Dr Ranson’s laboratory uses cellular and molecular biochemical techniques to study the role of the human plasminogen activation system in cellular tissue invasion and to use this basic biological knowledge to devise new targeted anti-cancer and anti-microbial therapeutics.

In invasive diseases, cells gain an increased capacity to bind the circulating blood zymogen plasminogen and convert it into plasmin, a powerful, broad-spectrum enzyme.

Plasmin promotes tissue degradation and thus allows the cells to invade their local environment and potentially spread throughout the body.

The over-expression of the plasminogen activation system is now regarded as a potent marker of malignancy.

We have shown that this can be targeted and inhibited by a specific, naturally occurring inhibitor called PAI-2.

We aim to understand the regulation of plasminogen binding and activation on cells and to develop PAI-2 as a delivery vehicle of potent cytotoxins to malignant cancer cells.

Group A streptococcus (GAS) is a bacterium that causes minor human infections but also highly invasive flesh-eating diseases. Subversion of host plasminogen is believed to play a critical role in GAS invasive disease.

We aim to understand the mechanisms by which GAS can bind and activate plasminogen to plasmin and potentially identify new anti-microbial targets.

Current research questions include:

- Characterisation of plasminogen receptors on the surface of human cancer cells.
- Defining the mechanism of PAI-2 internalisation and regulation of plasminogen activation in cell invasive processes
- Pre-clinical development of PAI-2 conjugates for the treatment and imaging of cancer
- Characterisation and development of a marine cytotoxin as a novel anti-cancer agent.
- Preclinical development of new anti-cancer drug formulations
- Characterisation of GAS plasminogen receptors and host derived factors and the role they play in GAS invasion