3]Department of Neurology
[4]Department of Medicine
[5]Department of Biochemistry
[6]University of Maryland School of Medicine, USA

β-defensins are antimicrobial peptides secreted by epithelial cells that can bind to cellular receptors. CCR2 and CCR6 are the two cellular receptors known to bind human ß-defensin (hBD) 2 and -3. Both of these receptors are of crucial relevance in HIV infection. CCR6 is expressed, often in concert with CCR5, on cells that are highly susceptible to HIV infection: memory T cells, Th_{17} cells, $\alpha 4ß7^+$ cells, and defects in $CD4^+CCR6^+$ cells have been associated with faster AIDS progression. CCR2 is expressed on monocytes and macrophages, cells that are reservoirs of HIV infection and that are known to mediate central nervous system damage. Our studies show that hBD2, hBD3, and CCR6 ligand MIP-3α/CCL20 inhibit HIV infection via CCR6 by increasing expression of the antiviral protein APOBEC3G. This increase is due to a transcriptional mechanism mediated by intracellular signaling. hBD2 also inhibits HIV replication in macrophages that express CCR2. Our findings suggest novel therapeutic and preventive approaches that exploit CCR6 and CCR2-mediated intracellular signaling to inhibit HIV infection in highly susceptible cells.