



# Meeting Announcement

**Date: August 23, 15.00–16.00**

**Venue: Rikshospitalet, Auditorium A3.3067**



## NKG2 receptors and IL-15: a dialog between CTL and tissue targets

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The mechanisms underlying control of CTL activation in the tissue environment remain poorly understood. There is growing evidence that NK receptors expressed on CTL play an essential role in their regulation and in tissue destruction in celiac disease and organ specific autoimmune diseases such as type I diabetes. Because NK receptors can monitor the health of tissue cells through the recognition of MHC class I and class I-like molecules, they allow CTLs to discriminate between harmful and harmless antigens. Importantly, IL-15, which is induced upon infection and stress in tissue cells, up-regulates the expression and function of activating NKG2 receptors in CTL. Uncontrolled expression of IL-15 is associated with uncontrolled expression of activating NKG2 receptors on CTL and tissue damage. CTL-mediated tissue damage occurs either because NKG2 receptors lower the TCR activation threshold or because they endow CTL with NK-like cytolytic properties. NKG2D/DAP10 is a unique example of a non-ITAM (Immunoreceptor Tyrosin-based Activation motif) that can mediate direct cytolysis in CTL, allowing the destruction of stressed tissue targets in an antigen non-specific manner. In some conditions CTL undergo a more general NK reprogramming with the expression of the ITAM-bearing adaptor molecule DAP12, leading to the expression of activating NK receptors able not only to induce TCR-independent cytolysis, but also proliferation and cytokine secretion. Interestingly, the population of DAP12+ CTL is highly oligoclonal. The progressive loss of TCR control over CTL effector functions and proliferation may constitute an important step in the perpetuation of tissue damage and in the development of CTL lymphoma as seen in celiac disease.

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

**Norwegian Society for Immunology (NSI)**  
**&**  
**Faculty Division Rikshospitalet, University of Oslo**