



NSI Meeting Announcement

Date: June 16, 12.00–13.00

Venue: Rikshospitalet, Green Auditorium

IL-15 and coeliac disease

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Interactions between the intestinal epithelium and the immune system**



Coeliac disease offers a unique model to decipher the links between environment and genetics in the control of the immune response in the intestine. Recent work has shown how the structural properties of HLA-DQ2/8 molecules, which confer the major genetic risk, promote the recognition of a subset of gluten-derived peptides by lamina propria CD4+ T cells and the subsequent activation of adaptive immunity.

More recent evidence points to the complementary role of innate immunity orchestrated by the proinflammatory cytokine IL-15 and triggered by a distinct peptide common to the N-terminus of A-gliadins. In CD patients, exposure to this peptide triggers IL-15 expression and results in:

- 1- expansion and activation of intraepithelial lymphocytes via mechanisms implicating innate immune receptors, including the NK receptor NKG2D and its epithelial ligand MIC;
- 2- enhanced activation of dendritic cells promoting the stimulation of the CD4+ adaptive T cell response;
- 3- impaired local immuno-regulation due to the blockade of the signalling pathway of TGF-beta.

Furthermore, in a subset of CD patients who become refractory to the gluten-free-diet, self-sustained production of IL-15 by enterocytes promotes the emergence of lymphoid malignancies. Although more work is needed to decipher the mechanisms underlying the abnormal expression of IL-15 in CD, these results point to IL-15 as a key player in the loss of lymphoid homeostasis in CD and as a potential therapeutic target, particularly in the subset of patients become refractory to the diet.

Refreshments will be served after the meeting. Open to all. Welcome!

Norwegian Society for Immunology (NSI)