



Endothelial cell activation in inflammation and immunity

Date: Friday, November 18, 2005, 15.00–15.45

Venue: Auditorium A1.1001, Domus Odontologica



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The physiologic circulation (homing) of leukocytes and their local recruitment to inflammatory sites is mediated by adhesion molecules and chemokines presented at the vascular surface. Such chemokines are either the product of endothelial cells themselves or transcytosed from the surrounding tissue. In the former case, endothelial cells can store chemokines in intracellular organelles and release them upon subsequent demand. To this end, we have mapped the properties of several chemokines, finding chemokines that predominantly attract innate immune can originate from two different granular compartments that are differentially sensitive to PKA, PKC and diacylglycerol signalling.

A striking feature of endothelial cells in the high endothelial venules (HEV) of secondary lymphoid tissues is their ability to recruit high numbers of circulating lymphocytes. Endothelial cells of chronically inflamed tissues adapt this phenotype but the signals that induce this change remain unknown. We recently identified a nuclear factor of HEV (NF-HEV). Of particular interest is the observation that NF-HEV shares structural homology with homeobox genes, one of which, PROX1, controls the differentiation of lymphatic vessels, therefore suggesting that NF-HEV may be involved in the control of HEV-differentiation. NF-HEV responds to TNF α and IFN- γ , and is induced in vessels of rheumatoid arthritis, inflammatory bowel disease and psoriasis.

Note: Guttorm Haraldsen is NSI's invited speaker at the Annual General Meeting. Immediately after a short break the scientific part of the annual meeting continues with:

1545 Poster presentations

1615 Abstract Session I

1745 Abstract Session II

Refreshments will be served. Open to all. Welcome!