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## Biopharmaceutical Platform: Anti-Cancer, Anti-Inflammatory and Immunosuppressive Fusion Proteins

McGill University is seeking to out license a broad portfolio of fusion proteins. Fusion transgenes are of great interest due to their many advantages, amongst others longer protein half-life, higher synergistic effects and targeting of specific receptors. Our platform allows the creation of many different fusion proteins to suit specific needs. There is a clear medical need for new therapeutic approaches which could be filled by novel fusion proteins in a growing biopharmaceutical therapies market.

### Applications

Depending on the fused moieties, the resulting fusion proteins have unique characteristics different from the individual moieties and can be used as therapeutics in organ transplantation, autoimmune diseases. They can also be used in cancer therapy and for their antimetastatic or pro-angiogenic effects.

### Description

We have created fusion transgenes using a number of different molecules amongst others: granulocyte macrophage colony stimulating factor (GM-CSF), interleukins, monocyte chemoattractant protein-1 (MCP-1) and transforming growth factor  $\beta$  (TGF $\beta$ ). Each has different therapeutic advantages; for example, GIFT15 is an immunosuppressant which can promote allogeneic and xenogeneic engraftment and prevent transplant rejection; GMME1 is a CCR2 antagonist with both immunosuppressive and anticancer properties, whereas FIST has immunostimulatory properties resulting in inhibition of both tumor proliferation and metastasis formation.

All the fusion proteins are the subject of patent protection, from provisional applications to national phase entry in Europe, USA and Canada.

### Validation

GIFT15, has been shown to 1) suppress the recruitment of natural killer (NK) cells and NK T cells *in vivo*, 2) block the IL15 dependent IFN $\gamma$  response in mouse splenocytes, and 3) sustain the engraftment of allogeneic and xenogeneic cells rejected in immunocompetent mice.

GMME1, has been shown to 1) lead to the apoptosis of cells expressing CCR2, 2) block Th1/Th17 responses in mouse splenocytes, 3) inhibit inflammation in an EAE mouse model, and 4) sustain the engraftment of xenogeneic cells in immunocompetent mice.

FIST 1) inhibits the TGF $\beta$  pathway in immune effector cells *in vitro* by overcoming tumor-derived TGF  $\beta$  dependent immunosuppression, 2) stimulates effector cells to secrete 34 fold amounts of IFN $\gamma$  compared to an equimolar concentration of the immunostimulatory component A 3) promotes the robust recruitment of NK, NKT, cytotoxic T cells, and B cells *in vivo*, and 4) inhibits tumor cell proliferation and blocks metastases *in vivo*.

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Reference codes: ROI #06087. 08079, 08099