In 2012 more than 226,870 new cases of invasive breast cancer will be diagnosed (1). In the 1970s the connection was first made between cancer and virus infection. One particular virus that has been associated with breast cancer is human cytomegalovirus (HCMV). This virus is endemic in the human population and establishes life-long latency in the host.

HCMV infected cells produce cmvIL-10 protein, a viral homolog of the human interleukin-10 protein (hIL-10), which activates the JAK-STAT signaling pathway (2). Like hIL-10, cmvIL-10 has also been shown to bind to the IL-10 receptor and activate the JAK-STAT pathway as well (3).

Here, we show that cmvIL-10 activates Stat3 in human breast cancer cells through phosphorylation (pStat3). Stat3 has been identified as a key factor in tumor progression and evasion of programmed cell death (4). These results suggest that the cmvIL-10 protein may cause breast cancer cells to become more invasive or metastatic.

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**Results**

**cmvIL-10 upregulates the hIL-10 receptor**

- cmvIL-10
- cmvIL-10

**cmvIL-10 triggers Stat3 phosphorylation**

- cmvIL-10
- cmvIL-10

**Conclusion**

MDA breast cancer cells treated with cmvIL-10 exhibit upregulation of the IL-10 receptor on the cell surface as compared to untreated cells. Furthermore, MDA cells express Stat3 protein which becomes phosphorylated and activated by the binding of cmvIL-10 to the IL-10 receptor.

To further examine the role of cmvIL-10 in the progression of breast cancer, PCR arrays will be used to evaluate resulting changes in gene expression. Genes encoding adhesion factors and extracellular enzymes are of particular interest, as they play a crucial role in dissemination of cancer cells and the establishment of secondary tumors. Additional follow up experiments will include ELISAs to monitor enzymes levels and migration assays to evaluate invasive potential.

If cmvIL-10 does effect these cellular changes, this might suggest that HCMV positive cancer patients would benefit from anti-viral therapy combined with traditional chemotherapy. Our results are expected to clarify the role of HCMV in breast cancer and could have significant implications for patient treatment strategy.

**Works Cited**


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