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Vpu is responsible for HIV-1 evasion of natural killer cells

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Natural killer (NK) cells are recognized as being crucial in the defense against viruses. NK cells interaction with virus-infected cells ultimately leads to the death of the virus-infected cells. However, HIV-infected cells are refractory to lysis by NK cells. We have found that resistance to lysis is not due to the failure of HIV to activate NK cells but rather to HIV's ability to prevent NK cell release of its lytic granules. These outcomes are the result of the combined action of viral proteins that both lead to NK activation but ultimately act to suppress the lytic function of NK cells. The key findings from our laboratories, demonstrate that: 1) HIV-1 Nef down modulates HLA-A and –B and 2) HIV-1 Vpr induces ligands to NK cell activation receptor NKG2D. Both of these events lead to NK cell activation. However, HIV-1 Vpu counters NK cells' ability to degranulate by down modulation of NTB-A a homotypic ligand to the NK cell coactivation receptor, NTB-A. NTB-A on the infected cells is critical for eliciting NK cell cytolytic response because degranulation requires both simultaneous engagement of activation with coactivation receptors. Vpu acts to prevent NTB-A surface expression by retention of NTB-A within the trans-Golgi network of the infected cell. Vpu actions on NTB-A are independent of Vpu's activity against CD4. Vpu acts on NTB-A in a similar fashion as Vpu mediated suppression of the host cell innate factor tetherin/BST-2. The ultimate goal will be to use the knowledge gained from studies on Vpu-NTB-A interactions to devise novel therapeutic approaches aimed at rendering HIV-infected cells sensitive to NK cell killing.

Biography

Ed Barker has completed his Ph.D. at the University of Illinois at Chicago and postdoctoral studies from University of California at San Francisco. Currently, he is an Associate Professor in the Department of Immunology and Microbiology at Rush University Medical Center in Chicago. Ed is working on his sixth year of an NIH grant to study how HIV evades natural killer cells.

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