Imperial College London

Imperial technology portfolio

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STp Peptides

Professor
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Professor Stella Knight is an immunologist and pioneer in studies of dendritic cells. She has established the Immunopathology laboratory, the Antigen Presentation Research Group, Imperial College as a major research facility for St. Mark's Hospital.
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Overview

Current treatments for inflammatory bowel disease (IBD) include aminosalicylates, corticosteroids and immunosuppressants but many patients fail to respond.

By capitalising on the cross-talk between regulators of the immune system and commensal bacteria, Imperial College team has developed a novel therapy for IBD, consisting of STp protease resistant peptide. STp is secreted from L. plantarum and is detectable in healthy intestine; i.e. a natural product. It appears to modulate a regulatory cytokine profile in dendritic cells (DC), to dampen down immune responses to gut organisms in IBD.
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Technology

- STp effects epithelial cells to promote mucosal barrier function.
- It increases IL10 and decreases IL12 production.
- STp restores TLR expression on UC derived DCs to 'normal' level (i.e. reduced TLR2 and TLR4).
- It restores maturation potential (CD40, CD80) and stimulatory capacity (proliferating T cells) of UC derived DCs.
- Protease resistant and, unlike biologics, suitable for oral administration.
- There is potential for development of a companion diagnostic due to absence of STp in patients with UC vs healthy individuals.

Development Stage

In vivo data in a murine model of ulcerative colitis show reduced gut inflammation adverse signs of the disease.
The team continues to validate the clinical diagnostic potential of STp, evaluate the efficacy and PK/PD of STp in animal models vs conventional anti-TNFα. They are currently addressing questions around recombinant expression/ production of STp, and build data on mechanism of action.

Intellectual Property

This composition or matter and method of use is protected by a patent in EU, USA, Canada, Australia, China, Japan and Israel).